

**Cognitive Content and Processes Related to Cravings for Alcohol**

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## Summary

This thesis reports on the relationships between craving, craving metacognitions, and craving-related cognitive processes. Processes around alcohol craving and metacognitions are poorly understood. Application of metacognitive theories suggests that the craving and metacognitions are related, yet to date no research has tested this assumption.

Four studies of individuals' responses to and beliefs about craving are presented here. The first investigated the effect of craving on metacognition by manipulating conditions of actual and perceived intoxication. Craving and metacognition were not predictive of one another; however, perceived consumption of alcohol was significantly related to metacognition about craving usefulness. As the metacognition measure used contained a limited metacognition range, the second study involved the development of a new scale, the Craving Metacognition Scale (CMS). This measure addresses the perceived antecedents and consequences of craving.

For the third study, improvements were made to the initial study, including testing a larger sample with a wider range of drinkers, applying the new metacognitions measure, and expanding craving measurement to include physiological responsiveness to cue via heart rate and galvanic skin response. Cues were provided for an earlier phase of the consumption cycle, whereby participants did not consume alcohol, thus removing any effects of alcohol on cognition, and water was provided as a neutral stimulus to avoid confounding by appetitive responses to stimuli.

Given the limited and highly specific relationships observed between craving and metacognition in my first three studies, my final study focuses on the cognitive processes related to craving. In this final study, the predictions of anxiety sensitivity and elaborated intrusions were tested against one another, to determine the role interoceptive ability plays in

craving. Although craving did not interfere with an interoceptive accuracy task, this task interfered with subsequent craving, providing support for EI Theory over anxiety sensitivity models.

Overall, by using experimental designs in three of four studies, and with the inclusion of either problematic or treatment-seeking drinkers in every study, this thesis demonstrates that craving and metacognition share relationships, but that these can be quite specific and must involve careful measurement and conceptualisation of craving and metacognition. Both craving content and processes should be considered in craving research.



## **Declaration**

I hereby declare this manuscript to be my original work, and that to the best of my knowledge, any text that is the work of other authors has been acknowledged as such and referenced appropriately. I declare that I am the student whose name appears below and that this text has not been previously submitted for the award of any other degree or diploma.

This thesis contains chapters with multiple authors. I am the primary author of each of these papers and as such am responsible for the accuracy of their content. The contributions of each author are as follows.

**Chapter 2:** General features of the overall design of this study were planned in discussion between co-authors Dr Stapinski, Ms Subotic and me, with advice from A/Prof. Baillie. The unique aspects of this study that I solely contributed included the measurement of metacognition variables, to determine their relationship with craving. Data collection was paired with other unique material collected by Dr Stapinski and Ms Subotic; however, these variables are not included here. Data were collected and managed by Dr Stapinski, Ms Subotic and me. I conducted analyses under the supervision of A/Prof. Baillie, and I wrote the manuscript, with feedback from A/Prof. Baillie, Dr Stapinski and Ms Subotic. A Macquarie Research Development Grant awarded to Dr Stapinski and A/Prof. Baillie funded the research, and participants were recruited as part of a larger clinical trial, which was funded by a National Health and Medical Research Council Grant awarded to A/Prof. Baillie and his colleagues.

**Chapter 3:** I was responsible for the design and development of the research described in this chapter under the supervision of co-author A/Prof. Andrew Baillie, including application for Ethics Committee approval, data collection and management, data analysis, and the authorship of this paper. The manuscript was reviewed and comments provided by A/Prof. Baillie and fellow PhD candidate Ms Mirjana Subotic.

**Chapter 4:** Ms Subotic and I designed the general features of this study collaboratively and jointly prepared the Ethics Committee application, with advice from A/Prof. Baillie. I was responsible for the inclusion of measures of metacognitions. Data collection was paired with other unique material collected by Ms Subotic; however, these variables are not included here. Ms Subotic and I collected and managed the data. I analysed the data under the supervision of A/Prof. Baillie. I wrote this chapter, with feedback from A/Prof. Baillie and Ms Subotic.

**Chapter 5:** Ms Subotic and I collaboratively designed this study, with advice from A/Prof. Baillie and Dr Stapinski. I uniquely planned the inclusion of salivation, heart rate detection, anxiety sensitivity and elaborated intrusion measurement for this study. Data collection was paired with other unique material collected by Ms Subotic; however, these variables are not included here. Ms Subotic and I collected the data, which I analysed under the supervision of A/Prof. Baillie. I prepared the manuscript with feedback from A/Prof. Baillie and Ms Subotic.

Approval for this research was granted by the Human Research Ethics Committees at Macquarie University (#HE01MAY2009-D06444; #HE26JUN2009-R00026; #5201001453, #5201100016 LAB;), and Sydney South West Area Health Service (#X10-0065 & HREC/10/RPAH/119). Approval letters can be found in Appendix A.

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## **Chapter 1**

### **General Introduction**



## CHAPTER 1: GENERAL INTRODUCTION

This thesis investigates metacognition and cognitive processes underlying alcohol craving. Craving research in previous decades has focused strongly on craving content. Following poor relationships with behavioural outcomes such as relapse rates, inconsistent relationships with physiological measures such as heart rate, and disagreement over conceptual boundaries for nomenclature such as ‘craving’ ‘urge’ and ‘desire’, research interest in the area declined, yet it remained a central construct to addiction. Recently craving has re-emerged as a construct worthy of further investigation (Kavanagh & Connor, 2013), potentially due to conceptualisation of craving as a variable that is part of a broader cognitive process. Modern craving theories conceive of craving as a cognitive, affect-laden event to which individuals respond with higher-level processing such as metacognition and elaboration (Hoyer, Hacker, & Lindenmeyer, 2007; Kavanagh, Andrade, & May, 2005). This thesis explores the possibility that craving metacognition and cognitive processes around craving may deliver on earlier promise of improved understanding of addiction.

This thesis comprises six chapters, four of which are empirical papers prepared for publication. This chapter reviews craving theories and research. Theories of metacognition, craving measurement and cognitive processing are examined, and predictions for a relationship between cravings and metacognitions are provided, along with the aims of this thesis and each of the following chapters. Finally, this chapter outlines the four empirical studies that follow.

Craving is defined by the International Classification of Diseases as ‘a strong desire to take the drug’ (ICD-10; World Health Organisation, 1992, Dependence syndrome section, para. 1), while the Diagnostic and Statistical Manual (DSM-5; American Psychiatric Association, 2000) describes it as ‘a strong desire or urge to use alcohol’ (p. 491). For these classification systems cravings are one of a number of criteria for identifying an alcohol use

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disorder. Cravings are neither a necessary nor sufficient condition for diagnosis, given they can be experienced by non-dependent individuals (e.g., Jones et al., 2013; Rosenberg & Mazzola, 2007). Both ICD-10 and DSM-5 definitions of craving give a highly subjective definition of craving, placing the subjective experience as prominent. Cravings provide an important indication of disorder onset and severity (Keyes, Krueger, Grant, & Hasin, 2010). Pavlick, Hoffman, and Rosenberg (2009) found that clients of 96% of drug and alcohol treatment agencies in the United States asked for assistance with cravings, indicating it remains a cause of considerable concern or discomfort. This is particularly important given dependent drinkers perceive that wanting to drink and not being able to, will lead to consequences such as anger, depression, anxiety, stress, negative thoughts and memories, and embarrassment (Toneatto, 1999b), findings that have been replicated in cue reactivity studies (e.g., Fox, Bergquist, Hong, & Sinha, 2007).

As a subjective experience, craving is an affect-laden thought of using alcohol (Hoyer et al., 2007; Kavanagh et al., 2005). The concern individuals experience about their cravings constitutes metacognition. Metacognitions are the mechanisms and beliefs involved in regulation, processing and evaluation of thoughts (Wells, 2000). Theories of metacognition posit that the evaluations individuals make about their thoughts can have a significant impact on their subsequent wellbeing (Wells & Matthews, 1996). In many metacognitive models, there are two forms: metacognitive regulation (planning, checking, monitoring) and metacognitive beliefs (knowledge of internal states including cognitions and emotions; processes, etc.; Spada & Wells, 2008). Individuals will employ metacognitive regulation when, for example, attempting to direct their attention or concentration, or comparing their thoughts to an ideal of how they feel they “should” think. In contrast, metacognitive beliefs and knowledge include content, attitudes and evaluations of perceptions. Within this form, individuals will hold opinions about their own abilities and their own knowledge.



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Metacognitions may provide a useful framework for understanding craving processes in addiction.

Further cognitive processes implicated in addiction have been explored in recent years. The elaborated intrusion theory of desire (Kavanagh et al., 2005) focuses on the cognitive processing of intrusive, alcohol-related thoughts. In this theory, cravings are said to be initially pleasurable so are mentally expanded upon via imagery and memory recollection. Elaborations provoke negative affect due to a sense of associated deficit, which triggers further elaboration. Individuals' elaborations are said to be cognitively taxing, and can be interrupted via competing cognitive tasks (Andrade, Pears, May, & Kavanagh, 2012). Elaborated intrusion theory provides a number of avenues through which craving processes can be explored.

### **Theories and Models of Craving**

There are four main conceptualisations of cravings: phenomenological, conditioning, neurological and cognitive (Anton, 1999; Drummond, 2001; Skinner & Aubin, 2010). Each identifies a different cause of craving, emphasises a different component of the craving cycle and offers different explanations and predictions for the phenomenon. Examining the focus, strengths and shortcomings of each demonstrates the key features of a strong model of cravings, which at times may be best referred to in plural to reflect the variability in content individuals may experience.

### **Phenomenological Formulations**

Many of the central tenets of modern craving models and theories have been in use for close to a century, yet have their roots in a phenomenological approach. Although Anton credits Jellinek et al. (1955) with first recognising craving as a central component of alcohol

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dependence, the discussion of craving as a symptom associated with heavy alcohol use is considerably older. Initially, physicians wrote observations of cravings as an epiphenomenon of alcohol dependence (e.g., in an oration by Kinsey in 1883; in an open letter from Peddie to fellow physician Bucknill in 1877). These authors spoke of cravings as a compulsion for intoxication, while others discussed cravings as a symptom of alcohol dependence requiring treatment (Harris, 1876) or referred to its role in loss of control over alcohol consumption (Wilks, 1876).

These early conceptualisations of cravings laid the foundations for interest in the ways in which cravings developed. In 1906, Campbell referred to cravings as a learned response, by identifying the relationship between repeated alcohol use under specific circumstances, and that these circumstances themselves later provoked cravings. These modest beginnings to conditioning models arose as Doane (1909) discussed the role of physiological alcohol tolerance on cravings, whereby increasing doses of alcohol are required for craving control or prevention. These publications contain the roots of later models such as classical conditioning and withdrawal models. In 1924, Park asserted that cravings are a component of alcohol dependence, and that it was indeed a key indicator of a ‘pathological condition’ (p. 156). More recently, Drummond (2001) and Addolorato, Leggio, Abenavoli, and Gasbarrini (2005) both cite Modell, Glaser, Cyr, and Mountz (1992) as an example of a phenomenological model, where cravings were described as a subset of OCD symptoms. However, any discussions that are phenomenological by their very nature make limited predictions and are neither directly testable nor generalisable, which is untrue of Modell and colleagues’ work. Accordingly, very few truly phenomenological formulations have been developed. Moreover, phenomenological models provided no explanation for content or processes of craving, nor any explanation for the relationship between craving and other

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constructs. The phenomenological exploration of craving gave rise to new models, as researchers considered the role of conditioning in craving development.

### **Conditioning Models**

According to conditioning models, craving is the result of a learned association between stimuli and response. Early conditioning models such as Wikler's (1948) conditioned withdrawal model argued that craving is a negative and dysphoric learned response. This response mimics physiological withdrawal following the presentation of conditioned stimuli (e.g., seeing a bottle of alcohol). Conditioning models predict that, when craving, the individual learns to use alcohol to alleviate cravings via negative reinforcement.

The conditioned opponent process model provides an alternative interpretation of aversive withdrawal symptoms (Siegel, 1989; Solomon & Corbit, 1973, 1974), whereby dependence on a substance renders the body so accustomed to its presence that its sudden absence results in overcompensation by the body. Here, craving is a homeostatic response whereby the absence of substance produces the opposite effect of the substance itself. While conditioned opponent process models provide valuable insight into craving processes, they neglect to explain the positive valence that can be associated with craving; a piquancy that itself makes the substance all the more enjoyable upon acquisition (Kavanagh et al., 2005). Accordingly, Stewart, de Wit and Eikelboom's (1984) conditioned drug-like model attempts to overcome this limitation by proposing that craving is a learned response that pairs alcohol consumption cues and behaviours with the pleasurable nature of alcohol consumption. In this explanation, positive reinforcement may be responsible for continued use. This certainly enhances the applicability of the model; however, this model and its predecessor both assume that the outcome of reinforcement is continued use, rather than aversion, whereby subsequent alcohol consumption is not a reinforcer but a punisher (Drummond, 2001).

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The two-process theory proposed by Glautier and Remington (1995) does not make this assumption. This theory described processes of both classical and operational conditioning. Conditioned stimuli elicit a multitude of responses including behavioural, emotional and autonomic responses, which in turn may or may not lead to use; that is, conditioning makes drinking either more or less appealing. Craving within the two-process theory is an emotional state that arises from conditioning between cues and substance effects. However, this theory does not provide specific predictions about the circumstances required for a particular response. There is limited information on the processes by which one response may occur over another, both within and between individuals.

Broadly, while conditioning models provide a thorough explanation for the development of craving, and often make strong predictions about the influence of craving on subsequent drinking episodes, these predictions have received only modest support from empirical testing. The relationship between craving and relapse is smaller than anticipated. Moreover, the explanation of individual differences in conditioning models is limited. For example, researchers often use cue reactivity paradigms to test conditioning models, providing strong demonstrations of autonomic processes of physiological responsiveness, albeit at the expense of more subjective experiences. These theories do not explicitly address differences in qualitative content (of which there is some evidence; e.g., Heinz et al., 2003; Kavanagh, May, & Andrade, 2009) or valence of cravings. Conditioning models also do not address inconsistencies of craving occurrence. This includes why some individuals experience cravings when others do not, inconsistent responses to stimulus presentation, craving in apparent absence of any conditioned stimulus (as is reported by some individuals; e.g., May, Andrade, Kavanagh, & Penfound, 2008), and finally why cravings may occur years after abstinence is commenced, long after extinction could reasonably have taken place. Neurological models of craving address some of these criticisms.

### **Neurological Models**

Neurological craving models focus on changes from repeated alcohol administration, on neurons, neurotransmitter release and brain region activation. Many of these models retain links to the conditioning models; for example, Robinson and Berridge (1993, 2001, 2008) proposed their incentive sensitisation theory, which argued that prolonged use of a substance renders the neural system hypersensitive to the substance use incentives. As with conditioning models, learning processes modulate expression of drug-seeking behaviour, such that the individuals only experience sensitisation in conditioned situations. This sensitisation is said to endure for years beyond abstinence. According to Robinson and Berridge, this theory provides the distinction between ‘wanting’ a drug (a craving for relief via substance use) and ‘liking’ a drug (use for pleasure). However, this conceptualisation oversimplifies the role of affect in craving, as it is primarily concerned with the valence of use, rather than the valence of craving itself.

Anton’s (1999) neuroadaptive model builds on incentive sensitisation theory and provides specific information about the role of neural systems. The neuroadaptive model argues that cravings can arise from a number of mechanisms including withdrawal and reward memory, following neuroadaptation along neural pathways between the amygdala (emotions; stress, mood), the nucleus accumbens (reward), the frontal cortex areas (dorsal lateral prefrontal cortex; integration of sensory information, memories) and the basal ganglia (repetitive thought and behaviour patterns). Repeated alcohol consumption may lead to associations between sensory cues of alcohol consumption, reward, and memory of reward, providing the individual with sensitivity to alcohol cues in their environment. Simultaneously, the individual’s potential for relapse increases due to damage to the frontal lobes and specifically executive function capabilities (Anton, 1999) while damage to the amygdala can lead to stronger craving (Wrase et al., 2008). Neurological models of cravings

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are the basis of medication as a therapeutic intervention. However, at best these have had only partial success at alleviating craving (e.g., Maisel, Blodgett, Wilbourne, Humphreys, & Finney, 2013; Richardson et al., 2008; Verheul, Lehter, Geerlings, Koeter, & Van den Brink, 2005), with evidence that there are additional factors contributing to the experience including gender, high levels of anxiety, and more severe dependence (Verheul et al., 2005). Moreover, neurological models can be too reductionistic for adequate explanation of craving as a subjective experience. These models can only crudely explain the content and sensory experience of cravings, with similarly limited opportunities for cost-effective non-medically based interventions.

An attempt to acknowledge such variability in craving occurrence has been made by Verheul and colleagues (Verheul, Van den Brink, & Geerlings, 1999), who produced the three pathway psychobiological model. This model argues cravings can arise for several reasons (e.g., psychological needs, neurochemical dysfunction), and aims to cater to the range of individual experiences by exploring personality styles, neurotransmitter (dys)regulation, and stress reactivity, as they relate to craving. Three types of cravings are proposed. Reward cravings are the desire for benefits of alcohol use, which arise from reward-seeking personality style and/or dopaminergic/opioidergic dysregulation. Relief cravings are the desire for relief from aversive symptoms, which arise from stress reactivity (anxiety sensitivity) and/or gamma-aminobutyric acid-glutamatergic dysregulation. Obsessive cravings are the lack of control over intrusive thoughts about alcohol, which arise from those with disinhibition and/or a serotonin deficiency. To date, research testing the accuracy of this model in predicting appropriate pharmacotherapies is unsupportive (Ooteman et al., 2009; Ooteman et al., 2005). However, some evidence has been found for varied qualities to cravings, consistent with the three-pathway model (Heinz et al., 2003). Finally, despite attempts to integrate the physiology and psychology of craving, the three-pathway model only

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minimally addresses sensory engagement; there is considerable scope for investigation of the somatovisceral features of cravings, yet these receive only cursory consideration. Cognitive models and the processes they describe explore individual experiences of craving via application of conditioning principles combined with greater consideration of the subjective experience espoused by phenomenological models and neglected by neurological models.

### **Cognitive Models**

Cognitive models attempt to explain the idiosyncrasy of craving responses neglected by conditioning and neurological models, by conceptualising craving as thoughts of using alcohol, and exploring the ways individuals think about their experiences. Cognitive models propose constructs and processes that facilitate strong, testable hypotheses about individual differences. However, many cognitive craving constructs share limited relationships with physiological data about cravings. This is particularly problematic when many of these models propose cognitive constructs based on interpretation of physiological symptoms or subsequent processes. For example, an early cognitive model, the cognitive labelling model (Ludwig, Wikler, & Stark, 1974), acknowledges that alcohol conditioning causes certain physiological symptoms (e.g., somatovisceral or cardiac changes). Upon detection, the individual cognitively labels these experiences as ‘cravings’. However, this model fails to explain adequately why some individuals report subjective craving while no objective symptoms are observed. Explanation of subjective and objective cravings is a complex issue. Given the inconsistent relationship found in data, a better explanation of these is likely to be a model that allows the individual to experience either or both, without one being a manifestation of the other.

Baker, Morse and Sherman’s (1987) dual affect model aimed to address the role of affect and cognition on craving. The dual affect model viewed craving as affect indicating

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motivation, with phenomenological, behavioural and physiological correlates. The individual may experience both appetitive (positively valenced) and withdrawal-based (negatively valenced) cravings, but not simultaneously. This is potentially problematic given that, firstly, positive and negative affect related to craving are often positively correlated (Drobes & Tiffany, 1997; Ooteman, Koeter, Verheul, Schippers, & Van den Brink, 2006a). Secondly, a single craving event can have both positive and negative valence, either simultaneously or more sequentially (e.g., Kavanagh et al., 2005).

Tiffany's (1990) cognitive processing model argues craving arises from impeded automated substance use. Presentation of conditioned stimuli activates drug use schemata, and if substance use is hindered (for external reasons, such as substance unavailability, or internal reasons, such as competing interests and motivations), the individual craves. This model has the benefit of including verbal, somatovisceral and behavioural aspects of craving, and for identifying and providing explanation of substance use independent of craving. However, this model does not account for continued craving even during substance use episodes. This model would predict that if individuals consume the substance, cravings abate; maintenance of drinking episodes does not support this. Additionally, the limits to automaticity described in Tiffany's model require further clarification, particularly for individual differences. The model does not sufficiently explain the ways in which some individuals develop automatic processes while others do not. Nor does it explain the point of distinction between automatic cognitive processes, and automatic behavioural processes. Many of Tiffany's examples of automatic behavioural processes involve relatively simple mechanical tasks (e.g., pressing a lever) that do not necessarily extend to more complex tasks like substance use. Tiffany acknowledges that particularly complex tasks may not be fully automatic, and rather that it is the coordination of smaller tasks that is automatised.



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Nevertheless, further information is required to explain thoroughly the mechanisms of craving and substance use as automatic processes.

Expectancy models attempt to explain mechanisms of craving by proposing that individuals hold certain beliefs about alcohol and its effects. Some of these beliefs may be learned vicariously through culture or modelling, while others are conditioned through personal experiences (Marlatt, 1985). Expectancies relate to craving via motivation, whereby an individual who believes alcohol will (for example) alleviate tension or stress will crave alcohol when these affective states are experienced (Tiffany, 1999). Motivational components of expectancy models began to take shape as metacognitive models, whereby craving constituted an evaluation of cognition.

Toneatto (1999b) described cravings as themselves being metacognitions; that is, a cognitive response to cognitions. From this perspective, cravings are a comment on the current mental state relative to the desired mental state; to crave is to crave *for* something, and specifically for change. Toneatto's exploratory research demonstrated that problematic drinkers in a desire state hold a range of perceived effects of consuming alcohol, and consequences of not doing so. Yet, these ideas predominantly focus on aversive mental states that the individual seeks to improve. Cravings for alcohol may instead arise from excitement, celebrating or 'making the good times better'. Moreover, the conceptualisation of cravings as a metacognitive commentary on mental state fails to account for circumstances in which craving appears to have no cause or trigger at all, and when the individual is otherwise occupied. Innovative new theories suggest that processing craving can involve elaborative cycles that account for these limitations.

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Kavanagh and colleagues developed the elaborated intrusion theory of desire (EI; Kavanagh et al., 2005). This innovative theory proposed a process of desire addressing many of the shortcomings of previous cognitive theories. In this theory, desire begins as an intrusion into the individual's stream of consciousness, which is initially enjoyable. As the individual becomes aware of the relative absence of alcohol, this once pleasurable thought becomes an irritation, resulting in ruminations or elaborations about the desire. Elaborations may take the form of memories or imagery of drinking (May, Andrade, Panabokke, & Kavanagh, 2004), and especially multisensory imagery involving gustatory (tasting, swallowing), olfactory and visual sensations (Murray, 2008).

EI theory argues that as these elaborations are cognitively taxing, providing a competing cognitive task produces a reduction in craving. Some success in reducing these elaborations and cravings has been obtained by breaking the elaborative loop. Andrade et al. (2012), Kemps and Tiggemann (2009), and Versland (2006) have all reduced research participants' cravings for a range of substances by providing a competing cognitive task for individuals to complete. This competing task is thought to reallocate some of the attentional resources that are consumed by elaborations, and is consistent with the frequent anecdotal reports by those recovering from alcohol use disorder that they are at greater risk of drinking when bored or unoccupied.

However, EI theory contains a paradox. Research testing the theory has demonstrated the cognitively competing tasks can interrupt the elaborative process. The theory does not specify what happens when the individual directs their attention inward, to perceivable physiological correlates of craving. Postulation for either an increase or a decrease in craving is reasonable within the theory; accordingly, further clarification is required. Individuals with internally directed attention who experience an increase in craving will demonstrate evidence

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for an elaborative process that is enhanced via interoceptive ability; individuals who experience a decrease in craving will demonstrate evidence for attention-based interventions.

Craving theories developed to date have progressed substantially through close to a century of research. Much of this research has been dedicated to investigating craving cause and manifestation, which enables researchers to address two key concerns. Firstly, researchers can develop treatments to reduce craving discomfort that can contribute to distress and further mental health complications (Nosen, 2012). Secondly, researchers can determine the relationship between craving and further substance use. This has had varied success to date (cf. Becker, 2008; Tiffany & Carter, 1998; Tiffany & Conklin, 2000). Notwithstanding issues of measurement accuracy, any relationship between craving and substance use may be either mediated or confounded by other unmeasured variables. Recently, research attention has turned to metacognitions and metacognitive therapy as a potential solution to both of these broad research aims.

### **Metacognitive Theory: Useful for Addictions?**

Metacognitive theory argues that the way an individual evaluates their cognitions influences development and maintenance of psychological disorders (Wells, 2009). Metacognitive regulation and knowledge can be positive or negative. A positive metacognition may be an individual's belief that certain thoughts are beneficial or adaptive; for example, that thinking about alcohol helps the individual to avoid drinking. In contrast, some metacognitive beliefs are negative, such as that craving will be unbearable or make the individual lose control. Thus, metacognitions may influence an individual's craving experiences.

Individuals' perceptions of their own experiences have been shown to have an influence in a range of areas such as depression (Papageorgiou & Wells, 2003) and hypochondriasis (Bouman & Meijer, 1999). Metacognitions are important for alcohol research as they may help explain cycles of relapse (Spada, Moneta, & Wells, 2007; Spada & Wells, 2005), and different metacognitive beliefs may occur at different stages of the alcohol use process. For example, Spada, Caselli, and Wells (2012) identify that in a pre-alcohol use phase, positive metacognitive beliefs about extended thinking and negative metacognitive beliefs about a need to control thoughts can contribute to repetitive thought patterns that contribute to craving.

### **Metacognitions and Cravings**

Recent formulations of craving have highlighted its cognitive nature, whereby drinking cues activate alcohol use schemata (Tiffany, 1990) and ruminative, affectively-charged thoughts of alcohol use (Kavanagh et al., 2005). Conceptualising cravings as cognitive suggests that belief and knowledge that individuals hold about their cravings are metacognitive. Craving metacognitions may include antecedents and consequences of craving, what the process of craving is like, how it could or should be alleviated or avoided, what it means to crave, or what craving says about them as an individual.

Metacognitions about cravings offer researchers and clinicians opportunities to work with clients on two fronts. Firstly, craving metacognitions predict relapse (Türkçapar, Kose, Ince, & Myrick, 2005), and metacognitive therapy reduces likelihood of relapse across a number of substances in a number of formats (e.g., Grant, Kunic, MacPherson, McKeown, & Hansen, 2003; Lee, Pohlman, Baker, Ferris, & Kay-Lambkin, 2010; Nosen & Woody, 2014). Secondly, metacognitive therapy teaches participants detached mindfulness, or acknowledging cognitions (in this case, cravings) without acting on them (Wells, 2009). This

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may reduce discomfort in the long term by breaking cycles of reinforcement that perpetuate harmful behaviours. Participants who are able to cease both positive and negative reinforcement of craving improve their chances of having fewer craving episodes.

Application of more general metacognitive theory would suggest that individuals' evaluations and beliefs about their cravings would influence their mood and behaviours. Craving-specific metacognitions may be valuable as a separate construct to general metacognitions due to the specificity of the cognition. Metacognitions may vary depending on the types of cognitions initially experienced; accordingly, craving metacognitions may also vary depending on the initial craving. Nosen (2012) found only moderate correlations between a purported measure of general metacognitive (Metacognition Questionnaire; Wells & Cartwright-Hatton, 2004) and the Appraisals of Craving Questionnaire (Nosen & Woody, 2009), suggesting a distinction between craving-specific and general metacognitions. Research to date indicates that substance-dependent individuals tend to hold metacognitions relating to craving's unpleasantness, usefulness, uncontrollability and personal significance (Hoyer et al., 2007; Nosen & Woody, 2014).

Further distinction between types of alcohol-related metacognitions can be made between drinking metacognitions and craving metacognitions. Spada and Wells (2008) described a range of positive and negative beliefs about alcohol use when developing two measures, the Positive Alcohol Metacognitions Scale (PAMS) and the Negative Alcohol Metacognitions Scale (NAMS). These measures focus on the effect of alcohol on cognition, whereas craving metacognitions (Hoyer et al., 2007; Lee et al., 2010; Nosen & Woody, 2014) address individuals' beliefs about cravings. Both types of metacognitions are likely to be involved in processes that maintain drinking behaviours.

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Models of metacognitions have not yet explicitly addressed craving metacognitions. Spada et al. (2012) developed the triphasic model of problem drinking, which is not specifically a craving model, but describes the metacognitive beliefs involved pre-, during and post-alcohol use. This model proposes the individual experiences a trigger, which may be a craving, image, memory or thought of alcohol, which provokes positive metacognitive beliefs, such as ‘Alcohol will make me feel good’. Individuals with a certain metacognitive style (termed Cognitive Attentional Syndrome by Wells & Matthews, 1994) experience an increase in craving and consequent negative affect, followed by reinforcement of negative metacognitive beliefs about drinking (e.g., about the adversity of not drinking and the need to control thoughts), potentially leading to further consumption. While drinking, alcohol-impaired metacognitive abilities activate positive alcohol metacognitions (e.g., perceived increased coping skills due to alcohol) while reducing the individual’s ability to effectively regulate their alcohol use and cognitive processes. In the aftermath of alcohol use, positive metacognitive beliefs (e.g., about the value of rumination about alcohol) are again activated and subsequently lead to further negative metacognitive beliefs, recommencing the cycle. Repeated cycles of alcohol-use episodes reinforce negative metacognitive beliefs about alcohol use, especially those about its uncontrollability. The triphasic model offers good explanation for craving and alcohol use cycles, and differentiates well between individual responses (those with CAS compared to those without). Yet, it remains focused on the individual's perception of alcohol effects. Perceived uncontrollability refers to uncontrollable alcohol use, rather than uncontrollable craving.

Given a lack of a specific craving metacognition model, predictions may be drawn from other metacognition models. Cyclical relationships like the ones described in the triphasic model seem likely, whereby a cue (internal or environmental) provokes an initial craving. The individual’s metacognitive response to this initial craving may amplify craving.

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Alternatively, stimuli may directly activate the metacognition, like schemata, whereby the individual is then primed for craving.

The veracity of the proposed relationship between cravings and metacognitions is unknown. Previous studies have exclusively used self-report questionnaires to measure metacognitions. Metacognitions have never been measured under controlled or experimentally-manipulated conditions. In many metacognition studies, the participant's current level of craving was not measured and participants were asked their beliefs about cravings that occurred in the unspecified past (Chang et al., 2011; Grant et al., 2003; Hoyer et al., 2007; Sims et al., 2002; Toneatto, 1999a). Thus, the finer nuances of the craving experience may be lost in retrospective reporting (Krahn, Bohn, Henk, Grossman, & Gosnell, 2005; Shiffman, 2009). An exception is the work by Nosen (2012), who used ecological momentary assessment and found that nicotine cravings did not predict changes in metacognitive evaluations, but rather contributed to negative mood, which in turn exacerbated unhelpful metacognitions. Nevertheless, metacognitions have never been measured during a controlled cue reactivity study, such as exposure to substance of choice or craving induction via imagery. As such, the directionality of relationships between cravings and metacognitions remains unknown; the variability in craving experiences ensures this is a complex issue.

Craving metacognition research has further been limited by a lack of studies investigating the effect of in situ cravings and craving metacognitions. Cue reactivity paradigms are often used to measure the effect of craving on a range of variables (Carter & Tiffany, 1999; Drummond, 2000). Based on conditioning models, cue reactivity methodologies propose that presentation of conditioned stimuli for alcohol use will provoke craving. Stimuli may include olfactory cues (smelling alcohol; Miranda et al., 2013), visual cues (viewing images of alcohol brands or people drinking; Garland, Carter, Ropes, &

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Howard, 2012) or imaginal cues (Versland & Rosenberg, 2007); these have consistently shown reliable induction of craving for a range of substances and populations (Carter & Tiffany, 1999). Studies employing cue reactivity methods have included substance administration, often to observe the effects on individuals' cognitions (Eddie et al., 2013) and emotions (Snelleman, Schoenmakers, & van de Mheen, 2014).

Research into the effect of alcohol on craving metacognitions is lacking. This is an essential component of craving metacognition research: two important phases of the alcohol consumption process are the initiation of consumption, and the continuation of consumption. Cravings in the initiation phase have been extensively researched (e.g., Miranda et al., 2013; Ramirez & Miranda, 2014), often using cue reactivity paradigms; however, the role of craving in maintenance of alcohol consumption behaviours is less well understood. Craving metacognitions have not been adequately included in either type of research.

One potential way of elucidating the relationship between cravings, metacognitions and alcohol consumption is by using real versus placebo alcohol. Studies of this nature have reliably shown that small amounts of alcohol will increase craving (Hutchison, McGeary, Smolen, Bryan, & Swift, 2002; Hutchison et al., 2001; Ludwig et al., 1974) and will not substantially interfere with cognitive function (Kennedy, Turnage, Wilkes, & Dunlap, 1993; Pihl, Paylan, Gentes-Hawn, & Hoaken, 2003). A placebo alcohol group will control for expectancy effects. A central tenet of cue exposure research is that cues activate cravings, yet the effect of cues on metacognitions is to date unknown. There has been no research on the impact of alcohol cues (such as a priming dose of alcohol, or environmental cues) on metacognitions.



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Testing the role of cue exposure in activating cravings and metacognitions enables measurement of physiological correlates of craving. Measuring these variables provides a valuable breadth of craving measurement, particularly given individuals who do not report subjective cravings may still respond to cues physiologically (Szegedi et al., 2000). Cognitive processes related to interoceptive sensitivity and physiological indicators of craving may inform craving, metacognition and addiction research.

### **Aims of This Thesis**

This thesis aims to determine some of the variables related to craving metacognitions, via triangulation of methods and procedures. It aims to understand the role of metacognitions and cognitive processes in cravings. These aims will be pursued via investigation of the relationships between cravings, metacognitions, elaborated intrusions and measures of interoceptive accuracy.

Metacognitive models are a promising link between cravings and substance use, potentially contributing to the relationship between craving use and relapse. They propose that the individual's responses to substance-related thoughts are predictive of substance use and addiction recovery (Spada & Wells, 2005). Preliminary investigations of metacognitions have partially confirmed these predictive abilities (Lee et al., 2010; Türkçapar et al., 2005), and research to date indicates unhelpful metacognitions can be reduced via therapy (Grant et al., 2003; Loeber, Croissant, Heinz, Mann, & Flor, 2006).

Beyond these findings, a number of questions remain unanswered. Known influences on craving include alcohol use history (Day, Celio, Lisan, & Spear, 2014), cues (Litt et al., 2000), and time of day, albeit inconsistently (Rohsenow & Marlatt, 1981). Known influences on craving metacognitions only extend to level of alcohol use (abstinent, social or dependent

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drinker; Aslan, Türkçapar, Eser, & Uğurlu, 2012), smoking cessation experiences (Nosen & Woody, 2009), and psychotherapy (Lee et al., 2010; Loeber et al., 2006). Similarly, craving metacognitions are more often investigated for their ability to predict other outcomes such as treatment success (Grant et al., 2003) including abstinence (Lee et al., 2010; Türkçapar et al., 2005). Research has yet to determine how cravings and metacognitions co-vary, or how they influence one another.

The most recent craving/metacognition findings relate to nicotine cravings (Nosen & Woody, 2009, 2014). Some of these have shown highly specific relationships (e.g., cravings are related to metacognitions of personal relevance; Nosen & Woody, 2014). The existence of such specific relationships for alcohol cravings and craving metacognitions is unknown. Relationships may not have been found due to limitations with metacognition measurement. For example, development of one craving metacognition measure was deliberately restricted to a subset of metacognitions perceived to be clinically relevant (Metacognitions Questionnaire for Alcohol Abusers; MCQ-A; Hoyer et al., 2007). Another metacognition measure exclusively addressed craving antecedents (Lübeck Craving-Recurrence Risk questionnaire; Veltrup, 1994). Broader conceptualisation of metacognitions may result in heretofore-unknown relationships between the broader construct and craving.

Finally, craving metacognition research to date has relied heavily on self-report cravings. The relationship between physiological measures of craving and metacognition has not yet been explored. Exploration of the full spectrum of cravings and metacognitions is required to determine the nature of these relationships. Limiting the measurement of either variable inhibits our ability to understand it. There is evidence that some individuals are more physiologically reactive to certain alcohol cues, despite reporting few subjective cravings (Szegeedi et al., 2000). Although we conceive of cravings as cognitive, physiological

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indicators of craving may contribute to our understanding of craving as a construct. As we are unsure of the nature of this relationship, a relationship between physiological reactivity and metacognitions may indicate a degree of interoceptive sensitivity that could improve our understanding of both constructs. The present thesis addresses some of these under-researched areas.

### **Chapter Summaries**

#### **Chapter 2: Treatment Seekers' Cravings Activate Specific Metacognitions in a Placebo-Controlled Alcohol Consumption Task**

The purpose of this study is to test the relationship between alcohol cravings and craving metacognitions under different cue exposure conditions. Metacognitive theory proposes that cognitions relate to metacognitions. Additionally, previous studies have shown cravings for nicotine share relationships with specific metacognitions (Nosen & Woody, 2014). Yet, no study to date has manipulated craving via varied cue presentation and subsequently measured metacognitions. Accordingly, I proposed that cravings differentially predict metacognitions as a function of cue exposure. I hypothesise that those given a priming dose of alcohol will produce the strongest metacognitions, followed by those expecting to but not actually receiving alcohol, followed by those administered soft drink.

#### **Chapter 3: Development and Initial Evaluation of the Craving Metacognition Scale (CMS)**

Few measures of craving metacognitions address a comprehensive range of craving metacognitions. Additionally, many measures are only developed with participants with a narrow range of consumption. The second study reported in this thesis describes the development and testing of a new measure of craving metacognitions, the Craving

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Metacognition Scale. This measure includes craving antecedents and consequences, where items were created based on semantic content extracted from existing craving measures. The questionnaire was developed with samples of inpatient alcohol treatment seekers and undergraduate students who drank regularly. I aimed to develop an internally consistent, parsimonious measure for testing the relationship between cravings and craving metacognitions.

### **Chapter 4: Metacognitions Relate to Cravings Following Cue Reactivity**

After examining craving prediction of metacognitions and developing an internally consistent measure of craving metacognitions, I aim to test the strength of the relationships between metacognition measures (CMS and MCQ-A) and alcohol cravings. Here, I aim to trigger metacognitions through alcohol cue exposure. I test the effect of craving metacognitions on subjective (AUQ) and objective (heart rate and galvanic skin response) measurements of cravings. I hypothesise that craving metacognitions will predict stronger craving when participants are exposed to alcohol cues compared to water cues. I further expect that these relationships will be stronger for the CMS compared to the MCQ-A.

### **Chapter 5: Interoceptive Accuracy Task Interferes With Rather Than Amplifies Craving in a Cue Reactivity Task**

Relationships between craving metacognitions and cravings are demonstrably highly specific and at times inconsistent, indicating that craving content may be less important than craving process. Accordingly, I examined alternative theoretical explanations. Following the use of cue exposure tasks in Chapters 2 and 4, I applied a similar methodology to test the effect of interoceptive ability on craving. Consistent with elaborated intrusion theory as a

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theory of craving process, I predicted individuals would crave less following an intrusive interoceptive task.



## **Chapter 2**

### **Treatment Seekers' Cravings Activate Specific Metacognitions in a Placebo-Controlled Alcohol Consumption Task**

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### **Abstract**

Metacognitions about cravings may amplify or reduce cravings; however, there is limited research on the craving-metacognition relationship. The present study investigated the relationships between alcohol cravings and associated metacognitions. We propose that, as a cognitive experience, cravings activate metacognitions. We induced cravings in alcohol-dependent individuals by manipulating environmental, cognitive and pharmacological drinking cues, to activate metacognitions. In this complex-design study, 39 participants (mean age = 36 years,  $SD = 10.75$ ; 19 female, 20 male) seeking treatment for alcohol use disorders were randomly allocated to one of three groups who consumed either vodka, placebo alcohol or soft drink, rating their cravings and metacognitions before and after drinking. We observed a main effect for craving but no interaction with beverage type, and no significant changes in whole-measure metacognitions score. However, significant specific metacognition subscale effects were found. Participants who believed they had consumed alcohol found cravings more useful. Alcohol-group participants with high pre-drink Unpleasantness subscale metacognitions found cravings more unpleasant following beverage consumption. These findings suggest cravings and metacognitions are not predictive of one another. However, craving metacognitions may be responsive to changes in alcohol expectancies and consumption.

*Keywords:* Craving, metacognition, alcohol, drinking, alcohol administration, cue reactivity



### **Treatment Seekers' Cravings Activate Specific Metacognitions in a Placebo-Controlled Alcohol Consumption Task**

Although considerable research has focused on cravings for alcohol (cf. Kavanagh & Connor, 2013; Kavanagh et al., 2013; Kozlowski & Wilkinson, 1987; Rosenberg, 2009; Tiffany & Wray, 2012) and beliefs about drinking (e.g., Clark et al., 2012; Spada & Wells, 2005, 2008), less is known about the beliefs, appraisals and evaluations (metacognitions) about cravings. As a feature of alcohol dependence (American Psychiatric Association, 2000), cravings can be indicative of dependence severity. They can be distressing for individuals attempting abstinence (Pavlick et al., 2009) and are arguably linked to relapse (cf. Witkiewitz & Marlatt, 2008).

Craving definitions and conceptualisations vary (cf. Chapter 1). As a cognitive construct, they have been described as 'highly intense alcohol-related thoughts' (Hoyer et al., 2007; p. 822), which are intrusive and affectively charged (Kavanagh et al., 2005). They are triggered by environmental, cognitive, emotional and/or pharmacological cues (Carter & Tiffany, 1999; Drummond, 2000; A. Jones, Rose, Cole, & Field, 2013; Lieb et al., 2013). Previous research has investigated craving by manipulating these cues (Versland & Rosenberg, 2007) and measuring subsequent behaviours (e.g., Kruse et al., 2012), emotions (e.g., Kavanagh et al., 2006; ) and cognitive events (e.g., Garland, Gaylord, Boettiger, & Howard, 2010). Research into cognitive events related to craving mostly focuses on beliefs about the outcomes of substance use (e.g., Monk & Heim, 2013; Toneatto, 1999b), rather than their beliefs about the craving per se (e.g., Hoyer et al., 2007).

Cognitive responses to automatic thoughts such as cravings are termed metacognitions. Examples include 'I crave too much' or 'Craving means I am crazy'.

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Metacognitions can be positive (the individual views the cognition as helpful, healthy or adaptive) or negative (the individual considers the cognition harmful, dangerous or problematic). Metacognitive models have an obvious appeal. Beliefs that cravings are dangerous, permanent or indicate a serious deficit are likely to amplify cravings via cycles involving negative affect (Nosen, 2012; Nosen & Woody, 2014), while beliefs that cravings are transient and inconsequential are not. As beliefs, metacognitions may be trait-like but changeable, although limited evidence exists on their nature beyond rudimentary findings, and such features may depend on how and with what tools they are measured. Metacognitive processes in craving are under-researched, yet may provide valuable insight into the addiction experience. Given the paucity of research in this area, research from other areas may provide direction.

Metacognitive theory suggests cognitions and metacognitions occur in cycles, so are mutually influential (e.g., the cognitive-attentional syndrome; Wells et al., 2009), and that metacognitions are latent until activated by relevant cognitions. Briefly, the individual experiences a particular cognition (e.g., ‘I want to drink’) that activates relevant metacognitions. These metacognitions can involve evaluation of the cognition (Is this craving good or bad? How personally relevant is it? What are the implications?), and compete with information processing resources (Koriat, 2007) that allow the individual to deal with the stimuli effectively (Wells, 1995). Unhelpful metacognitions may result in negative affect including low mood or distress (Nosen, 2012). The individual may subsequently monitor for similar cognitions or supporting evidence, leading to increased sensitivity and a confirmation bias, at which point the cycle begins again (Wells & Matthews, 1996). Altering unhelpful metacognitions may aid the individual in processing information and consequently limit the associated negative affect (Koriat, 2007). This cycle is purported to occur for cognitions in a number of disorders, so may be applicable to cravings.

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Metacognitions about craving are proposed to follow the same processes. For example, an individual perceives a cue that triggers a craving. Cues can be internal (such as sensations, emotions, memories, or expectancies about alcohol) or external (such as seeing or smelling alcohol or alcohol paraphernalia). Craving is primarily cognitive; an affect-laden thought of using alcohol. Craving activates metacognitions – beliefs and evaluations about the craving itself – which may include thoughts of the craving’s valence, worth, meaning, controllability, antecedents or consequences. The experience of craving is then amplified or reduced depending on the valence and strength of metacognitive appraisal.

Only one study has examined the basic assumption that cravings provoke metacognitions. Nosen and Woody (2014) found small but significant relationships between nicotine craving strength, catastrophic appraisals of those cravings, assessed by the Catastrophic Appraisals Inventory, and negative general metacognitions (danger, uncontrollability) using the Metacognitions Questionnaire (MCQ-30; Wells & Cartwright-Hatton, 2004). Areas other than craving have also demonstrated relationships between cognitions and metacognitions (e.g., anxiety and depression, Wells, 2009; schizophrenia, Moritz, Veckenstedt, Randjbar, Vitzthum & Woodward, 2011). No research has been conducted yet for alcohol cravings, although a similar relationship seems likely given the findings above.

Despite the lack of a craving-specific metacognition theory, existing craving metacognition research has produced a number of interesting results. Firstly, there is evidence that craving metacognitions covary with dependence. Aslan, Türkçapar, Eser and Uğurlu (2012) reported that alcohol abstainers and social drinkers held comparable craving beliefs measured by a self-report questionnaire, the Craving Beliefs Questionnaire (CBQ,

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Beck et al 1993), which were significantly different to beliefs held by alcohol-dependent participants.

Secondly, the inclusion of metacognitions in treatment outcome studies has demonstrated that craving metacognitions are amenable to change, which is consistent with conceptualisations of metacognitions in other areas of research (e.g., generalised anxiety disorder, Wells, 1995; depression and anxiety, Normann, van Emmerik & Morina, 2014). Grant et al. (2003) noted a reduction in craving beliefs measured by the CBQ following a substance abuse treatment program for prison inmates; Loeber et al. (2006) achieved similar success using cognitive behavioural therapy for addiction. However, neither of these studies explored the relationship between craving and metacognition.

Craving metacognition scores are related to clinical outcomes. CBQ scores predicted abstinence success with methamphetamine users following treatment (Lee et al., 2010), prompting a call for further research into the relationship between cravings and metacognitions. Comparable results may have been found by Türkçapar et al. (2005). Sixty-nine alcohol-dependent men received inpatient treatment. Türkçapar and colleagues followed them up six months later, measured relapse rates and attempted to establish variables that predicted relapse. However, the results of this study are unclear: mean CBQ scores for abstainers were reported as significantly higher than those of relapsers in Table 1 of their publication, yet the text reported the opposite, "...patients who relapsed had higher scores on the CBQ (Table 1)" (p. 850). Clarification of this finding is required to determine the direction of the results; however, this study seems likely to be consistent with Lee et al. based on other findings reported in the study by Türkçapar et al.

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Most studies of craving metacognitions have not included craving measures, despite many studies being treatment-focused (Aslan et al., 2012; Chang et al., 2011; Grant et al., 2003; Lee et al., 2010; Nosen & Woody, 2009; Sims et al., 2002; Türkçapar et al., 2005). Craving management coupled with craving beliefs treatment may influence treatment outcomes; hence, measurement of craving levels would have added considerable strength to these studies. If cravings and metacognitions share a relationship, cravings should be controlled for within studies. If they are not related, this stimulates further questions of the variables that do influence craving metacognitions.

Research that has measured both cravings and metacognitions has produced only highly specific results. For example, Nosen (2012) used a version of the Obsessive Compulsive Drinking Scale (OCDS; Anton, Moak & Latham, 1995) for smoking, to investigate nicotine cravings during smoking cessation. Their study found that cravings were significantly positively correlated with the perceived personal significance of craving appraisals (e.g., ‘This thought means I am a bad person’). Additionally, craving appraisals predicted whether participants were successfully abstinent or still smoking at one-month follow-up, beyond prediction by initial smoker status, years of smoking and other variables. Nosen and Woody further found that obsessional subscale cravings for nicotine did not significantly correlate with thought suppression metacognitions (e.g., ‘I preferred not to think about smoking’). Nevertheless, these findings suggest craving metacognitions are an important part of craving treatment.

Specific relationships between craving and metacognition were also reported by Hoyer et al. (2007), who measured alcohol craving using an amended OCDS, with items measuring duration, frequency, interference, distress, and strength of craving, along with effort and success in resisting and distress at abstaining. Hoyer et al. found significant, moderate,

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positive correlations between the amended OCDS and two subscales of their measure, the Metacognition Questionnaire for Alcohol Abusers (MCQ-A). Subscales Thought-Action Fusion and Unpleasantness were significantly correlated with the OCDS  $r = .52$  and  $r = .34$  respectively. However, the correlation between the MCQ-A Subjective Utility subscale and the OCDS was not significant ( $r = .07$ ). Collectively, these indicate a relationship between the manifestations of craving (for example, more frequent cravings co-occur with stronger maladaptive metacognitions), and suggest that the types of craving and metacognition influence the relationship.

Both Nosen and Woody (2009) and Hoyer et al. (2007) show specific craving-metacognition relationships using the OCDS (Anton et al., 1995). However, the OCDS primarily measures craving frequency, duration and interference. Measuring the frequency of a phenomenon should not be confused with the phenomenon itself (Kavanagh et al., 2013). Although specific relationships were found with the OCDS, these relationships cannot comment on potential relationships between the content of cravings and metacognitions. Moreover, measures like the OCDS address a past rather than current craving episode, increasing potential for confounding influences such as memory bias. To measure the effects of craving and metacognition on one another adequately, the relationship must be tested using a craving measure with an immediate timeframe.

In sum, research has demonstrated correlational relationships between a range of metacognition measures and some craving measures. Measurement of cravings has been restricted to a tool that does not focus on the content of craving. A metacognitive model of craving would be beneficial to addiction treatment, as there is evidence that metacognitions are variable and can be changed via therapy. Yet, there is limited information on the



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relationship between craving and metacognitions, and no study has attempted to determine influences on this relationship.

We attempt to build on previous work in three key ways. Firstly, we tested implications of general metacognitive theory that craving metacognitions are activated by cravings. Secondly, this study attempted to control the variability of craving triggers by manipulating the participants' environment, expectations of alcohol consumption, and actual alcohol consumption. Finally, the study measured in situ craving using a briefer measure with higher face validity in a controlled environment.

The present study aims to investigate whether changes in cravings result in changes in metacognitions in an alcohol-dependent sample, following environmental triggers and a priming dose of alcohol. We proposed that cues activate cravings, which activate metacognitions. We manipulated the cues presented between three groups – a soft drink control, a drink participants believed contained alcohol but was in fact a placebo, versus a dose of alcohol estimated to give BAC of 0.05, to trigger cravings differentially. Greater craving is more likely to activate metacognitions; therefore, we predicted metacognitions would be strongest for the participants who drank actual alcohol, then in the placebo group, and weakest in the control group. If these conditions were met, we hypothesised that pre-drink cravings would independently predict post-drink metacognitions. We predicted a similar relationship for metacognitions: that pre-drink metacognitions would independently predict post-drink cravings

### Method

#### Participants

**Recruitment and screening.** Participants were recruited from a clinical trial comparing treatments for comorbid alcohol use disorders and social phobia (Baillie et al., 2013). Participation in the present research occurred prior to treatment commencement. All individuals had received DSM-IV diagnoses of alcohol use disorder (AUD). Individuals experiencing psychosis and substance use disorder for substances other than caffeine or nicotine were excluded. For participant safety, we further excluded those with: i) current suicidal intent; ii) medical conditions for which alcohol consumption was contraindicated; iii) current pregnancy or breastfeeding; iv) risk of severe withdrawal (a score of 20 or higher on the CIWA-Ar; Sullivan, Sykora, Schneiderman, Naranjo, & Sellers, 1989); v) an abstinence treatment goal. Participants were further only eligible if they were aged over 18 years and had in the past two months consumed an alcohol dose equivalent to the dose given in this study, which was calculated using gender-specific formulae (Curtin & Fairchild, 2003). Thirty-nine participants were involved (19 female, 20 male), aged 20-59 years (mean age = 36 years,  $SD = 10.75$ ). Twenty-four participants received a primary diagnosis of alcohol use disorder; the remainder had a primary diagnosis of social phobia. Group demographics appear in Table 2.1.

Table 2.1

*Group Demographics, and Means and Standard Deviations of Caffeine and Food Consumption, Dependence Severity and Craving*

Group	N	Age range in years (M, SD)	Primary diagnosis of AUD (n) <sup>1</sup>	AUD /SP only (n)	Mean # diagnoses	# Drinking days of past 7: M (SD)	# Drinks in past 7 days: M (SD)	Caffeine in mg today: M(SD)	Hours since caffeine consumption: M (SD)	Hours since last meal	SADQ score M (SD)	PACS M (SD)	OCDS M (SD)
<b>Total</b>	<b>39</b>	<b>20-59</b> <b>(36, 10.75)</b>	<b>24</b>	<b>10</b>	<b>3.36</b> <b>(1.25)</b>	<b>3.28 (2.21)<sup>a</sup></b>	<b>27.57 (23.56)<sup>a</sup></b>	<b>132.22 (361.93)</b>	<b>4.94 (1.56)<sup>b</sup> (n = 30)</b>	<b>3.63 (1.08)</b>	<b>25.53 (12.61)<sup>a</sup></b>	<b>15.68 (5.70)<sup>a</sup></b>	<b>18.68 (6.10)<sup>a</sup></b>
<b>Alcohol</b>	<b>12</b>	<b>21-57</b> <b>(33, 10.83)</b>	<b>9</b>	<b>4</b>	<b>2.75</b> <b>(0.62)</b>	<b>4.17 (2.59)</b>	<b>44.67 (30.41)</b>	<b>160.82 (129.88)</b>	<b>4.70 (1.03) (n = 10)</b>	<b>4.25 (1.12)</b>	<b>24.76 (9.63)</b>	<b>14.75 (5.85)</b>	<b>18.17 (7.67)</b>
Male	9	21-57 (32, 11.62)	6	4	2.67 (0.71)	3.56 (2.70)	38.11 (30.48)	155.37 (148.48)	4.43 (0.98) (n = 7)	4.11 (1.17)	24.13 (10.78)	13.33 (5.57)	15.33 (6.28)
Female	3	26-45 (36, 9.54)	3	0	3.00 (0.00)	6.00 (1.00)	64.33 (24.42)	177.17 (63.68)	5.33 (1.04) (n = 3)	4.67 (1.04)	26.67 (6.11)	19.00 (5.29)	26.67 (4.62)
<b>Placebo</b>	<b>13</b>	<b>24-59</b> <b>(37, 13.23)</b>	<b>7</b>	<b>2</b>	<b>3.69</b> <b>(1.32)</b>	<b>2.77 (2.01)</b>	<b>15.38 (11.47)</b>	<b>98.97 (88.38)</b>	<b>5.08 (1.60) (n = 10)</b>	<b>3.36 (0.72)</b>	<b>26.92 (16.20)</b>	<b>16.62 (6.71)</b>	<b>19.54 (5.55)</b>
Male	5	26-56 (39, 13.76)	3	1	3.40 (1.14)	2.40 (2.07)	21.40 (13.24)	134.82 (86.18)	4.40 (1.92) (n = 5)	3.50 (0.87)	31.80 (5.54)	18.60 (6.31)	20.80 (4.97)
Female	8	24-59 (36, 13.74)	4	1	3.88 (1.46)	3.00 (2.07)	11.62 (9.13)	76.56 (87.49)	5.75 (0.97) (n = 5)	3.27 (0.67)	23.88 (20.12)	15.38 (7.07)	18.75 (6.07)
<b>Control</b>	<b>14</b>	<b>20-50</b> <b>(36, 8.23)</b>	<b>8</b>	<b>4</b>	<b>3.57</b> <b>(1.45)</b>	<b>2.96 (1.94)<sup>a</sup></b>	<b>23.98 (16.28)<sup>c</sup></b>	<b>138.59 (118.70)</b>	<b>5.05 (2.02) (n = 10)</b>	<b>3.36 (1.17)</b>	<b>24.84 (11.81)<sup>c</sup></b>	<b>15.62 (4.68)<sup>c</sup></b>	<b>18.31 (5.38)<sup>c</sup></b>
Male	6	20-43 (34, 8.73)	3	1	4.00 (1.27)	2.67 (2.34)	23.93 (23.41)	102.01 (100.32)	6.38 (2.69) (n = 4)	3.5 (1.38)	26.65 (12.54)	15.00 (7.07)	20.33 (7.37)
Female	8	30-50 (37, 8.12)	5	3	3.25 (1.58)	3.21 (1.68) <sup>b</sup>	24.03 (8.56) <sup>d</sup>	166.02 (130.28)	4.17 (0.82) (n = 6)	3.25 (1.07)	23.29 (11.91) <sup>d</sup>	16.14 (1.22) <sup>d</sup>	16.57 (2.23) <sup>d</sup>

<sup>1</sup> All participants had comorbid social phobia; for some participants this was their primary diagnosis.<sup>a</sup> n = 38<sup>b</sup> n = 30<sup>c</sup> n = 13<sup>d</sup> n = 7

### **Procedure**

**Beverage groups.** Participants were randomly allocated by computer to receive alcohol, placebo alcohol or soft drink to consume. We calculate alcohol quantities with the aim of producing a Breath Alcohol Concentration (BrAC) of .05g of alcohol per 210L of breath, which is equal to 0.05g alcohol per 100mL of blood (blood alcohol concentration; BAC). Beverage calculations were derived from gender-specific formulae developed by Curtin and Fairchild (2003) with consideration of body water levels (Watson, 1989). Placebo and control conditions were formulated to produce a comparable total quantity of beverage to the alcohol condition. Recommendations by Rohsenow and Marlatt (1981), and Martin and Sayette (1993) were included to increase the likely success of the placebo administration. These were: i) the participant observed beverage preparation, and placebo alcohol was poured from a commercially labelled, clean vodka bottle; ii) 5ml of vodka was added to the surface of each beverage to provide sensory cues associated with alcohol (McKay & Schare, 1999); iii) during drinking and absorption, all participants watched a neutral DVD of nature scenes set to classical music, to minimise attention to interoceptive cues of alcohol consumption; iv) participants in the placebo and alcohol groups were provided with a BrAC reading fixed to 0.053%.

**Ethical considerations.** Due to the population under consideration, there were particular ethical considerations. Previous alcohol administration studies have often excluded alcohol-dependent participants to reduce risks to participants' safety and likelihood of participating in treatment; however, the resulting research is unrepresentative of the alcohol dependent treatment-seeking population. Moreover, there is limited evidence that participation in alcohol administration studies poses a significant risk to alcohol dependent participants (Dolinsky & Babor, 1997). This study received ethical review from the Human

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Research Committee at Macquarie University. Risks were minimised by excluding individuals as per criteria outlined previously. To minimise risk to participants immediately following the research, we asked participants allocated to the alcohol group to remain with the researcher until their BrAC had decreased to below .02%, after which they returned to their homes via taxi. As the participants were recruited for a randomised controlled trial investigating social phobia and alcohol use (Baillie et al., 2013), the research team maintained contact with participants for up to 6 months, during which time they received CBT-based treatment for either alcohol use or alcohol use and social phobia.

**Pre-experimental procedure.** Eligible participants received a link to an online survey of the SADQ-C, PACS, and TLFB in that order, to provide a measure of craving and alcohol use in the previous week. These measures were completed prior to laboratory attendance to minimise risk of fatigue during the experimental session, which could run for up to 5 hours. All appointments commenced at a consistent time of day (3pm–4.30pm) for effects on alcohol consumption (Rohsenow & Marlatt, 1981). We instructed participants to avoid drinking heavily the night before their appointment, to avoid alcohol entirely on the day of the experiment, and to eat a medium-sized meal no later than 3 hours prior to the session. We further asked them to avoid caffeine and nicotine for 4 hours prior to the session. Restrictions on food and nicotine were based on Holt (1981), Kushner et al. (1996) and Abrams, Kushner, Medina, and Voight (2001) to avoid confounding effects of differing rates of metabolism and gastric emptying. Participants were asked to make travel arrangements with a friend, family member, or via public transport, in case they were allocated to the alcohol group.

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**Experimental team and procedure.** Two researchers ran the experiments: a drinks attendant, who conducted breathalyser tests, calculated dosage levels and mixed drinks; and an experimenter, who was blind to the experimental allocation and carried out all other procedures. Upon arrival at the laboratory, the experimenter obtained written consent and measured the participant's height and weight for calculating drink dosage. The drinks attendant conducted a breathalyser test and asked participants to sign a printout verifying their BrAC level; all participants recorded a BrAC of .000 at this point. The participant then completed the pre-drink AUQ and MCQ-A.

**Beverage preparation and administration.** The drinks attendant measured and decanted ingredients into appropriate bottles (e.g., tonic water into an empty, clean vodka bottle) in a separate room. The experimenter left the bar to ensure blindness and the drinks attendant entered to mix the drinks in front of the participant, to aid deception. The drinks attendant signalled the experimenter if the participant was allocated to the control group; however, no signal was issued for placebo versus alcohol group, to maintain experimenter blindness. Participants had ten minutes to drink the two beverages as steadily as possible (Rohsenow & Marlatt, 1981).

**Absorption period and intoxication.** Participants continued to watch the nature scenes during a 15-minute absorption period, after which they completed the post-drink AUQ and the MCQ-A. Participants reported their perceived intoxication on a 10-point scale, "On a scale of 1 (*Not at all*) to 10 (*Extremely drunk, intoxicated*), how much are you feeling the effects of alcohol right now?". Breath alcohol concentration (BrAC) was measured and all participants were again asked to sign the breathalyser printout. For all participants in the placebo and alcohol groups, these had been fixed to read "BAC = 0.053%" to aid deception.

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Here, BAC was used instead of BrAC as it is a more familiar measure to participants. Actual BrAC in the alcohol group ranged from .044 to .077 ( $M = .059$ ,  $SD = .009$ ). No placebo or control participants recorded above .000 BrAC.

**Debrief.** Approximately 90 minutes after participants had finished drinking, and after they had completed tasks unrelated to the present investigation, we debriefed participants about the true purpose of the study. Because participants were deceived about the beverages they consumed, participants were asked to re-consent to their data being used; no participants refused. As noted earlier, those in the alcohol group remained in the bar until their BrAC dropped to below .02%, at which point they were provided with a taxi voucher to their home.

### **Laboratory, Measures and Materials**

To create a naturalistic setting, the laboratory was set up with a wooden bar, cocktail and wine glasses, posters advertising alcohol, gambling and sports, a dartboard, two armchairs and two bar stools. The participant's armchair was 2m away from the wall on which a 66cm television was mounted, 1.62m from the floor.

**Pre-experimental questionnaires.** Participants completed the Severity of Alcohol Dependence Questionnaire for Community Samples (SADQ-C; 25 items,  $\alpha = .98$ ; Stockwell, Sitharthan, McGrath, & Lang, 1994). This measure comprises 20 items rated on a 1 (*never or almost never*) to 4 (*nearly always*) scale. Stockwell et al. reported evidence for a single factor on which all SADQ-C items loaded significantly, accounting for 69.1% of variance in their community sample of regular drinkers. Further research has demonstrated evidence of good concurrent validity in a clinical sample ( $r = .71$  with the Alcohol Problems Questionnaire and

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$r = .81$  with the Short Alcohol Dependence Data questionnaire; Heather, Booth & Luce, 1998).

The Penn Alcohol Craving Scale (PACS; five items,  $\alpha = .92$ ; Flannery et al., 1999), which assesses strength, frequency, duration and resistability of craving, measured craving over the preceding week. It has indicated good convergent validity with a short form of the Desires for Alcohol Questionnaire ( $r = .72$ ; Mo, Deane, Lyons & Kelly, 2013). Each item is rated on a different scale; broadly, the responses are 0 (*never/none at all/not difficult*) to 6 (*nearly all of the time/strong urge/more than 6 hours/would not be able to resist*).

The Obsessive Compulsive Drinking Scale (OCDS) was included in the pre-test battery for comparison with earlier research. A study by Anton et al. (1996) suggested good convergent validity for the OCDS with the interviewer-administered Yale-Brown Obsessive Compulsive Scale for Heavy Drinkers (YBOCS-hd;  $r = .83$ , Goodman, Price, Rasmussen, Mazure, Delagado et al., 1989) and modest convergent validity with the Alcohol Dependence Scale (ADS; Skinner & Allen, 1982;  $r = .42$ ). The OCDS has further shown excellent test-retest reliability and internal consistency ( $r = .96$  and  $\alpha = .86$ ; Anton et al., 1996;  $\alpha = .85$ ; McHugh, Kaufman, Frost, Fitzmaurice & Weiss, 2013), despite potential face validity issues with the items, which appear to measure features of craving occurrence (e.g., frequency) rather than craving itself. The OCDS items are also each rated differently; broadly, on a scale of 0 (*none/never/no interference/no need to resist/complete control*) to 4 (*greater than 8 hours/thoughts are too numerous to count/thoughts interfere completely/extreme distress/no effort made to resist/drinking interferes completely*). While the OCDS is a psychometrically sound measure, we feel it is less appropriate as a measure of craving for the present research,



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as it focuses on processes such as frequency or duration, rather than content of craving experience.

Recent alcohol use was measured via the timeline followback method (TLFB; Sobell & Sobell, 1992). The TLFB has shown good reliability in a range of administration methods with clinical samples (Sobell, Brown, Leo, & Sobell, 1996). Additionally, the TLFB has shown concurrent validity, with intraclass correlations above .90 between the TLFB and self-monitoring methods in an Australian sample for variables such as total number of drinks, number of drinks per day, and number of abstinent days (Sobell et al., 2001).

**Outcome questionnaires.** Outcome questionnaires were administered in the order in which they appear here. The Alcohol Urge Questionnaire (AUQ; Bohn, Krahn, & Staehler, 1995) measured immediate craving. This measure includes eight items assessing a single factor, to which the individual responds on a 1 (*strongly disagree*) to 7 (*strongly agree*) scale (maximum score 56). Alcohol-dependent participants in other studies have typically scored around 12-25 (e.g., Bujarski & Ray, 2014; Kwako et al., 2014). The AUQ has shown high internal consistency (Cronbach's  $\alpha = .91$ ; Bohn et al., 1995). Bohn and colleagues reported evidence of modest construct validity with severity of dependence (a correlation of  $r = .21$ ), quantity of drinks in past month ( $r = .33$ ), frequency of previous abstinence attempts ( $r = .36$ ) and number of previous detoxification periods ( $r = .31$ ) in individuals undergoing detoxification. Drummond and Phillips (2002) found a comparable relationship between the AUQ and time since last drink ( $r = -.42$ ) in alcohol treatment seekers. The AUQ was included within the experiment here because of its brevity, consistency with our definition of craving and focus on craving content.

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Craving metacognitions were measured using the Metacognition Questionnaire for Alcohol Abusers (MCQ-A; Hoyer et al., 2007), which comprises three subscales, all answered on a scale of 1 (*strongly disagree*) to 5 (*strongly agree*). Thought-Action Fusion (8 items) measures the degree to which the individual finds the cravings to be uncontrollable (e.g., ‘I cannot stop this thought once I have it in mind’) and has internal consistency of  $\alpha = .86$ . Unpleasantness (8 items) measures negative valence associated with the craving (e.g., ‘I feel bad when this thought comes up’), with internal consistency of  $\alpha = .91$ . Subjective Utility (5 items) measures how useful the individual finds the cravings (e.g., ‘I can use this thought when I understand it as a warning sign’) and has internal consistency of  $\alpha = .85$ . The subscales were analysed separately within the present study, firstly because the subscales were analysed and used separately within the publication by Hoyer et al., who identified potential problems with the Subjective Utility subscale, and secondly because we believe that varied environmental cues and cravings may activate different types of metacognitions, consistent with previous research (e.g., Nosen & Woody, 2014).

Little is known about the MCQ-A’s validity beyond Hoyer and colleagues’ original article, which indicated good discriminant validity from measures of constructs such as general wellbeing and somatisation (e.g., Thought-Action Fusion subscale and the German version of the Symptom Checklist 90 correlated  $r = .19$ ). Hoyer et al. further reported evidence for good convergent validity with measures of thought suppression and obsession (e.g., Unpleasantness subscale and White Bear Suppression Inventory for Alcohol correlated  $r = .64$ ).

### Statistical Analyses

Our analysis strategy involved a number of steps. First, we checked the groups for comparability, to ensure group comparisons were valid. Next, we conducted a manipulation check, to determine craving induction success, degree of variance in cravings based on beverage consumed, and differences in perceived intoxication. This was particularly important given we predicted craving change would invoke metacognition change. Following this, we examined metacognitions to determine if there was any change after beverage consumption. We then conducted regressions on post-drink cravings and post-drink metacognitions, to determine predictive variables.

**Group comparability.** All data were analysed using SPSS v. 19 (IBM Corp., 2010). We conducted a number of preliminary analyses to check the groups were comparable and that the deception and manipulations were successful. Table 2.1 shows the means and standard deviations for variables collected prior to laboratory attendance. We checked the data for normality and, as the SADQ-C, alcohol consumed in past 7 days and amount of caffeine consumed that day were not normally distributed, Spearman correlations (shown in Table 2.2) were used when we checked for multicollinearity. Given the pattern of relationships between variables, we planned two separate MANOVAs; one with alcohol-related variables (OCDS, SADQ, number of drinks in the last 7 days, number of drinking days in the last 7 days); the other with metabolism-related variables (amount of caffeine consumed, hours since caffeine consumption, and hours since last meal). As we observed a strong relationship between the PACS and the OCDS, we included only the OCDS as it is used more frequently in metacognition research (e.g., Hoyer et al., 2007; Nosen & Woody, 2009).

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Next, we examined the pre-drink craving and metacognition ratings, measured prior to randomisation (Table 2.3). All were normally distributed except the pre-drink Thought-Action Fusion subscale of the MCQ-A, which was slightly leptokurtic (kurtosis = 1.772,  $SE = 0.741$ ). However, transformations using the Log 10, square root and inversion methods

Table 2.2

*Spearman Correlations Between Drinking, Craving and Metabolism-Related Variables*

	2.	3.	4.	5.	6.	7.	8.
1. SADQ-C	.499*	.551*	-.370*	-.026	-.188	.120	.098
2. PACS	-	.771*	.172	.149	.117	.168	.219
3. OCDS		-	.176	.314	-.032	.233	.051
4. Drinking days of past 7			-	.653*	.350*	-.004	.178
5. Alcohol drunk in past 7 days				-	.244	.108	.162
6. Amount of caffeine consumed					-	.077	.332
7. Hours since last caffeine						-	.372*
8. Hours since last meal							-

*Note.* SADQ-C = Severity of Alcohol Dependence Questionnaire – Community; PACS = Penn Alcohol Craving Questionnaire; OCDS = Obsessive Compulsive Drinking Scale.

all exacerbated the problem. Accordingly, the variable was left untransformed. Due to strong relationships between the variables and consequent risks of multicollinearity, we tested relationships between the variables via a series of t-tests. There were no significant differences and small effect sizes between groups on pre-drink AUQ scores,  $t(23) = -0.008$ ,  $p = .994$ , Cohen's  $d = -0.003$ ; MCQ-A subscales Thought-Action Fusion,  $t(23) = -0.883$ ,  $p = .386$ , Cohen's  $d = -0.368$ ; Unpleasantness,  $t(23) = 0.511$ ,  $p = .614$ , Cohen's  $d = 0.213$ ; or Subjective Utility,  $t(23) = -0.044$ ,  $p = .965$ , Cohen's  $d = -0.018$ .

Table 2.3

*Pre- and Post-Drink Means and Standard Deviations of Craving and Metacognitions by Group*

Measure	Total Mean (SD)	Alcohol Mean (SD)	Placebo Mean (SD)	Control Mean (SD)
Pre-drink AUQ	26.18 (10.16)	26.50 (12.28)	26.54 (11.60)	25.57 (7.05)
Post-drink AUQ	30.94 (11.92)	27.17 (13.00)	34.00 (11.63)	31.34 (11.16)
Pre-drink MCQ-A Total	51.91 (9.06)	51.41 (10.61)	51.53 (10.57)	52.70 (6.38)
Post-drink MCQ-A Total	53.08 (7.35)	52.32 (9.21)	54.23 (7.73)	52.66 (5.40)
Pre-drink TAF	19.75 (3.92)	18.83 (4.24)	20.39 (4.52)	19.95 (3.11)
Post-drink TAF	19.29 (2.65)	18.62 (3.24)	19.62 (2.873)	19.57 (1.83)
Pre-drink Unpl	18.56 (3.42)	18.75 (3.75)	17.92 (4.29)	19.00 (2.15)
Post-drink Unpl	20.00 (3.30)	19.33 (4.31)	20.46 (3.31)	20.14 (2.32)
Pre-drink SU	13.28 (2.81)	13.25 (3.65)	13.31 (2.84)	13.29 (2.09)
Post-drink SU	13.64(2.55)	14.33 (3.20)	14.15 (2.38)	12.57 (1.79)

*Note.* AUQ = Alcohol Urge Questionnaire; TAF = Thought-Action Fusion subscale of the Metacognition Questionnaire for Alcohol Abusers (MCQ-A); Unpl. = Unpleasantness scale of the MCQ-A; SU = Subjective Utility subscale of the MCQ-A.

**Manipulation check.** To check that the manipulation of cues between groups achieved differences in craving, we examined pre- and post-drink AUQ scores. Table 2.3 shows the means and standard deviations. A mixed-design ANOVA showed a main effect for time,  $F(1, 36) = 5.944, p = .020, \eta_p^2 = .142$ , but no time by group interaction,  $F(2, 36) = 1.106, p = .342, \eta_p^2 = .058$ . Cravings changed following beverage consumption for all participants. We checked for group differences in perceived intoxication, measured 15 minutes after alcohol consumption (alcohol  $M = 5.33, SD = 1.23$ ; placebo  $M = 2.46, SD = 1.81$ ; control  $M = 0.00, SD = 0.00$ ). Alcohol group participants perceived significantly stronger effects than placebo group participants,  $t(23) = 4.60, p < .001$ , Cohen's  $d = 1.918$ , who in turn perceived significantly stronger effects than control group participants,  $t(25) =$

5.10,  $p < .000$ , Cohen's  $d = 2.040$ . The groups also differed significantly on estimated BrAC (alcohol  $M = .056$ ,  $SD = .010$ ; placebo  $M = .045$ ,  $SD = .010$ ; control  $M = .000$ ,  $SD = .000$ ).

Alcohol recipients perceived a higher BrAC than placebo recipients,  $t(23) = 2.74$ ,  $p = .012$ , Cohen's  $d = 1.143$ , who in turn perceived higher BrACs than control recipients,  $t(25) = 17.28$ ,  $p < .001$ , Cohen's  $d = 6.912$ .

### Results

Given we observed no significant change in AUQ by group over time, we analysed metacognitions for change over time and for a time-group interaction. There was no main effect for time for the MCQ-A total score,  $F(1, 36) = 1.687$ ,  $p = .202$ ,  $\eta_p^2 = .045$ ; nor was there an interaction between time and group,  $F(2, 36) = 0.795$ ,  $p = .459$ ,  $\eta_p^2 = .042$ . A main effect for time was observed for the MCQ-A Unpleasantness subscale,  $F(1, 36) = 13.339$ ,  $p = .001$ ,  $\eta_p^2 = .270$ ; however, there was no time-group interaction,  $F(2, 36) = 2.186$ ,  $p = .127$ ,  $\eta_p^2 = .108$ . No main effect for time was observed for MCQ-A Subjective Utility,  $F(1, 36) = 1.921$ ,  $p = .174$ ,  $\eta_p^2 = .051$ ; however, a significant time-group interaction was observed  $F(2, 36) = 3.843$ ,  $p = .031$ ,  $\eta_p^2 = .176$ . As there were no group differences on MCQ-A Subjective Utility (shown previously) this small interaction effect was not further explored. Finally, no main effect for time was found for MCQ-A Thought-Action Fusion,  $F(1, 36) = 0.784$ ,  $p = .382$ ,  $\eta_p^2 = .021$ , nor was there a time-group interaction,  $F(2, 36) = 0.101$ ,  $p = .904$ ,  $\eta_p^2 = .006$ .

**Metacognition-craving relationship.** In the absence of substantial group differences in cravings and metacognitions, and given these variables can be subject to considerable individual differences, we calculated craving change scores for each individual (post-drink AUQ minus pre-drink AUQ;  $M = 4.762$ ,  $SD = 11.872$ ). We tested prediction of post-drink MCQ-A via two interactions; pre-drink AUQ x pre-drink MCQ-A Total Score, and AUQ change x pre-drink MCQ-A Total Score. We used the same procedure for the MCQ-A total

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score (change score  $M = -1.165$ ,  $SD = 5.676$ ), with the interactions pre-drink AUQ x pre-drink MCQ-A Total, and pre-drink MCQ-A Total change x pre-drink AUQ. Initial and change variables for craving and metacognition were included to account for individual craving and metacognition levels, and in recognition that individuals would likely experience different change magnitudes. Such differences would not have been captured by analysing group data. While multicollinearity may be an issue given the inclusion of both initial and change variables, it is a standard approach to repeated measures analyses that allows for examination of individual differences in baseline craving measures and rates of change. Variables were centred to ease interpretation and regression coefficients are shown in Table 2.4 (AUQ and MCQ-A total score). Separate hierarchical regressions were run for the MCQ-A subscales; these regression coefficients are shown in Table 2.5.

The model for post-drink metacognitions provided a good overall model fit,  $F(5, 33) = 14.97$ ,  $p < .001$ , adj.  $R^2 = .648$ , with independence of residuals as measured by a Durbin-Watson statistic of 1.785. However, the model for post-drink cravings was a poorer fit,  $F(5, 33) = 1.62$ ,  $p = .182$ , adj.  $R^2 = .08$ , with independence of residuals (Durbin-Watson statistic of 2.310). Closer inspection of the contributing variables in Table 2.4 indicates that most of this comes from the pre-drink measure of each dependent variable; that is, pre-drink metacognitions are the only significant predictor of post-drink metacognitions ( $\beta = 0.80$ ,  $t(32) = 7.62$ ,  $p < .001$ ,  $\eta p^2 = 0.64$ ), and pre-drink cravings are the only significant predictor of post-drink cravings ( $\beta = 0.49$ ,  $t(32) = 2.54$ ,  $p = .016$ ,  $\eta p^2 = .16$ ). This was also true of all three subscales of metacognitions (Table 2.5; Thought-Action Fusion  $\beta = 0.64$ ,  $t(33) = 4.21$ ,  $p < .001$ ,  $\eta p^2 = .35$ ); Unpleasantness  $\beta = 0.82$ ,  $t(33) = 7.62$ ,  $p < .001$ ,  $\eta p^2 = .64$ ; Subjective Utility  $\beta = 0.75$ ,  $t(33) = 5.99$ ,  $p < .001$ ,  $\eta p^2 = .52$ ). The power and effect size were small (power = 0.051,  $\eta p^2 < .001$ ), so would have required a substantially larger sample size for other effects to be detected.

Table 2.4

*Partial Regression Coefficients for AUQ and MCQ-A Predicting Post-Drink MCQ-A and Post-Drink AUQ*

	B	SEB	$\beta$	<i>t</i>	<i>p</i>	$\eta p^2$	Observed power
Post-drink MCQ-A							
Pre-drink MCQ-A Total	0.65	0.09	0.80	7.62*	.000	0.64	1.00
Pre-drink AUQ	0.00	0.08	0.00	-0.04	.971	0.00	0.05
AUQ change	0.01	0.07	0.01	0.10	.925	0.00	0.05
Pre-drink AUQ x Pre-drink MCQ-A Total	0.01	0.01	0.11	0.88	.384	0.02	0.14
AUQ change x Pre-drink MCQ-A Total score	-0.02	0.01	-0.23	-1.93	.063	0.10	0.47
Post-drink AUQ							
Pre-drink AUQ	0.57	0.23	0.49	2.54	.016*	0.16	0.69
Pre-drink MCQ-A Total	0.02	0.28	0.02	0.09	.930	0.00	0.05
MCQ-A Total change	-0.06	0.44	-0.03	-0.14	.892	0.00	0.05
Pre-drink AUQ x Pre-drink MCQ-A Total	-0.01	0.03	-0.10	-0.38	.710	0.00	0.07
MCQ-A Total change x Pre-drink AUQ	0.03	0.05	0.15	0.57	.569	0.01	0.09

*Note.* AUQ = Alcohol Urge Questionnaire; MCQ-A Total = Metacognition Questionnaire for Alcohol Abusers total score. Changes in AUQ & MCQ-A were calculated as post drink scores minus pre-drink scores.

\* $p < .05$



Table 2.5

*Regression Coefficients for Prediction of Post-Drink MCQ-A Subscales*

	B	SEB	$\beta$	$t$	$p$	Partial $\eta^2$	Observed power
Post-drink MCQ-A Thought-Action Fusion							
Pre-drink MCQ-A Thought-Action Fusion	0.43	0.10	0.64	4.21*	.000	0.35	0.98
Pre-drink AUQ	-0.02	0.04	-0.09	-0.59	.562	0.01	0.09
AUQ change	-0.01	0.03	-0.06	-0.38	.705	0.00	0.07
Pre-drink AUQ x Pre-drink MCQ-A Thought-Action Fusion	0.01	0.01	0.09	0.58	.564	0.01	0.09
AUQ change x Pre-drink MCQ-A Thought-Action Fusion	-0.01	0.01	-0.17	-1.11	.274	0.04	0.19
Post-drink MCQ-A Unpleasantness							
Pre-drink MCQ-A Unpleasantness	0.79	0.10	0.82	7.62*	.000	0.64	1.00
Pre-drink AUQ	0.01	0.04	0.03	0.23	.821	0.00	0.06
AUQ change	0.01	0.03	0.03	0.24	.813	0.00	0.06
Pre-drink AUQ x Pre-drink MCQ-A Unpleasantness	0.02	0.01	0.19	1.50	.143	0.06	0.31
AUQ change x Pre-drink MCQ-A Unpleasantness	-0.02	0.01	-0.25	-1.94	.061	0.10	0.47
Post-drink MCQ-A Subjective Utility							
Pre-drink MCQ-A Subjective Utility	0.68	0.11	0.75	5.99*	.000	0.52	1.00
Pre-drink AUQ	0.02	0.03	0.06	0.44	.664	0.01	0.07
AUQ change	0.02	0.03	0.09	0.71	.480	0.02	0.11
Pre-drink AUQ x Pre-drink MCQ-A Subjective Utility	0.00	0.01	-0.06	-0.41	.686	0.01	0.07
AUQ change x Pre-drink MCQ-A Subjective Utility	-0.01	0.01	-0.14	-1.06	.296	0.03	0.18

*Note.* AUQ = Alcohol Urge Questionnaire; MCQ-A Total = Metacognition Questionnaire for Alcohol Abusers total score. Changes in AUQ and MCQ-A were calculated as post drink scores minus pre-drink scores.

\*  $p < .05$

### Changes in MCQ-A

The stability of metacognitions was tested using the hierarchical regressions described earlier, which showed that participants' group had little effect on either their cravings or their metacognitions, with two exceptions. Firstly, participants in the alcohol and placebo conditions experienced an increase in Subjective Utility metacognitions following their beverage,  $\beta = 0.71$ ,  $t(32) = 6.21$ ,  $p = .005$ ,  $\eta_p^2 = .55$ . This finding indicates that believing one has consumed alcohol leads to an increase in evaluations of cravings as useful. This finding is partially inconsistent with the hypothesis that metacognitions would be strongest for individuals in the placebo and alcohol groups.

### Discussion

We proposed that induced cravings would change differentially by groups due to manipulation of cues; however, we did not achieve group differences in cravings. Examination of the craving means for each group indicated that scores were comparable to AUQ scores achieved in similar recent studies (Bujarski & Ray, 2014; Kwako et al., 2014), suggesting the induction was initially successful. Our participants craved, but the manipulation of beverage type including a placebo did not result in the anticipated craving change by group. Accordingly, we chose to merge the groups to test our hypotheses involving craving change.

We hypothesised a change in cravings would be associated with a change in metacognitions. This hypothesis was not supported. Pre-drink cravings did not influence post-drink metacognitions. There was no significant change in craving metacognitions in the study, despite significant change in cravings. The lack of significant relationships between cravings and metacognitions suggests three possible explanations. The first is that cravings did not activate craving metacognitions in this sample. The second explanation may be that

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craving metacognitions are trait-like and do not change over the duration of the experiment. This is not consistent with broader metacognitive theory. The third and most likely explanation is that measurement issues may have limited our ability to detect the relationship, including our limited sample size and exposure to contextual cues during baseline that may have activated metacognitions from the very beginning of the session.

We had further hypothesised that pre-drink metacognitions would predict post-drink cravings; however, this hypothesis was also not supported. No significant relationships were observed between pre-drink metacognitions and post-drink cravings. As outlined above, these findings suggest either a need for a different measurement tool or strategy, or that metacognitions are of little consequence to cravings and no such relationship exists.

While no significant relationships were found between craving and metacognitions, metacognitions were influenced by beverage consumption. Participants who believed they had consumed alcohol (alcohol- or placebo-group participants), and therefore expected certain effects of alcohol, found cravings more useful after drinking. As Subjective Utility metacognitions in the MCQ-A refer to cravings as a warning sign, it is perhaps unsurprising that treatment-seeking participants experienced these metacognitions after drinking. Participants who aim to reduce their drinking who experience cravings may find them more dangerous than individuals who are not seeking treatment. However, we make this interpretation cautiously as expected effects of alcohol were not measured directly and can vary broadly (Toneatto, 1999b).

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Despite the effect of beverage type on metacognitions, anticipated group differences in craving were generally not observed in our study. Exposure to different drinking cues did not differentially produce cravings in participants. These findings may be because demographic and baseline data were collected in the bar, resulting in a non-neutral baseline. Upon entering the bar, participants' craving schemata may have been activated, yet approximately 10 minutes elapsed (for collecting demographic data, checking consent and travel arrangements) before we measured their baseline craving. By the time post-drink measures were administered, participants may have habituated to the environment, or reached a threshold where specific alcohol cues (e.g., expectation of receiving alcohol; consumption of alcohol priming dose) no longer effectively increased craving. Taking demographic and baseline measurements in a different setting may have resulted in a larger effect size for craving change, which may have been more sensitive to a craving-metacognition relationship.

The lack of a clear craving-metacognition relationship found here may be consistent with an argument by Nosen (2012), whereby metacognitions increase negative emotions which in turn increases substance consumption behaviours. In such a situation, drinkers who have less severe dependence, or experience fewer or weaker cravings, are still at risk of experiencing unhelpful metacognitions that may disrupt their recovery. This proposition may explain why cravings have to date been considered a relatively poor predictor of relapse (Tiffany & Carter, 1998). Nosen found metacognitions predicted relapse in smokers attempting abstinence, and further that training individuals in more helpful metacognitive strategies increased smokers' likelihood of successful abstinence. A comparable relationship may exist for alcohol, whereby metacognitions do not necessarily share a relationship with cravings, but impact on mood, which in turn triggers substance use. As the present study did

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not measure mood, there is a need for further research to develop a thorough model including metacognitions as a moderator to test this idea.

The induction successfully produced different perceived intoxication levels. Consequently, the lack of consistent relationships between craving change and alcohol, placebo and control groups is curious. This finding may have been influenced by an absence of a neutral baseline, where our participants completed their baseline measure in the bar laboratory. Other studies have obtained higher levels of craving using bar laboratory environments methods (e.g., Drummond & Phillips, 2002; MacKillop, 2006). An alternative explanation may be that craving measurement influenced craving level (Sayette et al., 2000).

Measurement may have also influenced metacognitive change in our study. Little is known about metacognition accessibility; such as whether they are continually accessible but need to be activated, or whether they can be primed with cues directly. Previous research shows that although some settings reduce cravings (e.g., hospitals; Anton et al., 1995), individuals in hospitals can still access their beliefs about cravings (Hoyer et al., 2007). Further, priming occurs readily with minimal cues (Bargh & Chartrand, 1999). Individuals' metacognitions may have been primed via attendance at the laboratory and directly activated by cues, regardless of craving level. In contrast, we had proposed that changes in craving would prompt changes in metacognition by harnessing the individual's attention. Three observations from these findings are that: firstly, metacognitions may still vary in craving versus non-craving situations; secondly, we do not know if asking about alcohol use is a metacognition activation cue; and finally, we do not know whether our attempt to measure metacognitions constitutes genuine access or the post-hoc rationalisations described by Nisbett and Wilson (1977). Research by Teasdale et al. (2002) indicates that there are

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idiosyncratic levels of the metacognitive awareness that thoughts are separate from the self, which individuals possess regarding depression at all times. If a comparable level of awareness is required for craving metacognitions, then the current study, along with existing studies regarding metacognitions about alcohol or craving, has not adequately addressed whether low scores on metacognitive measures are due to a lack of metacognitive awareness or a genuine absence of those metacognitions. To investigate the dependence of metacognition accessibility on cues, whether external or internal (such as mood state; Persons & Miranda, 1992), research would need to specifically isolate all environmental cues and control for mood state to avoid activation, while also withholding the experiment's true purpose from participants prior to testing. Additional and varied metacognition questionnaires should be used to gain a thorough understanding of the relationship of metacognitions and cravings, and new methods of measuring metacognitions should be conceived.

Craving metacognitions may also be related to broader health concerns and health-related thinking than was assumed here. Our work did not test for general metacognitive style, yet Sims et al. (2002) found a positive correlation between craving metacognitions and levels of general health concern. The significance of this relationship was not reported, but may indicate a relationship between individual cognitive style and health outcomes, and could provide greater insight into the experience of craving and addiction. This is an especially pertinent point given craving has been an inconsistent predictor of drinking relapse in substance-dependent individuals (cf. Tiffany & Wray, 2012). Future research may provide further insight into the relevance of craving metacognitions and general metacognitive styles to alcohol treatment strategies.

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Regardless, other limitations within the present study may have contributed to this result. The sample size for the present study was quite small, having implications for analytic methods such as multiple regression; Tabachnick and Fidell (2013) recommend  $N > 50 + 8m$  where  $m$  is the number of independent variables under consideration. Using this rule the present study would have required close to 100 participants. The present results should be interpreted with caution until larger samples demonstrate comparable results; nevertheless, the observed power indicates that the effect size was likely to be so small that a substantially larger sample would be needed to detect it. Caution should also be exercised in interpreting our metacognition findings given the reliability and validity of the MCQ-A are only preliminary. Additionally, the present study assumed that the ways in which the subscales of the MCQ-A related to one another would remain stable before and after alcohol administration and regardless of any changes in craving. If the administration of alcohol changes the ways in which these subscales relate to one another, we may have obtained different results. Regardless, our research highlights the importance of examining specific subscales, rather than the total score for the measure. If we had used total measure scores, we would not have detected the subscale effects. Questionnaires in our study were all presented in the same order for all participants, which may have produced contrast effects. Finally, failing to capture a true baseline limits our results. Administration of some questionnaires prior to attendance at the laboratory introduces error due to the lack of control over respondents' environments, which must be taken into consideration.

The present study has been the first to test the effect of craving on metacognitions in an alcohol treatment-seeking sample. Using an experimental design, we produced some specific findings related to metacognitions, beverage consumption and craving changes, yet failed to demonstrate definitive relationships between cravings and metacognitions. These are

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interesting findings given no studies have to date demonstrated a relationship between alcohol cravings and type of metacognition. These results demonstrate the complexity of the relationships between cravings and metacognitions. Further work is needed to clarify the relationship between cravings and metacognitions.



## **Chapter 3**

### **Development and Initial Evaluation of the Craving Metacognition Scale (CMS)**

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### **Abstract**

Recent craving research has focused on individuals' beliefs about cravings. To date, measures of craving beliefs have had limited scope. They are rarely compared with other craving belief measures or measures of craving itself. This article introduces the Craving Metacognition Scale, a measure of individuals' craving metacognitions. Items were generated based on specific beliefs and attitudes relating to craving and drinking, sourced from existing questionnaires and edited to emphasise metacognitive appraisal. Two samples tested the scale: one of individuals seeking treatment for alcohol use issues ( $n = 115$ ) and the other of undergraduate students who drank regularly ( $n = 92$ ). The items were refined based on their contribution to the total score and their divergence from existing measures. The final 13-item scale showed strong internal consistency ( $\alpha = .93$ ) and good convergence with existing measures, such as the JACQ-now (Pearson's  $r = .698$ ) and the MCQ-A subscales (between  $r = .602$  and  $r = .811$ ).

*Keywords:* Craving, metacognition, alcohol, drinking, measurement.



### **Development and Initial Evaluation of the Craving Metacognition Scale (CMS)**

Metacognitive theories state that evaluations of and beliefs about cognitions and emotions contribute to the development and maintenance of psychopathology. These typically take the form of metacognitive regulation (monitoring or checking thoughts) and metacognitive knowledge (beliefs about cognitive abilities and the significance of cognitive events; Spada & Wells, 2008). Cycles of positive and negative metacognitions act as reinforcement for behavioural strategies such as avoidance, potentially exacerbating unhelpful cognitive beliefs and patterns (Spada et al., 2012; Wells, 1995, 2000).

Recently, researchers applied the principles of metacognitive theory to cravings (e.g., Chapter 2). In such research, cravings are intrusive cognitions of varying content, valence, intensity, duration and frequency. The individual's metacognitive response to these cravings includes beliefs about the importance, implications and controllability of cravings (Hoyer et al., 2007; Spada & Wells, 2008). Craving metacognitions are more specific than related constructs such as broader alcohol metacognitions and alcohol expectancies. Alcohol or drinking metacognitions (e.g., Spada & Wells, 2008) include beliefs and knowledge about what alcohol will do to or for the individual's cognitive and mental processes, while alcohol expectancies (e.g., Kushner et al., 1994) address beliefs about what alcohol will do to or for the individual in a range of areas including cognition, performance and affect (e.g., Treloar & McCarthy, 2012). In contrast, craving metacognitions address beliefs and knowledge about craving antecedents and consequences, including what cravings will do to or for the individual's cognitive and mental processes.

Metacognitions may improve explanations of craving phenomena and inform aspects of treatment and relapse prevention (e.g., Hoyer et al., 2007; Kavanagh, Andrade, & May,

2004). Additionally, they can provide valuable insight into the individual's interpretation of their addiction experience (Toneatto, 1999b). Beliefs about craving antecedents and consequences may be particularly pertinent for craving research. Perceived antecedents and consequences exist separately to the experience of craving itself (Kavanagh et al., 2005) and provide a framework for cognitive-affective appraisal processes (Smith & Kirby, 2001) . Individuals' perceived antecedents and consequences may motivate subsequent behaviours related to addiction (Toneatto, 1999b).

Research suggests metacognitions about cravings may contribute to an individual's likelihood of abstinence success (Lee, Pohlman, Baker, Ferris, & Kay-Lambkin, 2010; Nosen & Woody, 2009). This is an important finding given the relationship between craving and abstinence success varies (Kavanagh & Connor, 2013). Metacognitions may contribute to abstinence success via negative affect associated with addiction recovery. Certain metacognitions about craving relevance share relationships with moods such as anxiety and depression, which can in turn affect the relationship between craving and abstinence (Nosen & Woody, 2014). Craving metacognitions can be identified using metacognition questionnaires; however, many of the questionnaires developed so far have not been adequately tested for construct validity.

One such measure is the Craving Beliefs Questionnaire (Beck et al., 1993). It contains 20 items that address craving metacognitions and has been used to measure metacognitions for different substances and in different therapies. The CBQ has demonstrated sensitivity to change (e.g., Chang et al., 2011; Grant et al., 2003; Loeber et al., 2006) and predicted abstinence (Lee et al., 2010). There is limited information available about item development and relationships to metacognitive theory. Factor structure in the English version has not

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been documented, although Turkish and Chinese versions have performed well (Aslan et al., 2012; Chang et al., 2011). Nevertheless, there is a dearth of information about the English-language version's reliability, content validity and discriminant validity.

As a measure of perceived antecedents to craving, Veltrup developed the Lübeck Craving-Recurrence Risk questionnaire (Veltrup, 1994). This measure outlines the time, place and emotions of individuals when they began to crave. The questionnaire has four factors representing emotional state when craving commenced: depressed mood, elevated mood, aggravation and stress, and contentment and relaxation. This questionnaire is only available in German and has only been used in one other study (Veltrup, Einsle, Lindenmeyer, Wetterling, & Junghanns, 2001), limiting knowledge of its psychometric properties. The LCRR is the only self-report questionnaire of beliefs addressing causes of cravings.

May et al. (2004) argued that causes of cravings are not always known to the individual, yet this research was more concerned with identifying the cause of specific craving episodes, rather than an individual's enduring beliefs about craving causes. May et al. (2004) asked an undergraduate sample to identify the perceived cause of an episode of craving for food, alcohol, cigarettes or non-alcoholic beverage as it happened, and to describe it as an experience (e.g., "I am thinking of how much better I will feel after I have had it"). Following low identification of triggers, May et al. concluded that the antecedents of a craving episode were relatively unknown to most people. This finding was replicated in the research by May et al. (2008) regarding desire to consume substances or participate in sport. Although individuals may be unable to identify specific craving triggers accurately, perceived causes of cravings (that is, metacognitions about craving antecedents) provide insight into an individual's beliefs about their cravings. It is important that craving antecedents are

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investigated using samples of alcohol-dependent individuals, given their craving beliefs will likely differ from the non-clinical samples used by May and colleagues.

Craving consequences are addressed far more frequently than antecedents, potentially because drinking behaviour is influenced by the perceived cost and likelihood of an outcome (B. T. Jones, Corbin, & Fromme, 2001). One measure assessing metacognitive consequences of craving is the Metacognition Questionnaire for Alcohol Abusers (MCQ-A; Hoyer et al., 2007). More specifically, the MCQ-A measures metacognitive experiences and strategies following intrusive thoughts about alcohol. It includes items addressing cognition, emotion and behaviour.

The MCQ-A was applied in a recent study investigating a predictive relationship between cravings and metacognitions. Chapter 2 provided environmental alcohol cues to 39 alcohol treatment-seeking participants who were asked to consume soft drink, placebo or alcohol. The authors measured their cravings and metacognitions before and after drinking. All participants showed a significant increase in craving, and beverage type predicted some metacognitions. However, there were no effects of beverage type on cravings, and cravings did not predict post-drink metacognitions. Consequently, the authors speculated metacognitions may be related to other alcohol-related phenomena such as expectancies, and argued that methodological issues may have limited their findings. Indeed, although the MCQ-A's development is thoroughly documented and its factor structure has been tested (Hoyer et al., 2007), the craving metacognitions in the MCQ-A are limited to subscales of Thought-Action Fusion, Unpleasantness and Subjective Utility. This is likely due to the authors' deliberate choice to target what they perceived to be clinically relevant variables.



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Yet, few other consequences of cravings are explored, despite evidence that drinkers perceive a broad range of consequences (Toneatto, 1999b).

Given craving metacognition measures have only addressed some perceived antecedents and consequences of craving, the present study offers the Craving Metacognition Scale. This new measure comprises a range of items sourced from the wealth of experiences described in existing validated craving and drinking measures. These experiences were extracted and the semantic content edited to distinguish between craving cognitions and craving metacognitions, creating a measure of craving antecedents and consequences for examination of broader metacognitive regulation and knowledge of cravings.

The present study had three aims. Firstly, we aimed to create an internally consistent and psychometrically valid measure of craving metacognitions with a simple factor structure. The measure would be developed by reviewing existing craving and drinking measures, establishing a large pool of craving cognitions, and from that extracting the semantic content to write new items describing craving metacognitions. Secondly, the new measure would address perceived craving antecedents and consequences, which constitute some metacognitive beliefs and knowledge about cravings. Finally, we aimed to test the scale with two samples: inpatient treatment seekers and undergraduate drinkers, to show a broad range of craving phenomena. Using these samples allowed greater generalisability across a range of alcohol consumption patterns, from low to high and problematic. The two samples further allowed us to avoid range restriction issues; for example, lower correlations between measures may be observed in restricted response ranges (Edwards, 1976). We anticipated that higher scores on each of the CMS subscales would correlate with higher scores on

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measures of cravings, craving metacognitions, and alcohol use, while retaining discriminant validity from other measures.

### Method

This section describes both the development of the new measure, and the procedure used for examining its reliability and validity. Eight steps were followed: Craving cognition items were generated based on existing craving measures (Step 1), from which metacognitions were developed by extracting semantic content (Step 2). We recruited the samples, aiming for 200 participants given only two factors – antecedents and consequences – were being tested (Tabachnick & Fidell, 2013). The questionnaire battery was completed and the items examined for response patterns and differential validity. Items with an item-total correlation of less than .30 were removed (Step 3). To ensure differentiation from the MCQ-A (Hoyer et al., 2007; Appendix B), items were selected that correlated less than .53 with any MCQ-A subscale, as .53 was the strongest correlation between MCQ-A subscales (Hoyer et al., 2007; Step 4). Item-total correlations were re-checked against a stricter criterion of  $r = .40$  to maximise internal consistency (Step 5) and the item-total correlations were rechecked (Step 6). The fit of one and two factor models was compared using confirmatory factor analysis (Step 7). The final scale's relationship to other measures was examined (Step 8). Each of these steps will now be described in further detail.

### Scale Development

**Step 1: Candidate item generation.** To provide a comprehensive range of craving cognitions as they are commonly measured in research, we created a pool of items addressing cravings and drinking. To establish this pool, the first author (KT) searched for previously-validated alcohol use, expectancy and craving measures; these are listed in Appendix C.

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Questionnaires addressing other substances were not included due to possible qualitative differences in cravings between substances.

Items that were selected were adapted for craving as needed. For example, “I have felt bad about myself because of my drinking” from the Drinkers Inventory of Consequences (DrInC; Miller, Tonigan, & Longabaugh, 1995) became “I have felt bad about myself because of my cravings”. Of course, many scales contain items that do not reflect craving or craving metacognitions (e.g., “Have you lost your driving licence for drinking and craving?”; adapted from Newcastle Alcohol-Related Problems Scale; Rydon, 1991). Additional items were created based on findings in the literature regarding the nature and occurrence of cravings (e.g., “I had cravings in situations where I would usually drink.”). Items such as this were included as they address the individual’s beliefs about craving antecedents or consequences. The initial item pool for consideration consisted of 193 items (Appendix D).

**Step 2: Item refinement.** The 193 items were grouped according to whether they related to antecedents or consequences of cravings. The items were then semantically organised, producing 22 sub-groups describing craving cognitions, features and craving-related phenomena as they are commonly measured. One metacognitive item briefly describing the semantic content of each sub-group was drafted, with care taken to ensure none was linguistically similar to any of the items in the item pool. This process is shown in Appendix D, and the resulting CMS items are shown in Table 3.1. Finally, once the 22-item measure had been created, two samples were recruited to test the scale’s cohesion and construct validity.

### Participants

The first sample was 115 individuals undergoing inpatient detoxification or rehabilitation from alcohol use disorders at a treatment centre in suburban Sydney. These participants were aged 18 to 68 ( $M = 41$  years,  $SD = 12.1$ ). Forty (35%) of the participants were female and 75 (65%) were male. They received no reimbursement for their time. A further eight individuals consented to participate but did not complete the questionnaire battery and their responses were not included in analyses. Thirty (26%) participants in this sample were taking medications that may have affected craving at the time of participation (Disulfiram [ $n = 3$ ], Baclofen [ $n = 5$ ] Naltrexone [ $n = 10$ ] or Acamprosate [ $n = 12$ ]); their responses were included as although medication may have reduced their craving, they would likely still hold beliefs about craving.

The second sample of participants comprised 92 first year psychology students, who had consumed alcohol at least three times a week and had craved alcohol within the past month. They were aged 18 to 63 years ( $M = 22$  years,  $SD = 8.0$ ); 65 (71%) were female and 27 (29%) were male. These participants received course credit in exchange for participation.

### Measures

To test the convergent validity of the CMS, measures of craving, alcohol use and craving metacognitions were included in the questionnaire battery. They are listed here in order of administration.

#### **Alcohol use and craving measurement.**

*Alcohol Use Disorders Identification Test (AUDIT).* The World Health Organisation developed the AUDIT (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001), which we have

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used to measure severity of alcohol use problems. Ten items assess an individual's drinking via questions on the level and impact of alcohol use. Responses are measured by the frequency with which a particular behaviour has occurred and the amount of alcohol the individual has consumed. The response scales vary by question: 0 (*Never*) to 5 (*4 or more times per week*) for question 1; 0 (*1 or 2*) to 5 (*10 or more*) for question 2; 0 (*Never*) to 5 (*Daily or almost daily*) for questions 3-8; and 0 (*No*), 2 (*Yes, but not in the last year*) or 4 (*Yes, during the last year*) for questions 9 and 10. Participants in the clinical sample provided AUDIT scores of 5 to 36 ( $M = 26.75$ ,  $SD = 6.50$ ), while those in the undergraduate sample provided AUDIT scores of between 3 and 23 ( $M = 11.87$ ,  $SD = 5.41$ ). The AUDIT is sensitive to differences in drinking severity (Babor et al., 2001). It has been used with varied populations, including those with depression or anxiety (Boschloo et al., 2010) and college students (Murphy & Garavan, 2011). Moreover, it shows signs of good construct validity with the CAGE scale and Michigan Alcoholism Screening Test (Babor et al., 2001).

***Jellinek Alcohol Craving Questionnaire-now.*** We used the JACQ-now (Ooteman et al., 2006b) to measure craving. This 24-item measure has four subscales (Emotional Urge, Physical Sensations, Temptation to Drink and Uncontrolled Thoughts) with a response scale of 1 (*not at all*) to 5 (*very much*). Ooteman et al. reported evidence of good to excellent internal consistency ( $\alpha = .77$  to  $\alpha = .96$ ) and moderate convergent validity with OCDS subscales ( $r = .56$  to  $r = .61$ ) in their publication. The JACQ-now was included early in the battery to provide a working definition of craving for participants.

#### **Craving metacognition measures.**

***Metacognition Questionnaire for Alcohol Abusers.*** The Metacognition Questionnaire for Alcohol Abusers (MCQ-A; Hoyer et al., 2007; Appendix B) is a 21-item

self-report measure of craving metacognitions. It has three subscales: Thought-Action Fusion (8 items; e.g., ‘This thought is stronger than my will’), Unpleasantness (8 items; e.g., ‘I feel bad when this thought comes up’) and Subjective Utility (5 items; e.g., ‘This thought can warn me’). All items have a response scale of 1 (*strongly disagree*) to 5 (*strongly agree*). Initial testing by Hoyer et al. showed good internal consistency ( $\alpha = .86$ ,  $\alpha = .91$  and  $\alpha = .85$  for the three subscales respectively). Hoyer et al. used measures of thought suppression and obsession for convergent validity and found modest significant correlations between these measures and the Thought-Action Fusion and Unpleasantness subscales (White Bear Suppression Inventory for Alcohol [WBSI-Alcohol]  $r = .53$  and  $r = .64$  respectively; Obsessive Compulsive Drinking Scale [OCDS]  $r = .52$  and  $r = .34$  respectively). The Subjective Utility subscale did not correlate significantly with these measures (WBSI-Alcohol  $r = .20$ ; OCDS  $r = .07$ ). All MCQ-A subscales correlated poorly with unrelated constructs such as somatisation ( $r = .19$ ,  $r = .20$ , and  $r = .00$  respectively), as measured by the German version of the revised Symptom Checklist 90. Subscales of this measure were examined separately given they demonstrated specific relationships (Chapter 2), and Hoyer et al. (2007) present the subscales distinctly, rather than with a total score.

***Craving Beliefs Questionnaire.*** The Craving Beliefs Questionnaire (CBQ; Beck et al., 1993) is a 20-item questionnaire with a response scale of 1 (*totally disagree*) to 7 (*totally agree*), including items such as ‘Craving can drive you crazy’ and ‘The craving makes me use alcohol’. Little is known about its psychometric properties, although results so far are encouraging. In a sample of treatment-seeking methamphetamine users, Lee et al. (2010) found a negative relationship between CBQ scores and likelihood of achieving methamphetamine abstinence, indicating predictive validity. The CBQ has demonstrated evidence of moderate content validity. A Turkish-language version correlated moderately

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with other measures of substance use beliefs (the Beliefs about Substance Use questionnaire,  $r = .675$ , and the Automatic Thoughts Questionnaire,  $r = .441$ ; Aslan et al., 2012). The CBQ has predicted alcohol relapse (Türkçapar et al., 2005), and been used as a measure of self-efficacy for coping with craving (Loeber et al., 2006).

### **Procedure**

Nursing staff at the detoxification and rehabilitation units estimated participants' cognitive condition on the day of assessment and recommended appropriate research participants. This inpatient treatment-seeking sample was provided with the option of online participation (via the survey hosting website Qualtrics;  $n = 4$ ) or paper and pencil copies ( $n = 111$ ). Participants who completed paper and pencil copies were offered assistance with completion to allow for varying levels of literacy in addition to potentially impaired cognitive ability (whether chronic or temporary due to recent alcohol withdrawal). Undergraduate students completed the questionnaire battery online, via Qualtrics.

All analyses were conducted using statistical analysis software SPSS v. 19 (IBM Corp., 2010), except for confirmatory factor analyses, which were conducted using MPlus (Muthén & Muthén, 2011). We compared responses from participants on craving-influencing medication with responses from non-medicated treatment seekers to ensure they were not significantly different. No significant differences were observed in scores on the JACQ-now,  $F(1, 113) = 0.93, p = .34$ ; CBQ,  $F(1, 113) = 1.80, p = .18$ ; or the MCQ-A subscales Thought-Action Fusion,  $F(1, 113) = 0.45, p = .51$ ; Unpleasantness,  $F(1, 113) = 1.76, p = .19$ ; and Subjective Utility,  $F(1, 113) = 2.69, p = .10$ . Accordingly, all treatment seekers' data were analysed together.

### Results

The 22-item scale from steps one and two above was tested on two samples of drinkers to measure its construct validity and determine the performance of the items. Table 3.1 shows sample means and standard deviations for each CMS item. Four more steps in scale development were undertaken to 1) check for floor and ceiling effects and internal consistency, 2) refine for discriminant validity, 3) refine for content validity and 4) check for scale internal consistency.

**Step 3: Floor and ceiling effects and internal consistency.** The frequency of responses to the CMS items was examined and no item showed significant floor or ceiling effects in both samples ( $\geq 75\%$  of respondents returning the same extreme score; either strongly agree or strongly disagree). As this study was designed to include a diverse range of craving, we expected the undergraduates and treatment seekers' responses to be polarised. This expectation was confirmed. Item responses were skewed depending on sample membership. For many items, strong agreement by one sample was met with strong disagreement by the other sample. Only one item was endorsed by more than 25% of respondents from each sample (item 4: 'When I want to change the way I think, I get cravings for alcohol'). Thirty-seven percent of undergraduate students and 41% of treatment seekers strongly disagreed with this item.

The 22-item scale had good internal consistency (Cronbach's  $\alpha = .94$ ). Items were examined for their contributions to the scale as a whole, which are shown in Tables 3.2 and 3.3. One item, "I believe that when I feel good I'm more likely to get cravings", showed a corrected item-total correlation of .25 and was removed, leaving a 21-item scale (Cronbach's  $\alpha = .95$ ). Internal consistency for the antecedents and consequences sub-scales was good

Table 3.1



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#### *CMS Items, Means and Standard Deviations*

	Item	Treatment seekers		Students	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
1	I believe that when I feel good I'm more likely to get cravings.	2.77	1.42	2.70	1.11
2	My cravings lead to relapses.	3.77	1.25	2.27	1.19
3	How much I crave depends on how busy or active I am.	3.75	1.08	3.30	1.17
4	When I want to change the way I think, I get cravings for alcohol.	2.93	1.26	2.11	1.08
5	My cravings make me feel different physically.	3.43	1.31	2.37	1.19
6	My cravings occur frequently.	3.63	1.20	2.29	1.08
7	Cravings make alcohol seem almost irresistible.	3.98	1.22	2.51	1.23
8	I get cravings because I have no control over my drinking	3.70	1.36	1.68	0.81
9	My cravings will make me feel negative and aroused (e.g., angry, anxious, stressed, out of control).	3.60	1.36	1.97	1.01
10	There is nothing in my life that affects my cravings.	2.29	1.25	1.96	0.84
11	How much I crave depends on whether I am drinking or have had a drink recently.	3.88	1.19	3.34	1.28
12	I don't cope with social situations as well if I am craving.	3.78	1.20	2.14	1.03
13	Cravings change the way I act for the worse.	3.59	1.19	2.16	1.07
14	When I want to change the way I feel I get cravings.	3.27	1.21	2.00	1.04
15	My cravings last a long time.	3.35	1.24	1.99	0.93
16	I can't control my cravings.	3.23	1.39	1.75	0.89
17	My cravings are the result of alcohol still being processed by my body.	3.33	1.37	1.90	0.98
18	My cravings interfere with my life.	3.74	1.24	1.68	0.88
19	When I want to change the way I act I get cravings for alcohol.	3.02	1.28	1.95	1.05
20	My cravings will make me feel down.	3.60	1.18	1.96	0.90
21	I believe my cravings are the result of people, places or things that remind me of alcohol.	3.20	1.41	3.02	1.29
22	I believe that when I feel upset I'm more likely to get cravings.	4.15	1.15	2.83	1.36

## CHAPTER 3: DEVELOPMENT OF THE CMS

(Cronbach's  $\alpha$  for antecedents = .85; for consequences = .86) and no other items showed item-total correlations less than .30.

**Step 4: Refining for discriminant validity.** The 21-item scale examined for discriminant validity, to determine whether antecedents and consequences are distinguishable features of cravings. Initially, correlations between the CMS items and the MCQ-A subscales were examined (Tables 3.2 and 3.3). Five items were removed due to their strong relationship with the MCQ-A: items 12 (“I don’t cope with social situations as well if I am craving”), 13 (“Cravings change the way I act for the worse”), 15 (“My cravings last a long time”), 18 (“My cravings interfere with my life”), and 20 (“My cravings will make me feel down”). Item-total correlations were conducted for the remaining 16 items.

**Step 5: Refining for internal consistency.** Of the remaining 16 items, those which produced corrected item-total correlations of  $r \leq .40$  (poor contributors to the scale as a whole) were removed. These were items 3, “How much I crave depends on how busy or active I am”, 10 “There is nothing in my life that affects my cravings” and 21 “I believe my cravings are the result of people, places or things that remind me of alcohol”.

**Step 6: Checking scale internal consistency.** Correlations were re-run on the remaining 13 items to check all had corrected item-total correlations of  $r \geq .41$  with the total CMS and  $r \leq .52$  with the MCQ-A. No further items needed to be removed; the removal of items overly similar to the MCQ-A had not greatly reduced the CMS’s reliability. The final 13-item CMS is shown in the last column of Tables 3.2 and 3.3.

**Step 7: Factor analysis.** We conducted confirmatory factor analyses to determine whether a one- or two-factor (antecedents and consequences) model fitted the 13-item CMS

Table 3.2

*Process of Refining the Items of the CMS – Antecedents Items*

Item #	Item	Step 3	Step 4		Step 5	Step 6
			TAF	Unpl. SU		
Antecedents of Craving						
1	I believe that when I feel good I'm more likely to get cravings.†	-	-	-	-	-
3	How much I crave depends on how busy or active I am.	.35	.34*	.38*	.32*	.36
4	When I want to change the way I think, I get cravings for alcohol.	.63	.54*	.52*	.43*	.63
6	My cravings occur frequently.	.75	.68*	.58*	.44*	.75
8	I get cravings because I have no control over my drinking.	.80	.69*	.64*	.46*	.79
10	There is nothing in my life that affects my cravings. †	.30	.25*	.22*	.18*	.29
11	How much I crave depends on whether I am drinking or have had a drink recently.	.41	.31*	.34*	.25*	.41
14	When I want to change the way I feel I get cravings.	.74	.61*	.58*	.46*	.73
17	My cravings are the result of alcohol still being processed by my body.	.65	.58*	.56*	.46*	.62
19	When I want to change the way I act I get cravings for alcohol.	.68	.56*	.52*	.40*	.67
21	I believe my cravings are the result of people, places or things that remind me of alcohol.	.34	.27*	.26*	.22*	.35
22	I believe that when I feel upset I'm more likely to get cravings.	.62	.56*	.56*	.49*	.61

*Note.* Step 3 = Corrected item-total correlation; Step 4 = Correlation with MCQ-A subscales; Step 5 = Corrected item-total correlation after  $r < .53$  removed; Step 6 = Corrected item-total correlation after item-total correlations  $r < .40$  removed; TAF = Thought-Action Fusion; Unpl. = Unpleasantness; SU = Subjective Utility.

† Reverse coded

\* $p < .05$

Table 3.3

*Process of Refining the Items of the CMS – Consequences Items*

Item #	Item	Step 3	TAF	Step 4 Unpl.	SU	Step 5	Step 6
Consequences of Craving							
2	My cravings lead to relapses.	.76	.64*	.60*	.49*	.77	.78
5	My cravings make me feel different physically.	.67	.60*	.56*	.46*	.66	.66
7	Cravings make alcohol seem almost irresistible.	.80	.69*	.62*	.49*	.80	.81
9	My cravings will make me feel negative and aroused (e.g., angry, anxious, stressed, out of control).	.73	.60*	.59*	.49*	.70	.69
12	I don't cope with social situations as well if I am craving.	.79	.68*	.67*	.57*	-	-
13	Cravings change the way I act for the worse.	.72	.64*	.65*	.56*	-	-
15	My cravings last a long time.	.72	.63	.59	.51	-	-
16	I can't control my cravings.	.77	.71	.60	.47	.76	.77
18	My cravings interfere with my life.	.77	.67	.71	.58	-	-
20	My cravings will make me feel down.	.73	.61	.74	.68	-	-

*Note.* Step 3 = Corrected item-total correlation; Step 4 = Correlation with MCQ-A subscales; Step 5 = Corrected item-total correlation after  $r < .53$  removed; Step 6 = Corrected item-total correlation after item-total correlations  $r < .40$  removed; TAF = Thought-Action Fusion; Unpl. = Unpleasantness; SU = Subjective Utility.

\* $p < .05$

### CHAPTER 3: DEVELOPMENT OF THE CMS

best for each of the treatment seeking and undergraduate samples and for the samples combined. The fit indices for these are in Table 3.4.

Table 3.4

*Step 7: Confirmatory Factor Analysis of One- and Two-Factor Models of the 13-item CMS*

					RMSEA		WRMSR
	$\chi^2$	df	CFI	TLI	RMSEA	CI lower	CI upper
One-factor model							
Total sample	221.59	65	.97	.97	.11*	.09	.12
Clinical sample	183.13	65	.92	.90	.13*	.10	.15
Undergraduates	120.74	65	.97	.96	.10*	.07	.12
Two-factor model							
Total sample	218.60	64	.97	.97	.11*	.09	.12
Clinical sample	180.80	64	.92	.90	.13*	.11	.15
Undergraduates	119.22	64	.97	.96	.10*	.07	.12

*Note.* df = degrees of freedom; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index; RMSEA = Root Mean Square Error of Approximation; WRMSR = Weighted Root Mean Square Residual; CI = Confidence Interval.

\*  $p < .05$

All models met model fit requirements. The one-factor model was selected for parsimony and met recommendations of Tucker Lewis (TLI) and Comparative Fit (CFI) indices of approximately .95 (Hu & Bentler, 1999; Yu, 2002). Furthermore, the one-factor model produced a weighted root mean square residual (WRMSR) of less than 1.0, as recommended by Yu. Cronbach's  $\alpha = .93$  for this final model, indicating a high level of internal consistency.

**Step 8: Construct Validity.** Table 3.5 shows the correlations between the CMS and other measures for the treatment seeking and undergraduate samples. We ran correlations separately on the samples to check differential validity. This was important given item examination in Step 3 showed different response patterns depending on participant sample. Yet, in the confirmatory factor analysis, a single factor model was the best fit for both samples. Few correlations were significantly different between the samples; these are identified in Table 3.5.

Given that there were few significant differences in the correlations between the CMS and other measures across the two samples, tests of construct validity were conducted on the two samples combined. Positive relationships were anticipated, which would contribute to the scale's convergent validity. We anticipated the CMS would correlate most strongly with the CBQ and MCQ-A, given all address beliefs about cravings. The relationships observed between these measures showed that although the CMS is clearly correlated with similar measures, the relationships are of comparable strength to those between subscales within existing measures. For example, the strongest relationship between the total CMS and an MCQ-A subscale (Thought-Action Fusion) is  $r(207) = .81, p < .01$ , and the strongest relationship between two MCQ-A subscales (Thought-Action Fusion and Unpleasantness) is  $r(207) = .84, p < .01$  in our study. This indicates that the relationship between the CMS and MCQ-A is of comparable strength to the relationship of two subscales within the MCQ-A. Similarly, correlations were found between the CMS and other measures. The CBQ correlates strongly with the TAF subscale of the MCQ-A ( $r = .78$ ) and the JACQ-now ( $r = .70$ ), while the AUDIT correlates with the MCQ-A Unpleasantness subscale at  $r = .71$ . Broadly speaking, the CMS is as similar (or dissimilar) to the MCQ-A and CBQ as they are from each other, which previously has been an acceptable relationship.

Table 3.5

*Correlations Between Final 13-item CMS and Related Constructs*

		CMS						MCQ-A						CBQ			JACQ-now		
		TAF						Unpl.			SU								
		B	T	U	B	T	U	B	T	U	B	T	U	B	T	U	B	T	U
MCQ-A	TAF	.811*	.767*	.630*	-	-	-												
	Un	.758*	.548*	.652*	.840*	.674* <sup>†</sup>	.904* <sup>†</sup>	-	-	-									
	SU	.602*	.257* <sup>†</sup>	.686* <sup>†</sup>	.667*	.358* <sup>†</sup>	.834* <sup>†</sup>	.805*	.677*	.819*	-	-	-						
CBQ		.873*	.799*	.814*	.781*	.713*	.620*	.744*	.572*	.640*	.574*	.269* <sup>†</sup>	.644 <sup>†*</sup>	-	-	-			
JACQ-now		.698*	.603*	.597*	.606*	.501*	.431*	.521*	.293*	.430*	.405*	.137	.454*	.697*	.596*	.611*	-	-	-
AUDIT		.745*	.478*	.575*	.648*	.391*	.461*	.707*	.399*	.467*	.598*	.337*	.472*	.629*	.246*	.557*	.574*	.367*	.440*

*Note.* B = both samples; T = treatment seeking sample; U = undergraduate sample; CMS = Craving Metacognition Scale; MCQ-A = Metacognition Questionnaire for Alcohol Abusers; TAF = Thought-Action Fusion; Unpl. = Unpleasantness; SU = Subjective Utility; CBQ = Craving Beliefs Questionnaire; AUDIT = Alcohol Use Disorders Identification Test; JACQ-now = Jellinek Alcohol Craving Questionnaire-now

\*  $p < .05$

<sup>†</sup> Difference between the two samples is significant,  $p < .05$

### **Discussion**

This paper reports on the development and evaluation of a self-report craving metacognitions measure. Craving metacognitions are the beliefs an individual holds about their cravings as a cognitive experience (Hoyer et al., 2007; Beck et al., 1993), which can include beliefs about the antecedents and consequences of cravings. A 193-item pool based existing alcohol measures and craving research was generated via review of existing craving and drinking measures. From these measures, we extracted the semantic content to write craving metacognition items. The resulting scale was refined using samples of inpatient alcohol treatment seekers and undergraduate students.

The resulting scale, the CMS, has a simple factor structure, comprises 13 internally consistent items and converges with other craving metacognition measures, while appearing to retain good construct validity. We interpreted its discriminant validity as adequate given existing measures (CBQ and MCQ-A) were of comparable similarity to the CMS and the MCQ-A. It improves on existing measures as it is the first craving metacognition measure in English to address both antecedents, which were not addressed by the MCQ-A (Hoyer et al., 2007), and consequences, which were not addressed by the LCRR (Veltrup et al., 2001). Moreover, it was developed with methodical inclusion of a range of craving-related experiences, in contrast to the limitations imposed during the MCQ-A's development. Its factor structure has been confirmed, unlike the CBQ. The CMS performs comparably to the MCQ-A and CBQ but has fewer items for ease of response and parsimony. Testing the scale on a range of drinkers allowed for generalisation to drinking populations and avoids restricting range, an issue that is occasionally overlooked in craving questionnaire development (Hoyer et al., 2007; Statham et al., 2011).



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We found strong relationships between the CMS and other measures of craving metacognitions, indicating strong content validity, despite different face validity for items that address ‘craving’ (CMS) and ‘intrusive thoughts of alcohol’ (MCQ-A). These correlations were high compared to content validity correlations in other craving metacognition studies, yet this may be because other studies compare craving metacognition measures with general metacognition measures (e.g., Nosen & Woody, 2014), rather than comparing craving metacognition measures directly. The CMS may be a viable alternative to the existing measures. Correlations with craving and severity of alcohol problems were lower, as would be expected for less theoretically similar constructs.

The effect of alcohol problem severity on craving metacognitions is worthy of further investigation. Our samples of treatment seekers and undergraduate drinkers produced different response patterns, yet the same factor structure fit for both samples and there was limited differential validity between the two samples. This suggests that the CMS measured the underlying factor reliably and is valid for a range of drinkers. However, the relationships between cravings and craving metacognitions, and between the CMS and most other craving metacognition measures used here, are similar for treatment seekers and undergraduate drinkers. That is, drinking status does not change the way cravings affect craving metacognitions. When viewed with findings that craving metacognitions are subject to highly specific relationships within groups (Chapter 2; Nosen & Woody, 2009), we begin to develop a greater understanding of the ways craving metacognitions contribute to addiction experiences.

An alternative explanation may be that the undergraduate sample craved and interpreted cravings differently to the inpatient treatment-seeking sample. Rosenberg and

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Mazzola (2007) reported that mean craving scores for binge-drinking undergraduates were lower than those reported by clinical samples completing the same measures in other studies. Moreover, Slade, Teesson, Mewton, Memedovic, and Krueger (2013) found differences in the way young adults answer questions about alcohol use, which may have implications for the way young adults respond to questions about craving. However, these studies primarily addressed cravings and alcohol use, rather than craving metacognitions. Despite potential sampling differences, examination of an undergraduate sample contributes to broader exploration of the craving-metacognition relationship

The present study had a number of limitations. Author KT primarily developed the CMS items, which may have resulted in biased or restricted item selection. This could have been avoided with simultaneous, independent development with another researcher. The selected items resulted in predominantly negative metacognitions, due to existing measures' focus on negative craving experiences. Exploration of positive craving experiences is rare in alcohol metacognition models. Hoyer et al. (2007) included a very limited range of positive alcohol metacognitions in their smallest subscale and held only tentative support for it; moreover, although the metacognitions in this scale were positive because they described craving as useful, they were not positively valenced (e.g., where craving is enjoyable). Research into craving processes may help determine the role positively-valenced cravings and craving metacognitions play in addiction processes. In light of this, the CMS has limited ability to address metacognitive models of addiction processes fully (e.g., Spada et al., 2012).

An additional limitation may be that all participants received the questionnaire battery in the same order, which may have influenced responses, especially for the CMS, MCQ-A and CBQ given their similar content. For example, participants' responses in earlier measures

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may have primed them to make similar responses to comparable items in later measures. Such effects could falsely inflate correlations between measures, which may be of particular relevance given the high correlations between measures our study achieved. Future research investigating multiple measures of craving metacognitions should alternate the presentation order of questionnaires.

Finally, some items in the CMS share highly similar content with measures of craving severity. This makes it difficult to establish convergent validity with craving measures including the JACQ-now used here.

Additional research is required to determine the reliability and validity of the CMS, CBQ and MCQ-A, the predictive validity of the CMS should be ascertained. If it is a suitable alternative to existing measures, in time it may be a clinically useful tool. Metacognitive theory describes the relationship between metacognitions and psychopathology; accordingly, if the CMS is successful in predicting clinical outcomes (e.g., future craving or drinking episodes; level of anxiety or depression related to craving) it will provide additional evidence for the usefulness of metacognitive theory.

In conclusion, craving metacognitions constitute the beliefs and evaluations individuals make about their cravings. Craving metacognitions are clinically important as they have the potential to exacerbate cravings as unhelpful cognitions; yet existing craving metacognition measures were limited in their development and breadth of items. We addressed these issues by developing a new single-factor, internally consistent and potentially psychometrically valid 13-item measure, the Craving Metacognition Scale. This measure

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provides an alternative to existing craving metacognition measures as it performs comparably, but requires further testing to confirm its reliability and validity.



## **Chapter 4**

### **Metacognitions Relate to Cravings Following Cue Reactivity**

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### **Abstract**

Cravings and craving metacognitions are assumed to influence one another, yet a recent study found only highly specific relationships between the two (Chapter 2). The present study extends that work by increasing the sample size, testing individuals with a broader range of drinking experiences, using a new measure of metacognitions (the CMS; Chapter 3), and testing an earlier drinking phase to avoid alcohol effects on cognitive processes. Metacognitions appeared to possess trait-like qualities; accordingly, we used cues to activate metacognitions, to test whether this influences cravings. A sample of 78 individuals scoring either less than 8 (low-risk drinking) or more than 16 (high-risk drinking) on the AUDIT completed cue reactivity trials of listening to audio recordings and smelling water, then alcohol. Participants completed multiple metacognition measures, and self-report cravings and physiological indicators of craving were recorded. We successfully induced cravings, which increased significantly through baseline, water- and alcohol-reactivity trials. Metacognition ratings were comparable to those in other studies. The CMS showed a significant relationship with cravings in this study. Craving metacognitions did not differ significantly between low- versus high-risk drinkers.

*Keywords:* Drinking, alcohol, craving, metacognition, cue reactivity.





### **Metacognitions Relate to Cravings Following Cue Reactivity**

Metacognitions are the beliefs, knowledge and regulation attempts that an individual holds about their thoughts or cognitions (Spada & Wells, 2008). General metacognitive theories assume cognitions relate to metacognitions (Wells & Matthews, 1996), yet to date no theory or model has described craving metacognitions and how they may relate to cravings. Recent craving research has suggested that nicotine cravings are related to metacognitions of personal relevance (Nosen & Woody, 2014) and that metacognitions may be related to alcohol consumption (Chapter 2). Although such research findings are highly specific, broader relationships may depend on the way drinkers experience craving.

Cravings are intrusive thoughts about alcohol (Chapter 2). Researchers often examine cravings with dependent and/or treatment seeking individuals (e.g., Bottlender & Soyka, 2004; Oslin, Cary, Slaymaker, Collieran, & Blow, 2009; Pavlick et al., 2009), yet a range of drinkers experience cravings. Cravings have been observed in non-treatment seeking samples (e.g., Kruse et al., 2012), university students (e.g., Rosenberg & Mazzola, 2007) and general community samples of non-dependent drinkers (e.g., Connor et al., 2014). Greater understanding of cravings, craving metacognitions and related processes can be achieved by examining a range of drinkers.

Alcohol craving experiences differ within groups. Rosenberg and Mazzola (2007) reported that university students' alcohol craving varied depending on terminology used (e.g., 'craving', 'urge', etc.). Students' craving in this study was lower than for those with alcohol use disorder in other studies. Effects like these may be indicative of genuinely lower craving due to group differences, measurement issues such as differential interpretation of questions (Slade et al., 2013), or both.

## CHAPTER 4: METACOGNITIONS RELATE TO CRAVINGS

Such differences in craving experiences may result in differences in craving metacognitions. Alcohol metacognitions may differ between dependent and non-dependent drinkers. Aslan et al. (2012) found alcohol craving beliefs measured by the Metacognitions Questionnaire for Alcohol Abusers (MCQ-A; Hoyer et al., 2007) were similar in alcohol abstainers and social drinkers, but different for alcohol-dependent individuals. There is currently no information available regarding the relationship of cravings and metacognitions aside from in substance-dependent individuals (Nosen & Woody, 2014; Chapter 2). Investigating cravings held by a range of drinkers may provide insight into metacognition-craving relationships by sampling a wider range of the phenomena.

To date, little is known about the relationship between cravings and metacognitions. Many studies addressing craving metacognitions have not simultaneously measured craving (e.g., Aslan, Türkçapar, Eser & Uğurlu, 2012; Lee et al., 2010). Hoyer et al. (2007) included an amended version of the Obsessive Compulsive Drinking Scale (OCDS; Anton et al., 1995). However, their use of this measure was primarily to determine the validity of their own new measure, the Metacognitions Questionnaire for Alcohol Abusers (MCQ-A), rather than to establish the relationship between the measure and cravings themselves. Nevertheless, they observed significant positive relationships between the OCDS and the MCQ-A's Thought-Action Fusion and Unpleasantness subscales. Nosen and Woody (2014) also included the OCDS in their study, and found specific relationships between cravings and metacognitions of perceived personal significance of cravings. Both these studies used treatment-seeking individuals rather than broader samples of those who use substances, limiting understanding of the construct and the findings' generalisability.

## CHAPTER 4: METACOGNITIONS RELATE TO CRAVINGS

Although these studies have reported relationships between craving and metacognitions, they are yet to be replicated. In a study of 39 alcohol treatment-seeking participants, we found alcohol cravings did not predict alcohol craving metacognitions, despite significant craving change and significant relationships between beverage consumption and metacognitions (Chapter 2). In that study, we assumed cues would trigger cravings, which would activate metacognitions; and that both cravings and metacognitions would vary with manipulation. Although metacognitions can change over time (e.g., Loeber et al., 2006), we speculated the null result may have been due to a genuine lack of relationship. We further considered that the reasons metacognitions did not change may be due to trait-like qualities, or to measurement issues with the MCQ-A.

The MCQ-A only includes metacognitions that are consequences of cravings, and in areas the authors felt were clinically relevant (Hoyer et al., 2007), rather than a fuller range of consequences identified by other researchers (e.g., Toneatto, 1999b). Perceived antecedents of cravings are not addressed at all, and the measure was developed exclusively with dependent, treatment-seeking drinkers, limiting the breadth of craving metacognitions and consequently the degree to which the construct can be understood. The appropriateness of the measure for non-dependent or non-treatment seeking samples is unknown. To overcome these issues, we developed the Craving Metacognitions Scale (CMS; Chapter 3), developed with samples of inpatient alcohol treatment seekers and undergraduate drinkers. The CMS items are based on semantic content extracted from existing craving measures and written as craving metacognitions, providing a broader range of both craving antecedents and consequences. The resulting measure was an internally consistent, single-factor measure that may provide an alternative craving metacognition measure to those currently in use, particularly for assessing the relationship between cravings and metacognitions.

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Previous research into the relationship between metacognitions and cravings has focused solely on self-reported cravings. Physiological indicators of craving, such as changes in heart rate (Kaplan et al., 1985), skin conductivity (Nees, Diener, Smolka, & Flor, 2012), or saliva production (Pavlick, 2007) may also share a relationship with metacognitions. Early cognitive models (e.g., Ludwig et al., 1974) argued that craving was the act of labelling physiological experiences related to alcohol withdrawal. Craving theories have progressed considerably since that research. Yet, craving metacognitions may influence physiological indicators of craving.

Addiction research often uses cue reactivity methods to induce physiological responding by presenting substance use cues. A range of craving induction methods have been developed, such as presentation of substance use paraphernalia, images or footage of individuals using the substance, olfactory cues, or even substance consumption. After cues are provided to individuals, researchers often measure physiological responding, which can include heart rate, skin temperature, and skin conductance (sweat gland activity). Carter and Tiffany (1999) conducted a meta-analysis of these methods across studies of alcohol, nicotine, heroin and cocaine dependent people, finding that, for alcohol-dependent participants, effects observed in descending order of strength were self-report craving, heart rate, sweat gland activity then skin temperature. Overall, alcohol-dependent participants showed the smallest effects of any substance. More recent research shows that cue responsivity may be highly individual (Szegedi et al., 2000).

The influence of physiological indicators of craving remains an important consideration given they are particularly strong in dependent drinkers compared to non-dependent individuals (Nees et al., 2012). Szegedi et al. (2000) found alcohol dependent

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men's responses tended to cluster by those who were more responsive to cues that were psychological, physiological, both or neither. Differential responsiveness of this nature may provide internal triggers for craving metacognitions. If physiological indicators of craving activate individuals' craving metacognition schemata, individuals may interpret these physiological experiences as cravings.

Only the study in Chapter 2 has used cue reactivity to study the craving-metacognition relationship, providing environmental cues, a priming dose of alcohol for some participants (pharmacological, psychological, gustatory and olfactory cues) and environmental cue plus a placebo for a second group of participants (psychological, gustatory and olfactory cues). Our assumption was that cues would influence craving, which would influence metacognitions. In contrast, the findings demonstrated that cues had a direct effect on metacognitions, while cravings had no effect on metacognitions at all. Moreover, participants who believed they had received alcohol experienced a significant increase in Subjective Utility metacognitions measured by the MCQ-A. Measurement of alcohol expectancies may clarify the relationship between expectancies and metacognitions.

No experiment to date has examined the direct effect of cue reactivity on craving metacognitions. Cue reactivity studies often make use of neutral stimuli as a control condition. In some studies, neutral conditions use substances that do not afford eating (e.g., pot pourri; McCusker & Brown, 1995), probably confounding thirst or hunger with desire for alcohol by failing to provide a consumable alternative. Stronger studies (e.g., Coffey, Stasiewicz, Hughes, & Brimo, 2006; Thomas, Bacon, Randall, Brady, & See, 2011) provide water as a control condition, aiming to isolate thirst from desire for alcohol. This is

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particularly important given thirst may be a significant cue for an individual with an alcohol use disorder who regularly quenches thirst with alcohol.

Although significant relationships between cravings and craving metacognitions were not detected in Chapter 2, there is still cause to investigate a possible relationship between metacognitions and cravings. Metacognitive theory not specific to cravings argues that cognitions and metacognitions are cyclical, influenced by one another along with environmental triggers and affect (Koriat, 2007; Wells et al., 2009). Our Chapter 2 results may be due to a number of limitations within that study; namely, a sample that was restricted to treatment-seeking participants who passed strict inclusion criteria; restricted measurement of metacognitions; and alcohol effects on participants. Relationships may exist between cravings and metacognitions in a sample with greater variation in their drinking experiences.

The present study examined the relationship between cravings and craving metacognitions. Few studies have examined the craving-metacognition relationship; all of these have used a substance-dependent population and only very specific metacognition-craving relationships have been detected (Nosen & Woody, 2014). Accordingly, the present study built on this work in a number of ways. A larger sample was sought and included participants with a broader range of drinking experiences and fewer restrictions on diagnoses. Participants in the present study did not consume alcohol during the experiment, and we controlled for thirst by providing water as a neutral yet consumable stimulus. Finally, this study used several measures of alcohol-related metacognitions, to ensure measurement of the construct that is more comprehensive.

We aimed to investigate further the relationship between metacognitions and cravings. No metacognitive change was observed in Chapter 2, making it difficult to determine whether

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cravings influence metacognition changes. Here, we suggest cues may activate metacognitions directly, which in turn amplify an individual's cravings. We provided the same naturalistic setting used in Chapter 2, but changed the study to a within-participants design, using water as the control substance. Based on our Chapter 2 findings, we anticipated that metacognitions are trait-like, and will not change between trials. We proposed that in the presence of activated metacognitions, manipulating alcohol-related cues would produce differential craving between trials. We predicted stronger craving in the face of alcohol stimuli compared to water stimuli. We hypothesised that if these assumptions were met, baseline metacognitions would be significantly related to water- and alcohol-trial cravings. Given the foci of our metacognition and expectancy measures, we further hypothesised that the CMS would relate most strongly to craving, followed in order by the MCQ-A, PAMS/NAMS and AOEM.

### Method

#### Participants

Seventy-eight participants who drank at least monthly were recruited from universities and technical colleges, youth hostels, and bars in Sydney, Australia, along with community websites. We anticipated that 78 participants would be required to detect moderate relationships of .40 between craving and metacognition measures, with  $\alpha = .05$  and power = .80. Two hundred and seven potential participants were screened. To avoid range restriction that may have occurred in Chapter 2, only individuals who scored below 8 (low-risk drinkers) or above 16 (high-risk drinkers) on the Alcohol Use Disorder Identification Test (AUDIT; Babor et al., 2001) were included. To retain some comparability with strategies applied in Chapter 2, participants were excluded if they received any diagnosis from the Anxiety Disorders Interview Schedule for DSM-IV (ADIS; DiNardo, Brown, & Barlow, 1994) other than an alcohol-use disorder, social phobia, or specific phobia.



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The resulting sample comprised 40 females (51%) and 38 males (49%), aged 18-55 ( $M = 22.9$ ,  $SD = 6.23$ ), including 40 low-risk drinkers (aged 18-55,  $M = 23.4$ ,  $SD = 7.30$ ) and 38 high-risk drinkers (aged 18-40,  $M = 22.3$ ;  $SD = 4.89$ ). Twenty-two of the high-risk drinkers (58%) were alcohol dependent and a further five (13%) met DSM-IV criteria for alcohol abuse (American Psychiatric Association, 2000). Participants varied in recruitment source. Sixty participants (77%) were university students and 18 (23%) were recruited from other locations. Specific numbers of participants recruited from each non-university location were not recorded.

### Measures

**Diagnoses.** We initially screened participants for key symptoms of psychological issues using a series of questions devised by a clinical psychologist for this purpose. We further screened for alcohol use with the AUDIT, an accurate measure for detecting alcohol use disorder amongst university students. It has high specificity and sensitivity for identifying problem drinking (Dhalla & Kopec, 2007), and is equally valid for men and women (Babor et al., 2001). Participants meeting AUDIT inclusion criteria were invited to attend a clinical interview conducted by a graduate student (either author KT or MS), trained by author AB. The interviewer administered the ADIS (DiNardo et al., 1994). The ADIS is used for diagnosing mood and comorbid substance use disorders (Segal & Williams, 2014). It has shown excellent inter-rater reliability ( $K = .83$ ) for alcohol use disorder (T. A. Brown, DiNardo, Lehman, & Campbell, 2001), and has shown good inter-rater reliability with graduate interviewers (Segal & Williams, 2014). Interviews were audio recorded, and 20% of interviews were cross-checked between interviewers. Any discrepancies of greater than a one-point difference on the eight-point clinical severity scale were discussed between

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interviewers, and where necessary clinical supervision was sought from author AB. Using this method, consensus was reached on all participants.

**Psychophysiological measurement.** We recorded heart rate and galvanic skin response (GSR), as they can be responsive to alcohol-related cues (Kaplan et al., 1985). Heart rate was measured using a three-lead electrocardiogram (Giardino, Lehrer, & Edelberg, 2002); disposable electrodes (AD Instruments, 2009) were placed on the participant's right wrist and on each arm, slightly above the cubital fossa. Galvanic skin response (GSR) was measured via MLT117F GSR Electrodes for fingertips (AD Instruments, 2009) on the participant's non-dominant hand, on the middle phalanges of the first and second fingers. Heart rate data were recorded using the PowerLab 4/25T system (AD Instruments, 2009). GSR data were amplified via an ML116 GSR Amplifier into a computer operating LabChart Pro 7.1.1 software (AD Instruments, 2009). Baseline GSR and ECG were recorded for five minutes, while participants watched a DVD of nature scenes set to classical music, as described in Chapter 2. The GSR electrodes failed during one participant's session, leaving complete data for 77 participants for that variable.

**Questionnaires.** Participants received questionnaires in the same order that they are presented here. They completed the Alcohol Urge Questionnaire (AUQ; Bohn et al., 1995) as a measure of in situ cravings. Items are rated on a 1 (*strongly disagree*) to 7 (*strongly agree*) scale. The measure has high internal consistency ( $\alpha = .91$ ), good test-retest reliability ( $r = .82$ ), and good concurrent validity with the Obsessive Compulsive Drinking Scale ( $r = .42$ ; Bohn et al., 1995). It has demonstrated good construct validity under conditions of neutrality ( $r = .34$  with degree of alcohol dependence) and cue reactivity ( $r = .31$  with amount of alcohol consumed ad libitum), in support of a single-factor structure (MacKillop, 2006).

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Craving metacognitions were measured by the Metacognition Questionnaire for Alcohol Abusers (MCQ-A; Hoyer et al., 2007), which has three subscales: Thought-Action Fusion, Unpleasantness and Subjective Utility. These subscales' items are answered using a scale of 1 (*strongly disagree*) to 5 (*strongly agree*), and have respectively attained internal consistency of  $\alpha = .86$ ,  $\alpha = .91$  and  $\alpha = .85$ . Correlations between the three subscales with the Obsessive Compulsive Drinking Scale was  $r = .52$ ,  $r = .34$  and  $r = .07$  respectively, and with the White Bear Suppression Inventory (alcohol version),  $r = .53$ ,  $r = .64$  and  $r = .20$ , indicated moderate support for concurrent validity. The MCQ-A showed interscale correlations of  $r = .53$  between Thought-Action Fusion and Unpleasantness,  $r = -.16$  between Thought-Action Fusion and Subjective Utility, and  $r = .20$  between Unpleasantness and Subjective Utility (Hoyer et al., 2007). Our recent research with the MCQ-A has shown moderate to strong relationships with the Jellinek Alcohol Craving Questionnaire-now (JACQ-now; Ooteman, Koeter, Verheul, Schippers, & Van den Brink, 2006b;  $r = .61$ ,  $r = .52$  and  $r = .41$  respectively) and AUDIT ( $r = .65$ ,  $r = .71$  and  $r = .60$  respectively) in a sample of undergraduate drinkers and inpatient treatment seekers (Chapter 3).

A second measure of craving metacognitions, the CMS (Chapter 3; Appendix E) was used. This 13-item self-report questionnaire has a response scale of 1 (*strongly disagree*) to 5 (*strongly agree*), internal consistency of  $\alpha = .93$  and evidence for convergent validity with the MCQ-A of  $r = .81$  for Thought-Action Fusion,  $r = .76$  for Unpleasantness, and  $r = .60$  for Subjective Utility. Further support for convergent validity was found, as the CMS correlated  $r = .74$  with the AUDIT and  $r = .70$  with the JACQ-now (Ooteman et al., 2006b). It was included in addition to the MCQ-A to cover both craving antecedents and consequences, and due to problems identified in the development of the MCQ-A.

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We measured drinking metacognitions via the Positive Alcohol Metacognitions Scale (PAMS) and the Negative Alcohol Metacognitions Scale (NAMS; Spada & Wells, 2008). These measures have internal consistencies of  $\alpha = .81$  for PAMS – Emotional Self-Regulation (ESR; 8 items),  $\alpha = .87$  for PAMS – Cognitive Self-Regulation (CSR; 4 items),  $\alpha = .68$  for NAMS – Uncontrollability (Unc; 3 items) and  $\alpha = .72$  for NAMS – Cognitive Harm (CH; 3 items). Interscale correlations were ESR-CSR  $r = .47$ , ESR-Unc  $r = .14$ , ESR-CH  $r = .24$ , CSR-Unc  $r = .36$ , CSR-CH  $r = .32$ , and Unc-CH  $r = .57$  (Spada & Wells, 2008). These measures address thoughts of drinking, rather than beliefs and evaluations of cravings. All items were answered using a response scale of 1 (*do not agree*) to 4 (*agree very much*).

We included a measure of positive alcohol expectancies in lieu of activating such expectancies via alcohol administration, as we had done in Chapter 2. We used the Alcohol Outcome Expectancies Measure (AOEM; Kushner, Sher, Wood, & Wood, 1994). This 35-item questionnaire has a response scale of 0 (*not at all*) to 4 (*a lot*) and an internal consistency of  $\alpha = .86$  (Wood, Read, Pailfai, & Stevenson, 2001). This measure was selected as it was developed with university students (Kushner et al., 1994), and Monk and Heim (2013) identified age, gender and context as important factors in expectancy findings.

### **Procedure**

Screening for comorbid diagnoses was conducted first via telephone. We then assessed comorbid disorders using the ADIS (DiNardo et al., 1994). To retain comparability with the sample used in Chapter 2, we chose to exclude participants who met criteria for any Axis I disorder with the exception of specific phobias and social phobia. In addition, only

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participants with a preference for beer or wine were invited to participate, due to beverage cost.

Following the ADIS, participants attended a simulated bar, described in Chapter 2. All experimental sessions commenced between 2:30pm and 5:30pm to control for diurnal effects (Coffey et al., 2006). Participants abstained from any alcohol on the day of their appointment, and were required to have a breath alcohol concentration (BrAC) of 0.000, which all did. Participants completed expectancy and metacognition measures at baseline, and craving was measured at baseline and after each cue via AUQ, heart rate and galvanic skin response measurements.

**Cue reactivity stimuli.** Two cue reactivity trials were used: cue reactivity to water, and to alcohol. In the water trial, participants listened to an audio recording (transcript in Figure 4.1) describing a beach scene. Next, they watched the experimenter pour a glass of water, which they then held and smelled for three minutes (McCusker & Brown, 1995; Monti et al., 1999) while thinking as strongly as they could about what it would be like to drink the water. The alcohol trial replicated this procedure, although the audio recording described a bistro scene, and participants were provided red wine, white wine or beer to smell, based on their preference at screening. Wine was pre-measured to 100mL (10g alcohol) and 4.6% w/v beer was poured from a 250mL bottle (9g alcohol). Water was always presented prior to alcohol, as Monti et al. (1987) found that effects of water exposure were masked if alcohol is provided first.

### ***Control recording***

“You are sitting alone on the beach. You look up and a good friend of yours is walking towards you! They tell you they've brought you a beach towel -- you can see it in their hand. You notice the smell of laundry soap coming from the orange beach towel. You sit on the towel, and it feels rough against your skin, but it reminds you of holidays so it relaxes you. You pick up a bottle of sunscreen and the bottle is slightly greasy in your hand. You squirt some into your hand and you can smell it, it smells like coconut. The bottle makes a noise as you squirt more onto your palm. You want to put the sunscreen on quickly, you feel like you could get burnt soon. Suddenly you feel like everything's just right - here you are with your friend, sitting on the sand, and it's just so easy. The warmth from the sun is making your skin tingle a bit. This is such a relief, you've needed this break. You rub the cool, creamy sunscreen across your shoulders, onto your arms, down your legs, and it feels good against your skin. You stretch out on the sand and feel all of your muscles relaxing as the tension just melts away. You think about how much you enjoy being able to just take a break, and suddenly you can't wait to have more fun. This is exactly what you've needed; it feels better than anything has all week. You haven't even been here long before you're thinking about getting into the water.”

### ***Alcohol recording***

“You are sitting alone in a bistro. You look up and a good friend of yours is walking towards you! They tell you they've brought you your favourite alcoholic drink -- you can see it in their hand. You think about how you weren't going to drink, you've tried so hard not to. Maybe you will just leave it sitting there. You sit down together and start to have a chat. In spite of yourself, you find yourself reaching forward and taking the drink -- you can feel how smooth the glass feels against your hand, the thought of it's making your mouth water. You bring it to your lips and suddenly you can smell it, it's right there in front of you. You can hear the drink moving around in the glass, and you are really looking forward to it. You can't wait to taste it; your mouth is watering a lot now. Suddenly you feel like everything's just right -- here you are with your friend, having a chat and a drink, and it's just so easy. The drink hits your tongue and it's wonderful -- you can feel it moving around in your mouth and there's that taste, that taste that you've been waiting for. Such a relief. You can feel the liquid all around your mouth, around the inside of your cheeks; haven't you tried so hard at being good? You swallow, and suddenly you can't wait for the next mouthful. You take another mouthful and it's just as good, no, better than the last. You haven't even swallowed this mouthful before you are thinking about the next.”

*Figure 4.1:* Transcripts of audio recordings used for cue reactivity. The recordings were constructed to be as similar in structure, content and language as possible without the control recording inducing any kind of appetitive state for alcohol consumption. The sequence of events was similar and they described comparable levels of introspection to avoid diverting the individual's attention internally in one recording and externally in the other. The control recording was based on Versland and Rosenberg (2007) 'Combined guided imagery' but was amended to ensure the similarities above were met. The control recording was 267 words long and ran for 1 min 38s. The alcohol recording was 272 words and ran for 1 min 36s.

### **Preliminary data analyses.**

A number of steps were included in the data analysis. First, we checked low- and high-risk drinkers for comparability. Following this, we examined the effectiveness of the craving induction.

### **Manipulation checks.**

All analyses were conducted using SPSS v.19 (IBM Corp., 2010). Means and standard deviations are shown in Table 4.1. Data were examined for normality and transformed appropriately. Initial examination of the sample showed the non-university-recruited participants comprised significantly more high- than low-risk drinkers, while the university-recruited participants comprise significantly more low- than high-risk drinkers,  $\chi^2(1) = 9.01$ ,  $p = .003$ . Additionally, the participants recruited from outside the university were significantly older,  $F(1, 73) = 17.20$ ,  $p < .001$ , but did not differ by gender,  $\chi^2(1) = 1.14$ ,  $p = .286$ . Accordingly, participants' drinking status (high vs. low risk) was included in the main analyses as a covariate.

The craving induction was successful. Self-report cravings increased significantly between the water and alcohol trials for both low- and high-risk drinkers,  $F(1, 76) = 62.97$ ,  $p < .001$ ,  $\eta_p^2 = .453$ , although there was no significant effect of drinking status, in contrast to recent findings in a similar experiment (Papachristou, Nederkoorn, Corstjens, & Jansen, 2012). There was no significant change in galvanic skin response, regardless of drinking

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Table 4.1

*Means and Standard Deviations for Cravings and Metacognitions for Low- and High-Risk Drinkers.*

Measure	Baseline <i>M</i> ( <i>SD</i> )		Water <i>M</i> ( <i>SD</i> )		Alcohol <i>M</i> ( <i>SD</i> )	
	Low risk	High risk	Low risk	High risk	Low risk	High risk
AUQ	16.68 (8.82)	25.53 (11.39)	20.00 (9.45)	28.79 (13.75)	25.28 (11.96)	37.39 (13.13)
Heart rate	67.21 (9.99)	67.68 (11.04)	67.23 (10.12)	67.05 (11.81)	67.36 (9.67)	68.15 (10.62)
GSR	18.05 (6.53)	20.76 (8.79)	27.28 (13.07)	35.47 (16.73)	28.27 (16.79)	37.33 (17.90)
AOEM	21.68 (13.86)	38.26 (20.31)	-	-	-	-
MCQ-A Total	28.13 (13.25)	36.42 (9.94)	-	-	-	-
TAF	13.00 (4.31)	15.26 (3.24)	-	-	-	-
Unpl	9.60 (5.90)	13.21 (4.68)	-	-	-	-
SU	5.53 (3.92)	7.95 (2.99)	-	-	-	-
CMS	24.95 (7.85)	32.03 (8.24)	-	-	-	-
PAMS ESR	19.35 (5.23)	22.79 (5.84)	-	-	-	-
CSR	5.13 (1.91)	6.32 (2.53)	-	-	-	-
NAMS UNC	3.20 (0.88)	3.76 (1.32)	-	-	-	-
COG	4.28 (1.87)	4.97 (1.70)	-	-	-	-

*Note.* Low-risk  $n = 40$ ; high-risk  $n = 38$ .

status,  $F(1, 75) = 3.44, p = .068, \eta_p^2 = .044$ . Due to a lack of homogeneity of covariance (Box's  $M = 30.62, p < .001$ ), separate analyses were conducted for assessing change in heart rate. We found no significant change in heart rate between water and alcohol trials for either



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low-risk drinkers,  $t(39) = -0.501, p = .619$ , or high-risk drinkers,  $t(37) = -1.38, p = .175$ .

Because heart rate and galvanic skin response did not change between the water and alcohol trials, we then made the broader comparison of baseline and alcohol trials. Here, there was a significant change in GSR,  $F(1, 75) = 156.10, p < .001, \eta_p^2 = .675$ . However, there was no significant change in heart rate between baseline and alcohol trials, regardless of drinking status,  $F(1, 76) = 0.567, p = .454, \eta_p^2 = .007$ .

### Results

The first hypothesis for this study was that metacognitions about alcohol would share a unique and significant relationship with craving in an alcohol reactivity task, where relationships would be stronger following exposure to alcohol cues. We examined the relationships between each of the metacognition questionnaires and the AUQ, heart rate and GSR craving measures for the whole sample using Pearson correlations (Table 4.2). AUQ scores were significantly related to a number of measures including the AOEM, CMS and some subscales of the MCQ-A, PAMS and NAMS. Of physiological indicators of craving, GSR showed no significant relationship with any of the AUQ, metacognition, or expectancy measures except for PAMS Cognitive Self-Regulation (baseline and water trials only). Heart rate showed no significant relationships with any alcohol-related self-report measures. These findings are consistent with some research, which has shown poor relationships between subjective craving and physiological indicators of craving, potentially due to specificity of response requirements (cf. Carter & Tiffany, 1999). However, given there was also no significant change in heart rate following alcohol presentation, heart rate data were not analysed any further.

Table 4.2

*Correlations between Measures of Craving, Drinking, and Associated Beliefs*

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.
1. AUDIT																			
2. Baseline AUQ	.51																		
3. Water AUQ	.44	.70																	
4. Alcohol AUQ	.51	.72	.84																
5. Baseline HR	.03	-.01	-.12	-.11															
6. Water HR	.02	-.01	-.14	-.11	.96														
7. Alcohol HR	.05	.01	-.14	-.11	.92	.93													
8. Baseline GSR	.12	.14	.29	.18	-.23	-.19	-.23												
9. Water GSR	.22	.11	.23	.25	-.13	-.11	-.15	.52											
10. Alcohol GSR	.21	.06	.24	.32	-.13	-.10	-.13	.47	.94										
11. AOEM	.53	.58	.46	.48	.08	.06	.09	.10	.13	.10									
12. Total MCQ-A	.36	.21	.18	.31	.10	.11	.15	.05	.04	.07	.47								
13. TAF	.30	.17	.13	.23	.11	.10	.14	.09	.04	.03	.39	.90							
14. Unpl.	.35	.19	.19	.33	.08	.10	.15	.06	.06	.09	.47	.97	.81						
15. SU	.34	.22	.17	.30	.11	.12	.13	-.01	.01	.05	.44	.92	.73	.86					
16. CMS	.49	.49	.40	.44	.01	.04	.05	.10	.15	.14	.56	.73	.59	.70	.75				
17. PAMS ESR	.37	.36	.26	.30	.06	.05	.03	-.07	.10	.07	.72	.36	.31	.33	.37	.39			
18. PAMS CSR	.30	.39	.31	.28	.11	.09	.04	.31	.19	.13	.67	.37	.36	.38	.28	.39	.39		
19. NAMS UNC	.31	.34	.31	.33	.12	.13	.13	.00	.10	.13	.47	.51	.38	.51	.53	.54	.48	.21	
20. NAMS CH	.22	.05	.01	.05	-.04	.01	.02	-.02	.18	.19	.15	.40	.34	.42	.35	.31	.16	.10	.39

*Note.* AUDIT = Alcohol Disorders Identification Test; AUQ = Alcohol Urge Questionnaire; HR = Heart Rate; GSR = Galvanic Skin Response; AOEM = Alcohol Outcome Expectancy Measure; MCQ-A = Metacognition Questionnaire for Alcohol Abusers; TAF = Thought-Action Fusion; Unpl. = Unpleasantness; SU = Subjective Utility; CMS = Craving Metacognitions Scale; PAMS = Positive Alcohol Metacognitions Scale; ESR = Emotional Self-Regulation; CSR = Cognitive Self-Regulation; NAMS = Negative Alcohol Metacognitions Scale; Unc. = Uncontrollability; CH = Cognitive Harm.

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Our second hypothesis was that relationships with craving would be strongest for the CMS, followed by the MCQ-A, PAMS/NAMS and AOEM in order. To test this, we conducted a repeated measures analysis in general linear model (GLM) for each of AUQ and GSR, shown in Table 4.3. We entered the CMS, MCQ-A, PAMS/NAMS and AOEM as covariates to detect the relationship between craving and alcohol-related metacognitions and expectancies. Variables were entered simultaneously to avoid any order effects. Drinking status (high- versus low-risk alcohol use as measured by the AUDIT) was entered as the between-subjects factor, and we included age and gender as covariates given the low- and high-risk drinkers differed significantly on these variables.

A significant relationship was observed overall between the AUQ and the CMS,  $F(1, 62) = 5.469, p = .023, \eta_p^2 = .081$ , not accounting for different AUQ timepoints and drinking status (high- versus low-risk). The effect size for this relationship was small ( $\eta_p^2 = .081$ ), yet still detected with only moderate chance of doing so (observed power = .634). This relationship was unique to CMS as no other measures of metacognitions showed a significant overall relationship with the AUQ (top half of Table 4.3), despite moderate relationships (Table 4.2). Interestingly, there was no significant relationship between high- versus low-risk drinking and cravings, which is inconsistent with earlier research (e.g., Lee, Greeley, Oei, & Dean, 2004). The relationships observed with AUQ were indicative of a trait-like effect for metacognitions, whereby metacognitions did not change rapidly (consistent with Chapter 2), so may only broadly influence states.

We observed different outcomes for our repeated measures analysis of physiological craving measured by GSR. Relationships between GSR and metacognitions were virtually non-existent. Only gender significantly related to GSR response, with a small effect size and moderately low power ( $\eta_p^2 = .048$ , observed power = .414). Gender differences in GSR

Table 4.3

*Between-Subjects Effects for AUQ and GSR*

Craving measure	Variable	<i>F</i> ( <i>df</i> 1, 62)	<i>p</i>	Partial $\eta_p^2$	Observed power
AUQ	Gender	3.136	.081	.048	.414
	Age	0.358	.552	.006	.091
	CMS	5.469	.023	.081	.634
	MCQ-A TAF	0.201	.655	.003	.073
	MCQ-A SUUT	0.208	.650	.003	.073
	MCQ-A Unpl	0.003	.956	.000	.050
	PAMS ESR	0.526	.471	.008	.110
	PAMS CSR	0.053	.818	.001	.056
	NAMS Unc	1.021	.316	.016	.169
	NAMS CH	3.146	.081	.048	.415
	AOEM	3.352	.072	.051	.438
	Drinking status	3.587	.063	.055	.462
GSR	Gender	29.187	<.001	.313	1.000
	Age	1.732	.193	.026	.254
	CMS	1.756	.190	.027	.257
	MCQ-A TAF	0.414	.522	.006	.097
	MCQ-A SUUT	0.046	.831	.001	.055
	MCQ-A Unpl	0.083	.744	.001	.059
	PAMS ESR	0.193	.662	.003	.072
	PAMS CSR	0.761	.386	.012	.138
	NAMS Unc	0.119	.731	.002	.063
	NAMS CH	0.017	.896	.000	.052
	AOEM	0.668	.417	.010	.127
	Drinking status	0.322	.572	.005	.087

*Note:* AUQ = Alcohol Urge Questionnaire; GSR = Galvanic Skin Response; CMS = Craving Metacognition Scale; MCQ-A = Metacognition Questionnaire for Alcohol Abusers; TAF = Thought-Action Fusion; SUUT = Subjective Utility; Unpl = Unpleasantness; PAMS = Positive Alcohol Metacognitions Scale, ESR = Emotional Self-Regulation; CSR = Cognitive Self-Regulation; NAMS = Negative Alcohol Metacognitions Scale; Unc = Uncontrollability; CH = Cognitive Harm; AOEM = Alcohol Outcome Expectancies Measure; Drinking status = low- versus high-risk drinker.

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readings following stimulus exposure are common (e.g., R. Brown & Macefield, 2014; Heishman, Lee, Taylor, & Singleton, 2010; Sharma & Gedeon, 2011), including differential sensitivity to cues (Heishman et al., 2010). Additionally, Drobes and Tiffany (1997) found GSR could be increased following exposure to substance use cues, but not via imaginal induction techniques.

Broadly, relationships were shown between cravings as a construct and specific metacognition measures. The CMS in particular showed the strongest relationship to cravings, indicating that craving antecedents and consequences may be relevant to individuals' craving levels. Given that overall there was a small effect, we investigated the possibility of more specific effects between baseline, water cues and alcohol cues. This allowed us to determine whether the provision of different cues influenced the relationship between metacognitions and cravings.

We chose to examine the most specific relationship first, between water and alcohol trials (Table 4.4). Here, we observed a significant relationship between the AUQ and the trial/age interaction,  $F(1, 64) = 4.207, p = .044, \eta_p^2 = .064$ . This indicated older participants experienced greatest cue reactivity, although the AUQ showed significant autocorrelation for all participants,  $F(1, 64) = 7.389, p = .008, \eta_p^2 = .106$ . No other significant relationships were observed for the AUQ between water and alcohol trials. GSR was only significantly related to the NAMS Uncontrollability subscales, whereby individuals rating cravings as less controllable experienced greater conductance. GSR did not relate significantly to any other metacognitions, expectancies or any other covariates in water/alcohol comparisons. Effect sizes for GSR were predominantly very small, with the study potentially underpowered to detect genuine effects. An absence of significant relationships here suggested that the water stimuli provided the same appetitive cue as alcohol.

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Table 4.4

*Trial by Metacognition Unique Relationship with Significant Craving Change between Water Reactivity and Alcohol Reactivity Trials*

Craving measure	Variable	<i>F</i> ( <i>df</i> 1, 64)	<i>p</i>	Partial $\eta_p^2$	Observed power
AUQ	Trial	7.389	.008	.106	.763
	Gender	3.389	.070	.052	.441
	Age	4.207	.044	.064	.524
	CMS	0.561	.457	.009	.114
	MCQ-A TAF	.005	.946	.000	.051
	MCQ-A SUUT	1.318	.255	.021	.204
	MCQ-A Unpl	1.406	.240	.022	.215
	PAMS ESR	3.133	.082	.048	.414
	PAMS CSR	0.501	.482	.008	.107
	NAMS Unc	3.841	.055	.058	.488
	NAMS CH	0.016	.899	.000	.052
	AOEM	0.473	.494	.008	.104
	Drinking status	0.072	.789	.001	.058
GSR	Trial	.696	.407	.011	.130
	Gender	.357	.552	.006	.091
	Age	.213	.646	.003	.074
	CMS	1.045	.311	.016	.172
	MCQ-A TAF	.046	.830	.001	.055
	MCQ-A SUUT	1.950	.167	.030	.280
	MCQ-A Unpl	.424	.517	.007	.098
	PAMS ESR	2.526	.117	.038	.347
	PAMS CSR	.000	.989	.000	.050
	NAMS Unc	4.012	.049	.059	.505
	NAMS CH	.816	.370	.013	.145
	AOEM	.166	.685	.003	.069
	Drinking status	.057	.812	.001	.056

*Note:* AUQ = Alcohol Urge Questionnaire; GSR = Galvanic Skin Response; Trial = Baseline/Alcohol trial contrast; CMS = Craving Metacognition Scale; MCQ-A = Metacognition Questionnaire for Alcohol Abusers; TAF = Thought-Action Fusion; SUUT = Subjective Utility; Unpl = Unpleasantness; PAMS = Positive Alcohol Metacognitions Scale; ESR = Emotional Self-Regulation; CSR = Cognitive Self-Regulation; NAMS = Negative Alcohol Metacognitions Scale; Unc = Uncontrollability; CH = Cognitive Harm; AOEM = Alcohol Outcome Expectancies Measure; Drinking status = low- versus high-risk drinker.

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Table 4.5

*Trial by Metacognition Unique Relationship to Significant Craving Change between Baseline and Alcohol Reactivity Trials*

Craving measure	Variable	<i>F</i> ( <i>df</i> 1, 64)	<i>p</i>	Partial $\eta_p^2$	Observed power
AUQ	Trial	42.976	<.001	.409	1.00
	Gender	2.386	.128	.037	.330
	Age	0.522	.473	.008	.110
	CMS	0.907	.345	.014	.155
	MCQ-A TAF	0.172	.680	.003	.069
	MCQ-A SUUT	0.032	.859	.001	.054
	MCQ-A Unpl	3.205	.078	.049	.422
	PAMS ESR	0.78	.782	.001	.059
	PAMS CSR	0.895	.348	.014	.154
	NAMS Unc	0.004	.948	.000	.050
	NAMS CH	2.500	.119	.039	.344
	AOEM	1.087	.301	.017	.177
	Drinking status	0.637	.428	.010	.123
GSR	Trial	44.493	<.001	.410	1.000
	Gender	7.594	.008	.106	.775
	Age	.030	.862	.000	.053
	CMS	.161	.690	.003	.068
	MCQ-A TAF	.251	.618	.004	.078
	MCQ-A SUUT	.003	.958	.000	.050
	MCQ-A Unpl	1.436	.235	.022	.219
	PAMS ESR	.588	.446	.009	.118
	PAMS CSR	.118	.732	.002	.063
	NAMS Unc	3.017	.087	.045	.402
	NAMS CH	.015	.903	.000	.052
	AOEM	.005	.946	.000	.051
	Drinking status	.781	.380	.012	.140

*Note.* AUQ = Alcohol Urge Questionnaire; GSR = Galvanic Skin Response; Trial = Baseline/Alcohol trial contrast; CMS = Craving Metacognition Scale; MCQ-A = Metacognition Questionnaire for Alcohol Abusers; TAF = Thought-Action Fusion; SUUT = Subjective Utility; Unpl = Unpleasantness; PAMS = Positive Alcohol Metacognitions Scale; ESR = Emotional Self-Regulation; CSR = Cognitive Self-Regulation; NAMS = Negative Alcohol Metacognitions Scale; Unc = Uncontrollability; CH = Cognitive Harm; AOEM = Alcohol Outcome Expectancies Measure; Drinking status = low- versus high-risk drinker.

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Having made our most specific comparisons between water and alcohol trials and finding few significant relationships, we elected to reduce the specificity of our comparisons by examining baseline and alcohol trials. This allowed us to determine the strength of relationship between metacognitions and cravings with the presentation of an appetitive cue; in this case, alcohol. There were few significant and meaningful relationships between cravings and metacognitions, expectancies and other covariates. No significant relationships were observed between baseline and alcohol-trial AUQ measurement, beyond significant autocorrelation,  $F(1, 64) = 42.976, p = <.001, \eta_p^2 = .409$ . This indicates that participants' beliefs, knowledge and other covariates did not contribute to their post-alcohol cravings.

A gender/trial interaction was significantly related to alcohol-trial GSR,  $F(1, 64) = 7.594, p = .008, \eta_p^2 = .106$ . This indicated men and women reacted differently to the presentation of alcohol cues, further depending on their baseline GSR reading. There is no evidence that participants' metacognitions played a role in this relationship. Finally, GSR showed significant autocorrelation,  $F(1, 64) = 44.493, p = <.001, \eta_p^2 = .410$ .

### Discussion

Craving metacognitions that are activated by environmental cues may influence an individual's experience of craving. No research has yet examined the influence of metacognitions on cravings; however, recent research has shown highly specific relationships between the two phenomena (Chapter 2). The present study exposed a range of drinkers to environmental alcohol cues and measured their alcohol-related metacognitions and expectancies, and their craving change when presented with different appetitive stimuli.

We hypothesised that in an alcohol-related environment, participants' metacognitions would relate significantly and uniquely to alcohol cravings. This hypothesis was addressed in



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two ways; by examining relationships between participants with two different levels of drinkers, and within participants over trials including water reactivity trial to control for appetitive influence. When examining differences between participants, CMS scores significantly and uniquely related to AUQ scores, but not to physiological measures of craving (HR and GSR), partially supporting our hypothesis. This difference was not related to low- versus high-risk drinking status, suggesting that the relationship between cravings and metacognitions is not a result of risky or heavy alcohol use.

When examining craving change within participants between the water and alcohol trials, few relationships were found between craving change and metacognitions or expectancies, providing no evidence for our hypothesis. Observed relationships in water/alcohol comparisons were limited to a trial/age interaction for the AUQ scores, a trial/NAMS Uncontrollability interaction for GSR, and autocorrelation for AUQ. The finding involving the NAMS Uncontrollability is not supportive as the subscale describes the respondent's ability to control their drinking, not their craving ("I have no control over my drinking", "My drinking persists no matter how I try to control it", "Drinking controls my life"; Spada & Wells, 2008, p. 519). Nevertheless, the finding may provide support for drinking metacognitions as they may relate to links between psychological stress and skin conductivity (e.g., Snelleman et al., 2014).

Baseline/alcohol trial comparisons showed a trial/gender interaction for GSR craving change and autocorrelation for both AUQ and GSR. All of these relationships produced small effects, but do not support our hypothesis of a relationship between craving metacognitions and cravings. Further research is required to test this.

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Our second hypothesis was that the CMS would outperform other measures of alcohol metacognitions and expectancies, by showing stronger relationships with craving. As noted above, the CMS was the only measure to relate uniquely to AUQ scores regardless of trial. Additional measures of craving metacognitions (MCQ-A), drinking metacognitions (PAMS and NAMS) and alcohol expectancies (AOEM) were not significantly related to any measure of craving. However, this relationship was not observed with more specific comparisons between trials. This finding is inconsistent with other studies that have found highly specific relationships between cravings and metacognitions for alcohol (Chapter 2) and nicotine (Nosen & Woody, 2014). As our study failed to replicate these specific relationships despite using some of the same measures (AUQ and MCQ-A; Chapter 2), further research is required to determine the nature and specificity of the craving-metacognition relationship. This is particularly important given the effects shown here are small.

The results of the present study may provide additional validation for the CMS (Chapter 3), developed to provide an alternative measure of craving metacognitions. Here, the results initially suggested the CMS is sensitive to a relationship between cravings and craving metacognitions, suggesting discriminant validity from other craving metacognition measures, which did not detect any relationships. Significant relationships between craving and the CMS, but not other metacognition measures, may be due to differences in development strategies. CMS comprises items that were written to describe semantic content extracted from existing craving measures. These items were tested on inpatient and undergraduate drinkers. In contrast, MCQ-A was developed using preselected clinically relevant variables as determined by clinicians, and administered only to inpatient samples. As the relationships between the CMS and self-reported craving following different stimuli were not significantly different, our findings suggest the relationship is more dependent on the trait-

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like nature of metacognitions, and that both craving antecedents and consequences should be measured.

Detection of craving metacognition relationships with AUQ but not with HR and GSR is not surprising, given previous findings. As acknowledged earlier, physiological measures are sometimes poor correlates with self-report craving (Carter & Tiffany, 1999). Research conducted after the present study showed heart rate variability may be a stronger measure of physiological indicators of craving (Quintana, Guastella, McGregor, Hickie, & Kemp, 2013). Given all of our measures except for GSR and HR involved explicit self-report of thoughts or behaviours, variance attributable to self-report methodologies likely contributed to the relationship, and would not have done so for the physiological variables, providing potential explanation for the negative relationship between AUQ and HR. Indeed, as noted by Kavanagh et al. (2013), poor correlation between physiological and self-report measures of craving does not invalidate the self-report measures.

An alternative explanation for the present study and previous studies (Chapters 2 and 3) is that metacognitions do not adequately describe cognitive processes related to craving. Although effects have been observed between cravings and metacognitions, the use of strict criteria in these and other studies (Nosen, 2012; Nosen & Woody, 2009) has produced only very specific relationships. The studies outlined above have addressed the content of craving metacognitions as an area for concern, yet these were conducted without the framework of a dedicated theory of craving metacognitions. Better conceptualisation and explanation of cognitive processes around craving may be provided by the elaborated intrusion theory of desire (EI; Kavanagh et al., 2005). This theory offers an explanation of the processes by

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which cues influence individuals' thoughts and emotions about drinking and the bidirectional impact these have on craving. Given the restricted performance of metacognitions in the studies discussed here, further exploration of EI may produce better results.

Our study contained a number of limitations that may have restricted our ability to find relationships that are more robust. Firstly, our study used water as a control substance and therefore minimised risk of thirst confounding our results. However, we did not enquire after participants' thirst level so we could control for it statistically (Nagy, 2012) or ask them to drink a glass of water to minimise thirst (e.g., Papachristou et al., 2012). Consequently, thirsty participants may have produced the same levels of cravings in the water and alcohol conditions, potentially explaining small effects. This is particularly important given many alcohol-dependent individuals use alcoholic drinks as their primary source of fluid intake and do not consume recommended amounts of water. For these individuals, perception of thirst may trigger or be interpreted as cravings for alcohol.

Secondly, the study did not counterbalance stimulus presentation. This decision was deliberate based on findings that alcohol-first presentations may mask effects of later cue (Monti et al., 1987). Given we are unable to rule out order effects on our results, future research would benefit from improving distractor tasks between stimuli presentations to ensure appropriate counterbalancing can be conducted (Carter & Tiffany, 1999).

Thirdly, our study used a measure of heart rate as an indicator of craving, when heart rate variability may have been a more valuable variable. Recent research has demonstrated that heart rate variability is related to craving (Quintana et al., 2013) and that heart rate

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variability biofeedback training may reduce cravings in young men with substance use disorder (Eddie, Kim, Lehrer, Deneke, & Bates, 2014).

A fourth limitation of our study is that 77% of our participants were university students who participated during semester, many of whom reported anecdotally that their drinking patterns varied greatly depending on the time of year (e.g., exam versus holiday periods). Questions in the AUDIT that address year-long drinking patterns may have been due to drinking incidents that had occurred months prior, with considerably lower drinking since. This may have accounted for our inability to find differential effects for low- versus high-risk drinkers.

The fifth limitation to our study is in our measurement of craving metacognitions and expectancies. Both the CMS and the MCQ-A contain only metacognitions of craving that is perceived to be a negative experience. Conversely, AOEM includes only positive alcohol expectancies; that is, expectancies that alcohol consumption will have positive effects for the individual. Evaluation of positive craving metacognitions, of which there are currently no dedicated measures; or negative expectancies, such as those in the Drinking Expectancy Profile (Young & Oei, 1996) (Young & Oei, 1996) or the Negative Alcohol Expectancies measure (Jones & McMahon, 1994) may rectify this issue.

A sixth limitation is that multicollinearity may have been an issue in our analyses. Several of the questionnaires in our repeated measures analyses were strongly related, which may have weakened our analyses (Tabachnick & Fidell, 2013). Almost all of the high correlations that indicated multicollinearity involved the MCQ-A, suggesting that our analyses may have been stronger without it. The CMS showed more acceptable correlations with other measures, making it a more appropriate measure to include. Although knowledge

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of the reliability and validity of the CMS and MCQ-A is preliminary, strong correlations between MCQ-A subscales across a number of studies to date (the present study; Chapter 2; Hoyer et al., 2007) suggest it will be a difficult measure to implement in multivariate analyses, further supporting the use of the CMS.

Finally, participants received some questionnaires outside the laboratory, where we were unable to control the conditions under which they were completed, and all participants received their questionnaires in the same order, which may have produced order effects on their responses. This is particularly problematic given we anticipated differences between questionnaires; future research should randomly present questionnaires to avoid confounding effects.

Our study tested the relationship between craving metacognitions and cravings in two groups of drinkers. Metacognitive theories suggest a relationship between cognitions (such as cravings) and metacognitions (such as craving metacognitions), yet these relationships have only received limited attention, and rarely under experimental conditions (e.g., Nosen & Woody, 2014; Chapter 2). Cue reactivity paradigms and experimental procedures thoroughly tested our hypotheses of relationships between craving and metacognition measures, especially the CMS. These were supported; however, no differences were observed in metacognitions of low- versus high-risk drinkers, suggesting that craving metacognitions are influenced by variables other than drinking status. Additional research should investigate craving amplification resulting from beliefs or interpretations of cravings.

## **Chapter 5**

### **Interoceptive Accuracy Task Interferes With Rather Than Amplifies Craving in a Cue Reactivity Task**

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### **Abstract**

Craving involves detecting and processing internal sensations. Using anxiety sensitivity and elaborated intrusion theories, the present study tested the relationship between cravings and individuals' internal awareness. Enhanced interoceptive accuracy while craving, and increased craving following high heart rate detection accuracy, would indicate support for sensitivity models such as AS and SSA. Diminished interoceptive accuracy and a decrease in craving following an interoceptive task requiring high heart rate detection accuracy would indicate support for elaborated intrusion theory, which describes limitations on cognitive resources. Forty-four participants completed the AUQ, a self-report measure of craving, along with self-report measures of anxiety sensitivity, somatosensory amplification and elaborated intrusions. Participants' salivation production volume was measured during exposure to alcohol cues, and they completed heart rate detection tasks as a measure of interoceptive ability. Results from this study indicate that baseline craving is related to accurate heart rate detection, which in turn predicts later self-reported craving. However, anxiety sensitivity, somatosensory amplification and elaborated intrusion are not significantly related to these phenomena. The results are consistent with EI Theory and contribute to the known influences on cravings.

*Keywords:* Interoception, elaborated intrusions, alcohol, craving, anxiety sensitivity, heart rate detection



### **Interoceptive Accuracy Task Interferes With Rather Than Amplifies Craving in a Cue Reactivity Task**

As a subjective cognitive-emotional experience, cravings for alcohol involve internal awareness, or interoception. Cravings are notably a multidimensional experience with physiological indicators, to which individuals respond differentially (Szegeedi et al., 2000). Recently, research into variation of craving experiences focused on the relationships between craving and craving metacognition content (Chapters 2 and 4), yet only limited relationships were observed. Accordingly, an alternative approach to individual craving variation may be to focus on the process of craving, rather than the content. Theories differ in their explanation of individuals' interpretation of sensations. Anxiety sensitivity and somatosensory amplification models propose attending to sensations will exacerbate emotional state, while EI theory posits that elaboration of thoughts arising from such sensations can be alleviated by distraction. Sensitivity to sensations that occur during the craving process may contribute to craving variation between individuals (Verdejo-Garcia, Clark, & Dunn, 2012).

Interoception is the perception of sensations within the self. Interoceptive sensitivity refers to the degree to which individuals are able to detect such sensations. A recent research review has drawn links between interoception and craving (Verdejo-Garcia et al., 2012), arguing that the differing levels of interoceptive sensitivity may account for variation in the relationship between subjective and physiologically-measured craving (e.g., changes in salivation, heart rate and galvanic skin response). Interoceptively sensitive individuals may tend to interpret physiological symptoms as indications of craving, akin to early cognitive labelling theories (e.g., Ludwig et al., 1974), and may find cravings and withdrawal symptoms more aversive. Verdejo-Garcia et al. argue that this process of labelling occurs when interoceptive cues become conditioned stimuli for substance use.

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Interoceptive ability is often measured by asking participants to report on sensations or perceivable bodily functions, often associated with autonomic arousal. The most common method is the heart rate detection task (HRDT; e.g., Schandry, 1981; cf. Domschke, Stevens, Pfleiderer, & Gerlach, 2010). HRDTs were developed to investigate the assumption that emotional experience related to perception of physiological processes. They remain one of the stronger measures of interoceptive ability, and out-perform other measures such as detection of blood pressure (Fahrenberg, Franck, Baas, & Jost, 1995).

Individuals with high interoceptive sensitivity may have greater capacity for making associations between alcohol and conditioned effects (such as relaxing when they sit down with a drink at the end of the week) as they are more aware of certain sensations (e.g., a decrease in muscle tension). Individuals who have conditioned effects may develop expectations that invert cause and effect when interpreting sensations. An example of this may be, “Whenever I want a drink, my hands get sweaty. My hands are currently sweaty; therefore, I must want a drink.” In short, a change in sensations may be a conditioned stimulus for an alcohol-dependent person (Weingarten & Elston, 1990). This change may be particularly pertinent for thirst cues; for regular drinkers, alcohol may be their usual beverage for quenching thirst, rather than water or another beverage. Thus, innate appetitive cues such as thirst may be interpreted as cravings.

Early alcohol craving and interoception research predominantly investigated alcohol effect detection and the relationship these effects may share with craving (e.g., Greeley, Lê, Poulos, & Cappell, 1984; Ludwig et al., 1974; Weingarten & Elston, 1990). More recently, Szegedi et al. (2000) identified that individuals typically fit one of four categories in response to a cue reactivity task. Szegedi and colleagues’ sample was clearly divisible into groups who experienced physiological craving, subjective craving, neither or both. Verdejo-Garcia et al.

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(2012) argued that the underlying explanation for this separation may be interoceptive accuracy, whereby individuals who are more interoceptive experience both physiological and subjective cravings. However, to date there are limited if any experimental data available about the relationship between more general interoceptive ability and subjective cravings. Nevertheless, interoceptive cues to which the individual is sensitive, such as a change in salivation, may indicate physiological craving (e.g., Greeley et al., 1984; Ludwig et al., 1974; Weingarten & Elston, 1990).

Alternatively, exteroceptive and interoceptive cues may interact to produce subjective cravings. For example, the smell of alcohol may remind an individual of other drinking episodes, which in turn evokes a change in saliva production. If the individual attends to any or all of these experiences, they may develop a strong and intrusive urge to consume alcohol. Physiological indicators of craving that have been used in previous research include increased salivation (Kambouropoulos & Staiger, 2009; Monti et al., 1987), where alcohol-dependent participants salivated more than non-alcohol-dependent participants in response to alcohol, and increased heart rate and skin conductivity (Stormark, Laberg, Bjerland, Nordby, & Hugdahl, 1995). Alcohol-dependent individuals further showed greater heart rate acceleration and greater skin conductance after smelling beer than social drinkers (Stormark et al., 1995). Such reactivity is not always replicated (Chapters 2 and 4). Few studies investigate the role of subjective craving in influencing physiological indicators of craving.

There are two possible ways that subjective craving may influence physiological craving indicators. Firstly, individuals may become more sensitive to their internal environment (Gray & Critchley, 2007). Recent research by Dunn et al. (2010) showed interoceptive ability is related to cognitive-affective processing and intuitive decision-making. In this study, participants who showed greater interoceptive accuracy also had stronger links

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between physiological and subjective arousal following exposure to emotional images. Increased sensitivity to internal state has been discussed using concepts such as somatosensory amplification (Dunn, Dalgleish, Ogilvie, & Lawrence, 2007) and anxiety sensitivity (Domschke et al., 2010). Alternatively, individuals may become less sensitive to sensations as craving occupies their attention. This outcome is consistent with the elaborated intrusion theory of desire (EI; Kavanagh et al., 2005). Each of these proposals will be discussed below.

Somatosensory amplification (SSA) involves increased attention to sensations, selective attention to infrequent sensations and the cognitive-emotional intensification of symptoms. The individual assigns greater significance than is warranted to symptoms, resulting in reactions of excessive emotion and maladaptive cognitions (Bailey & Wells, 2013). SSA is frequently measured using the Somatosensory Amplification Scale (SSAS; Barsky, Goodson, Lane, & Cleary, 1988). It is identified as occurring in both state and trait forms (Ak, Sayar, & Yontem, 2004; Barsky et al., 1988), and appears idiosyncratically in individuals' symptomatology. Individuals with SSA may experience greater discomfort from sensations, and will be more prone to experiencing these difficulties due to their attentional bias towards these symptoms (Barsky et al., 1988). Chapter 2 reported those who found cravings were unpleasant experienced amplification of these beliefs, describing them as significantly more unpleasant after drinking alcohol. Although this research was conducted to investigate metacognitions, this finding may be indicative of somatosensory amplification.

Anxiety sensitivity (AS) is a tendency to interpret physiological symptoms of arousal as a sign of an impending threat. It has been linked with addiction (S. Stewart & Kushner, 2001) and negative affect associated with substance use (R. A. Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001). Individuals with high AS are said to become hypervigilant to their

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internal state, searching for interoceptive cues of “problematic” or “dangerous” symptoms as a sign that something is seriously wrong (Reiss, Peterson, Gursky, & McNally, 1986).

Individuals with high AS typically perform well on interoceptive accuracy tasks such as a heart rate detection task (e.g., S. Stewart, Buffett-Jerrott, & Kokaram, 2001; Sturges & Goetsch, 1996; Sturges, Goetsch, Ridley, & Whittal, 1998) and are considered sensitive to their internal state. It has been argued that those with greater AS will be less tolerant of symptoms of withdrawal and the associated craving (S. Stewart & Kushner, 2001).

Additionally, Kushner, Thuras, Abrams, Brekke, and Stritar (2001) found that individuals high on AS reported more and stronger withdrawal symptoms. AS may further exacerbate craving due a more immediate biofeedback loop: Individuals who are more sensitive to autonomic changes such as salivation levels and heart rate who also interpret these as signs of craving may experience craving increases. Samoluk and Stewart (1998) found positive relationships between temptation in drinking situations, where individuals perceive exteroceptive cues that may trigger conditioned autonomic responses, and the Anxiety Sensitivity Index (ASI; the most common measure of anxiety sensitivity; Reiss et al., 1986). The ASI was also positively correlated with other drinking variables such as frequency, giving additional credence to the potential relationship between AS and craving.

An alternative prediction is that cravings may decrease if individuals attend to other stimuli. EI theory (Kavanagh et al., 2005) posits that individuals experience an intrusive albeit pleasant thought of a desired substance or experience, which quickly becomes unpleasant with elaboration. This elaboration involves the activation of associated memories, cognitions and emotive states, highlighting the absence of the desired substance or activity. The act of expanding or elaborating upon this intrusion is cognitively taxing. Individuals attempting to perform other tasks while craving experience a reduction in cravings and worse performance on the alternate task (Andrade et al., 2012; Kemps & Tiggemann, 2009; Kemps,

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Tiggemann, & Grigg, 2008). Because cravings consume limited attentional resources, fewer resources are available for interoception. Therefore, conscious diversion of such resources to interoceptive tasks may result in craving decrease.

The following experiment tests the relationship between general interoceptive ability and craving. Competing predictions were drawn from models of internal sensitivity (anxiety sensitivity and somatosensory amplification), which describe enhanced sensitivity (e.g., Barsky et al., 1988; Kushner et al., 2001); and elaborated intrusion theory, which argues that elaborations of craving compete for cognitive resources (Kavanagh et al., 2005).

Predictions based on anxiety sensitivity and SSA theories would be that when an individual is in a heightened state of arousal, they become more aware of their internal sensations. Therefore, they would be more accurate in detecting internal sensations such as in a heart rate detection task (HRDT) when cravings have been invoked. Furthermore, participants with greater interoceptive accuracy may experience increased cravings due to amplification, whereby the individual labels sensations as part of the craving experience. Increased heart rate may lead to increased subjective craving, which may in turn lead to a further heart rate increase. Conversely, EI theory argues that cravings constitute competition for cognitive resources; accordingly, individuals who were craving would perform worse on the HRDT than their non-craving and low-craving counterparts. High accuracy in the HRDT would use considerable cognitive resources, hence should interfere with later craving due to cognitive depletion. Therefore, a number of hypotheses were proposed for the present study.

Participants with higher craving following alcohol cues would experience a change in HRDT accuracy compared to baseline accuracy. We hypothesised that an increase in accuracy may indicate support of AS/SSA models. A decrease in accuracy may indicate



support of EI Theory. We further expected HRDT accuracy to influence later craving. If participants' cravings increased from baseline, this would be consistent with AS models; participants would be sensitive to internal sensations, which would increase their cravings. Conversely, if their cravings decreased, this may be evidence for elaborated intrusion theory, as the HRDT would have interfered with their elaborations.

### **Method**

#### **Participants**

Forty-four participants were recruited via advertising at educational institutions, youth hostels, bars and on websites where participants were invited to contact the experimenters. Fifty participants were sought following recommendations by Gibbons et al. (1993); however, this number was not achieved due to resource limitations. Over 200 potential participants were screened for comorbid disorders via a telephone interview. More information about this method can be found in Chapter 4. Individuals were eligible to participate if they scored 8 or less, or 16 or above on the Alcohol Use Disorder Identification Test (AUDIT; Babor et al., 2001). Fifty participants met this criterion. Only participants with a preference for beer or wine were included as these beverages are the most cost effective. A further three participants were excluded for this reason. Finally, prospective participants were administered the Anxiety Disorder Interview Schedule (ADIS; DiNardo et al., 1994), to determine diagnoses and clarify eligibility. The ADIS is commonly used for assessment of comorbid alcohol use and anxiety disorders (Segal & Williams, 2014). Three individuals were excluded with diagnoses of panic disorder, due to strong relationships between panic disorder and interoceptive ability (cf. McNally, 2002).

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The final sample comprised 44 individuals, including 21 with low-risk drinking (12 females aged 18-55,  $M = 23$ ,  $SD = 10.29$ ; 9 males aged 18-31,  $M = 22$ ,  $SD = 4.83$ ) and 23 with high-risk drinking (11 females aged 18-40,  $M = 21$ ,  $SD = 6.42$ ; 12 males aged 18-31,  $M = 22$ ,  $SD = 4.14$ ). Thirty-five participants consented to provide saliva samples. Participants were each reimbursed \$40 for their participation. Ethical clearance was provided by Macquarie University Ethics committee and the Macquarie University Biosafety Ethics Committee.

### Procedure

Participants were screened by phone, completed trait questionnaire measures online and those eligible attended a face-to-face interview including the ADIS and cardiac history, including medication for heart, vein or blood-related problems that may affect their ability to detect their own heart rate. Participants attended the laboratory described in Chapter 2. Height and weight were measured before participating in a cue reactivity task. These details were used to calculate body mass index ( $BMI$ ;  $\text{weight}[\text{kg}]/\text{height}[\text{m}]^2$ ), as  $BMI$  can affect interoceptive accuracy (Ehlers & Breuer, 1992).

Five minutes of baseline heart rate and galvanic skin response data were recorded. Participants then completed baseline measurements of a heart rate detection task and the AUQ. Next, they watched five minutes of a neutral nature documentary on DVD and completed the water cue reactivity task, the heart rate detection task and the AUQ, then repeated this sequence for the alcohol cues. All participants were exposed to water first. Counterbalancing presentations in similar studies has caused such substantial order effects that water-cued variables cannot be adequately measured if they follow presentation of alcohol cues (McCusker & Brown, 1991; Monti et al., 1987). Following alcohol cues and

measurements, the participants watched ten minutes of the neutral DVD, then completed a final, post-experiment trial of the HRDT. These procedures will now be explained in detail.

### Materials

**Questionnaires.** Following telephone screening, participants were emailed a link to the survey-hosting website Qualtrics, where they completed three questionnaires, in the order in which they appear here. The Anxiety Sensitivity Index (ASI, Reiss et al., 1986) is a 16-item questionnaire with internal consistency of  $\alpha = .88$  (Peterson & Heilbronner, 1987) measuring an individual's beliefs about social and physiological outcomes of anxiety symptoms. It has moderate concurrent validity with the Body Vigilance Scale ( $r = .47$  for Physical Concern items,  $r = .31$  for Mental Incapacitation Concerns items, and  $r = .34$  for Social Concerns items; Zvolensky & Forsyth, 2002). Additionally, it shared a significant relationship with craving-related nicotine withdrawal effects,  $r = .48$  (Zvolensky et al., 2004). ASI items are rated on a 1 (*very little*) to 5 (*very much*) scale.

The Somatosensory Amplification Scale (SSAS; Barsky et al., 1988) is a 10-item scale measuring the degree to which individuals are bothered by uncomfortable somatic and visceral symptoms. It has good internal consistency ( $\alpha = .82$ , Barsky et al., 1990), and has shown moderate concurrent validity with the Whiteley Index, a measure of health anxiety ( $r = .42$ ; Bailey & Wells, 2013). Aronson, Feldman Barrett, and Quigley (2001) have questioned its use as a valid measure of somatic sensitivity due to poor relationships with daily symptom reports ( $r = .17$ ) and internal sensitivity ( $r = -.06$ ). In spite of these poor relationships, the SSAS was included here due to differences in the samples used here (low- and high-risk drinkers) and in the work by Aronson and colleagues (undergraduates with a certain level of physical experiences). SSAS items are rated on a 1 (*not at all*) to 5 (*extremely*) scale.

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The Alcohol Craving Experience questionnaire (ACE; Statham et al., 2011) is a measure of the strength and frequency of the individual's intrusive alcohol-related thoughts based on the thoughts' imagery, intensity and intrusion, over the preceding week. The six subscales have all shown good internal consistency (Strength: imagery  $\alpha = .91$ , intensity  $\alpha = .90$ , intrusion  $\alpha = .74$ ; Frequency: imagery  $\alpha = .93$ , intensity  $\alpha = .94$ , intrusion  $\alpha = .78$ ; Statham et al., 2011). The ACE showed evidence of moderate concurrent validity with the Obsessive Compulsive Drinking Scale ( $r = .34$  to  $r = .58$ ). Additionally, the measure successfully discriminated between a university sample and a clinical sample using AUDIT scores (Statham et al., 2011). ACE items are answered using a scale of 0 (*not at all*) to 10 (*extremely/constantly*).

Before and after cue presentations in the laboratory, participants completed the Alcohol Urge Questionnaire (Bohn et al., 1995), an eight-item measure of craving. The AUQ has excellent internal consistency of  $\alpha = .91$ ; good test-retest reliability of  $r = .82$ , and modest concurrent validity of  $r = .42$  with the Obsessive Compulsive Drinking Scale, and performs well in cue reactivity methodologies (MacKillop, 2006). The AUQ was completed following the baseline, water and alcohol-condition heart rate detection task (below), as a measure of craving. The AUQ response scale is 1 (*strongly disagree*) to 7 (*strongly agree*).

**Physiological assessment.** Participants were fitted with a three lead electrocardiogram (ECG) for heart rate and GSR electrodes to middle phalanges of the non-dominant hand for galvanic skin response (GSR). Recording equipment was routed through an ML116 GSR Amplifier (AD Instruments, 2009); further details of the equipment are outlined in Chapter 4.

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Cue reactivity stimuli. Each participant received two trials: a water-cue reactivity trial and alcohol-cue reactivity trial. In each trial, participants listened to an audio recording (see Chapter 4 for transcript) describing a scene. They then watched the experimenter pour a beverage. In the first trial, this was water; in the second trial, it was either beer or wine at the participant's choice. Beer (4.6% w/v ethanol) was poured from a 250mL bottle (9g alcohol), and 100mL wine (10g alcohol) was pre-measured. Once poured, participants held the beverage and smelled it for three minutes (McCusker & Brown, 1995; Monti et al., 1999) while thinking as strongly as they could about what it would be like to drink it. Water was always presented prior to alcohol, as Monti et al. (1987) found that effects of water reactivity were masked if alcohol is provided first.

In the present study, the weight of participants' saliva was measured during the water and alcohol cues as a physiological indicator of craving (Monti et al., 1987). In preparation for each session, the experimenter placed three 0.8 x 3.8cm dental rolls in a 30mL specimen jar with a screw-top lid. Two of these packages were prepared for each participant and each package was pre-weighed on a set of high-precision (0.001g) jewellery scales. Prior to presentation of the stimuli, participants inserted three dental rolls, one underneath the tongue and two buccally. After the cue reactivity tasks, the participant removed the dental rolls and returned them to the specimen jar. The experimenter re-weighed them and calculated the amount of saliva produced by weight. This process was repeated following alcohol cues. All processes for saliva collection, handling and disposal complied with biosafety regulations. Complete saliva data were available for 35 participants.

Heart Rate Detection Task (HRDT). The HRDT was based on methods by Schandry (1981), and Ehlers and Breuer (1992), and is a valid measure of interoceptive accuracy (cf. Pollatos, Traut-Mattausch, & Schandry, 2009). For the present study, participants were asked

to push the button on a Push Button Switch MLA92 (AD Instruments, 2009) each time they were aware of their heart beat. This switch was routed through the same amplifier as the ECG and all measures were concurrently recorded in Power Lab software (AD Instruments, 2009). Participants completed three randomised periods of 25, 35 and 45 seconds, interspersed with 30-second rest periods, for each of four occasions: baseline, water cue, alcohol cue, and post-experiment. The accuracy of participants' perception was indexed using the formula

$$1 - ([A-P]/A)$$

where A = actual heart rate and P = perceived heart rate. This is a reflection of the formulae used by Schandry, and Ehlers and Breuer, to show accuracy rather than error. In this study, HRDT served as both a measure of interoceptive awareness and means of focusing attention.

### **Data analysis**

In preparation for testing the hypotheses, data were checked for normality and missing data, and were transformed appropriately. During transformation, 1.0 was added to every HRDT score for every participant to account for the number of individuals who provided a score of zero (i.e., they did not detect any heartbeats). Therefore, while the original scale dictated that scores closer to 1.0 indicated greater accuracy, after transformation scores closer to 2.0 indicated greater accuracy. All analyses were conducted using statistical analysis software SPSS v.19 (IBM Corp., 2010). Means and standard deviations for participants' GSR, saliva weight, heart rate and AUQ are shown in Table 5.1. As the experiment tested several measures with a number of outcome variables, Spearman correlations (due to continued non-normality of some data following transformation) were conducted on all variables to confirm there were significant relationships. Table 5.2 shows significant relationships between AUQ and ACE, and between ACE and HRDT, while no significant relationships were observed between AS or SSA and measures of craving or interoception.

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Table 5.1

*Means and Standard Deviations*

	Baseline <i>M (SD)</i>	Water <i>M (SD)</i>	Alcohol <i>M (SD)</i>	Final <i>M (SD)</i>
AUQ	20.07 (11.71)	22.70 (13.05)	29.77 (14.30)	-
Heart Rate	66.57 (9.44)	66.15 (9.51)	66.82 (9.13)	-
GSR	19.73 (8.45)	31.37 (15.89)	32.34 (19.53)	-
Saliva Weight (grams) <sup>a</sup>	-	2.85 (1.41)	3.09 (1.34)	-
HRDT	0.447 (0.334)	0.467 (0.380)	0.455 (0.372)	0.475 (0.375)
ASI	20.70 (11.07)	-	-	-
SSAS	24.75 (6.58)	-	-	-
ACE F1	14.86 (7.47)	-	-	-
ACE F2	18.61 (9.45)	-	-	-
ACE F3	5.39 (3.46)	-	-	-
ACE S1	19.77 (8.37)	-	-	-
ACE S2	24.39 (11.04)	-	-	-
ACE S3	6.23 (4.14)	-	-	-
AUDIT	12.16 (7.39)	-	-	-

*Note.* AUQ = Alcohol Urge Questionnaire; HR = Heart Rate; GSR = Galvanic Skin Response; HRDT = Heart Rate Detection Task accuracy; ASI = Anxiety Sensitivity Index; SSAS = Somatosensory Amplification Scale; ACE = Alcohol Craving Experience questionnaire; F1 = Frequency, Intensity; F2 = Frequency, Imagery; F3 = Frequency, Intrusion; S1 = Strength, Intensity; S2 = Strength, Imagery; S3 = Strength, Intrusion; AUDIT = Alcohol Use Disorders Identification Test.

<sup>a</sup>  $n = 35$

The effectiveness of the cue induction procedure was checked to ensure cravings had been induced. Random effects regressions were conducted to determine relationship between craving and measures of AS, SSAS and EI. We chose random effects regressions as they are superior for accounting for person-specific effects and autocorrelation, compared to ANOVA and general linear model methods (Gibbons et al., 1993). The results of the random effects regressions for participants' self-reported craving (AUQ), heart rate, galvanic skin response and saliva production while they were in the cue induction phases are presented in Table 5.3.

Table 5.2

*Correlations between Measures of Craving, Elaborated Intrusions and Internal Sensitivity*

		AUQ			HR			GSR <sup>a</sup>			Saliva <sup>b</sup>		HRDT			
		Base	Water	Alcohol	Base	Water	Alcohol	Base	Water	Alcohol	Water	Change	Base	Water	Alcohol	Post
AUQ	Water	.62*														
	Alcohol	.82*	.75*													
HR	Base	-.37*	-.27	-.30												
	Water	-.32*	-.25	-.25	.95*											
	Alcohol	-.21	-.35*	-.25	.89*	.88*										
GSR <sup>a</sup>	Base	.16	.24	.24	-.10	-.01	-.16									
	Water	.14	.16	.39*	-.27	-.15	-.30	.46*								
	Alcohol	.06	.00	.16	-.11	-.05	-.10	.39*	.80*							
Saliva <sup>b</sup>	Water	.25	.33	.26	-.14	.00	.01	.09	.10	.13						
	Change	-.12	-.18	-.04	-.08	-.11	-.17	-.02	.20	-.09	-.23					
HRDT <sup>a</sup>	Base	.04	-.21	.07	.01	-.04	.06	-.29	.15	.06	-.07	.28				
	Water	.10	-.26	.06	-.06	-.08	.03	-.26	.15	.04	.02	.39*	.90*			
	Alcohol	.11	-.19	.10	-.01	-.01	.01	-.29	.06	-.08	-.01	.41*	.85*	.93*		
	Post	.10	-.17	.08	-.04	-.05	.02	-.32*	.04	-.05	.09	.36*	.80*	.90*	.93*	
ASI		.05	-.26	-.11	.00	-.02	.06	-.19	-.17	-.21	-.14	.30	.15	.19	.20	.11
SSAS		.23	.08	.07	-.30*	-.32*	-.19	-.11	-.11	-.26	.25	.14	.03	.05	.05	.03
ACE	F1	.66*	.35*	.51*	-.20	-.16	-.19	-.13	.13	-.07	.18	.11	.20	.31*	.36*	.30*
	F2	.63*	.32*	.45*	-.29	-.26	-.22	-.06	.17	-.12	.29	.14	.36*	.46*	.39*	.34*
	F3	.47*	.25	.40*	-.18	-.09	-.18	.10	.39*	.09	.06	.11	.13	.20	.16	.08
	S1	.74*	.45*	.67*	-.19	-.13	-.13	-.02	.20	-.07	.33	.04	.22	.23	.29	.23
	S2	.55*	.32*	.47*	-.31*	-.25	-.20	-.03	.21	-.07	.43*	.17	.40*	.48*	.44*	.40*
	S3	.37*	.15	.37*	-.12	-.03	-.04	-.10	.30	.06	.03	.30	.29	.35*	.39*	.31*
AUDIT		.62*	.39*	.49*	.02	.05	.04	-.09	.03	-.11	.19	-.04	.14	.20	.28	.22



Table 5.2 (cont'd)  
*Correlations between Measures of Craving, Elaborated Intrusions and Internal Sensitivity*

		ASI	SSAS	ACE					
				F1	F2	F3	S1	S2	S3
SSAS		.55*							
ACE	F1	.10	.25						
	F2	.08	.24	.78*					
	F3	.07	.08	.70*	.76*				
	S1	.02	.29	.83*	.67*	.51			
	S2	.00	.28	.65*	.81*	.54	.72*		
	S3	.09	.15	.60*	.61*	.81	.47*	.54*	
AUDIT		-.08	.20	.66*	.48*	.30	.70*	.44*	.30

*Note.* AUQ = Alcohol Urge Questionnaire; HR = Heart Rate; GSR = Galvanic Skin Response; HRDT = Heart Rate Detection Task Accuracy; Post = Post experiment; ASI = Anxiety Sensitivity Index; SSAS = Somatosensory Amplification Scale; ACE = Alcohol Craving Experience questionnaire; F1 = Frequency, Intensity; F2 = Frequency, Imagery; F3 = Frequency, Intrusion; S1 = Strength, Intensity; S2 = Strength, Imagery; S3 = Strength, Intrusion; AUDIT = Alcohol Use Disorders Identification Test.

<sup>a</sup> Transformed using Square Root function

<sup>b</sup>  $n = 35$

\*  $p < .05$

Participants' AUQ and weight of saliva increased significantly between the water and alcohol cues. However, participants experienced no significant change in heart rate or GSR. Thus, the cue induction procedure was successful in increasing AUQ and saliva weight.

Table 5.3

*Results of Random Effects Regression Comparing Baseline, Water and Alcohol Cues Across GSR, Saliva Weight, Heart Rate and AUQ*

Predicted variable	<i>df</i>	<i>t</i>	<i>p</i>	95% CI	
				Lower	Upper
GSR <sup>a</sup>	44	0.59	.558	-0.97	1.78
Saliva <sup>b</sup>	35	2.39	.022	0.04	0.44
Heart Rate	44	-0.48	.637	-1.15	0.71
AUQ	44	3.67	.000	1.36	4.57

*Note.* GSR = Galvanic Skin Response; AUQ = Alcohol Urge Questionnaire; all variables regressed by time.

<sup>a</sup> Square root transformed

<sup>b</sup> *n* = 35

### Results

The aim of this study was to determine firstly whether baseline cravings were related to interoceptive accuracy; and secondly, whether interoceptive accuracy would influence later self-reported cravings. Two steps tested the hypotheses: firstly, the HRDT was tested for practice effects and changes due to physiological appetitive state; and secondly, the effects of the HRDT and other variables on changes in the AUQ were examined. These steps will now be examined in detail.

The first hypothesis was that stronger craving would change HRDT accuracy. HRDT accuracy at baseline, water, alcohol and post-experiment were compared using random effects regressions. First, a linear trend over time and the specific difference between water and

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alcohol cues were entered ( $-2\text{LogLikelihood} = -394.91$ ; Table 5.4). Two dummy variables representing a linear trend over time ('order') and a comparison of alcohol cue induction versus water cue induction ('Alcohol v. Water') were entered, to test for practice effects and specific alcohol-cue effects respectively. No linear trend over time was observed, and alcohol presentation did not change the individuals' accuracy at detecting their own heart rate compared to water presentation.

Table 5.4

### *Cue Effect on Heart Rate Detection Task (HRDT) Accuracy*

Variable	<i>t</i>	<i>p</i>	95% CI	
			Lower	Upper
Step 1 (-2LogLikelihood = -394.91), <i>df</i> = 132)				
Linear trend	0.921	.359	-.002	0.005
Alcohol v. Water	0.683	.496	-.007	0.015
Step 2 Enter Covariates (-2LogLikelihood = -319.16, <i>df</i> = 35)				
Linear trend <sup>a</sup>	1.537	.127	-0.001	0.008
Alcohol v. Water <sup>a</sup>	0.976	.331	-0.007	0.020
AUDIT*	2.246	.031	0.001	0.012
Age	-1.108	.275	-0.010	0.002
Gender	-1.535	.134	-0.160	0.022
BMI <sup>b</sup>	-0.789	.436	-8.253	3.635
Salivation at water <sup>c</sup>	-0.185	.854	-0.036	0.030
Salivation change <sup>c*</sup>	3.160	.003	0.039	0.181

*Note.* Order = Linear relationship across Baseline, Water, Alcohol and Post-experiment conditions; Alcohol v. Water = difference in responses in the alcohol cues compared to the water cues; AUDIT = Alcohol Use Disorders Identification Test; BMI = Body Mass Index (weight(kg)/height(m)<sup>2</sup>).

<sup>a</sup>  $df = 105$

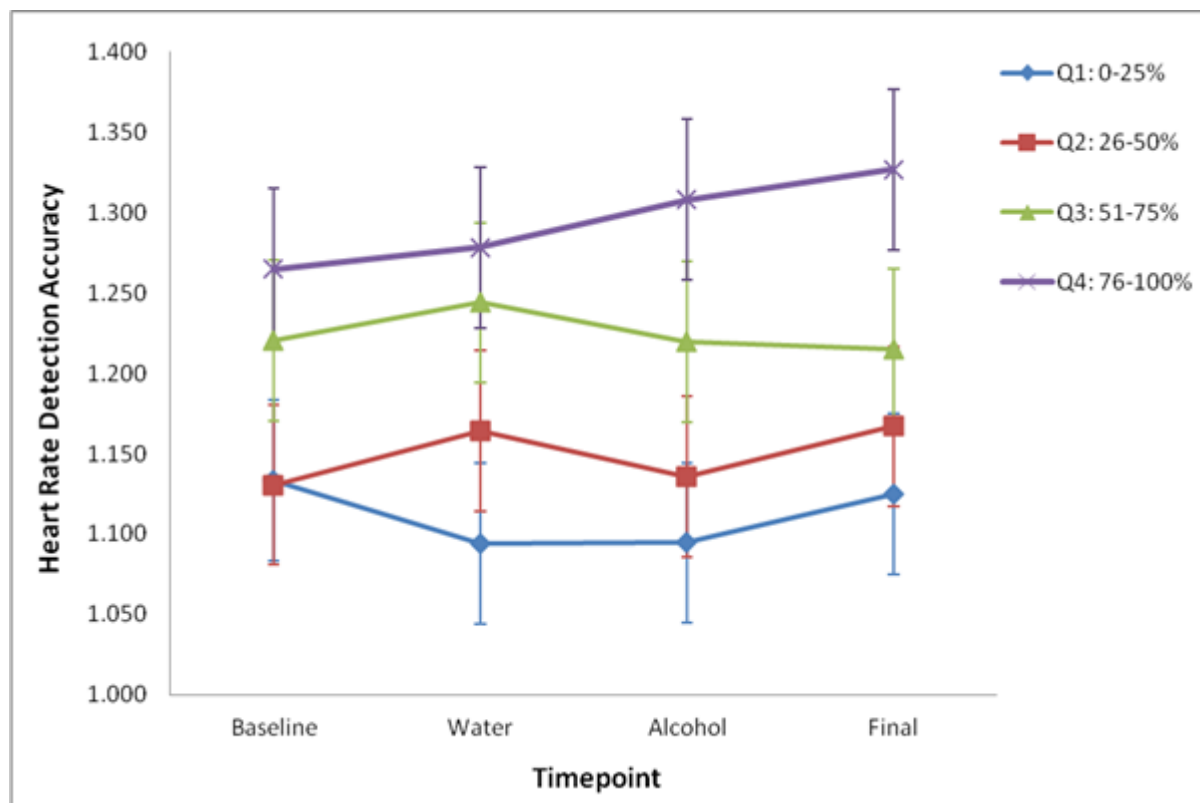
<sup>b</sup> Inversion transformed

<sup>c</sup>  $n = 35$ .

\*  $p < .05$

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In the second step, AUDIT scores, age, gender and BMI were added to control for potentially confounding variables. Weight of saliva produced during water cues and the subsequent change in salivation levels during alcohol cues (hereafter salivation change) were also entered, to determine their relationships with heart rate detection accuracy. Subjective craving, measured by AUQ, was not included in this analysis due to the documented lack of relationship between subjective and physiological measures of craving (Carter & Tiffany, 1999).



*Figure 5.1.* Heart rate detection accuracy as a function of salivation level. In this figure, the quartiles represent salivation change following the alcohol cues (Q1 = least salivation change between water and alcohol cues; Q4 = greatest salivation change), plotted on a scale of heart rate detection accuracy.

Saliva weight produced during exposure to water cues was not related to participants' heart rate detection accuracy. Salivation change was significantly related to HRDT accuracy; those who had experienced a greater increase in their salivation weight were more accurate at

detecting their heart rate,  $t(35) = 3.160, p = .003$ . For ease of interpretation, Figure 5.1 shows the change in HRDT accuracy with the sample divided into quartiles based on salivation change.

The second hypothesis tested the alternative explanations offered by AS/SSA and EI theories. Consistent with AS and SSA theories, we had hypothesised that the HRDT would direct participants' attention internally, and those with higher interoceptive accuracy would crave more. The alternative hypothesis was that in an elaborated intrusion model, participants would experience less craving following high accuracy in the alcohol-cued HRDT, due to interruption of the elaborative process. Initially a linear trend, water and alcohol cues, AUDIT, age, gender, BMI, salivation and salivation change were entered with HRDT accuracy as a time-varying covariate into the first step of a random effects regression. All covariates with exception of linear trends and HRDTs produced small and non-significant effects. Following these, the ASI, SSAS and ACE were entered. Table 5.5 shows the result of this final model. Consistent with the EI hypothesis, greater heart rate detection accuracy resulted in lower AUQ scores,  $t(44) = -3.267, p = .001$ . Additionally, the Frequency of Imagery subscale of the ACE predicted AUQ,  $t(44) = 2.306, p = .026$ , providing further support for EI theory. Moreover, neither ASI nor SSAS significantly predicted AUQ.

Collectively, these results showed that saliva weight was related to increased heart rate detection accuracy. In turn, heart rate detection accuracy was associated with decreased later AUQ scores, independently of initial craving and measures of AS and SSA.

Table 5.5

*Random Effects Regression for AUQ*

Variable	<i>t</i>	<i>p</i>	95% CI	
			Lower	Upper
Linear trend*	5.348	.000	1.490	3.263
Alcohol v. Water	-0.468	.642	-2.190	1.359
AUDIT	0.468	.643	-0.410	0.655
Age	0.432	.668	-0.331	0.510
Gender	0.581	.565	-4.940	8.904
BMI <sup>a</sup>	-1.005	.321	-686.662	230.703
Water salivation <sup>b</sup>	0.544	.589	-1.792	3.108
Salivation $\Delta$ <sup>b</sup>	0.753	.456	-3.278	7.167
HRDT <sup>c</sup> *	-3.267	.001	-58.426	-14.287
ASI	-0.789	.435	-0.467	0.205
SSAS	0.149	.882	-0.623	0.722
ACE – Frequency				
Intensity <sup>c</sup>	-0.240	.812	-7.492	5.909
Imagery*	2.306	.026	0.126	1.905
Intrusion	-1.654	.105	-4.715	0.461
ACE – Strength				
Intensity	1.597	.119	-0.152	1.277
Imagery*	-0.395	.695	-0.683	0.460
Intrusion	0.678	.502	-1.019	2.043

*Note.* AUDIT = Alcohol Use Disorders Identification Test; BMI = Body Mass Index (weight(kg)/height(m)<sup>2</sup>); HRDT = Heart Rate Detection Task as a time-varying covariate; ASI = Anxiety Sensitivity Index; SSAS = Somatosensory Amplification Scale; ACE = Alcohol Craving Experience questionnaire.

<sup>a</sup> Inverted

<sup>b</sup> *n* = 35

<sup>c</sup> Square root

\* *p* < .05

### Discussion

This study contributes to knowledge of craving processes, highlighting the ways interoceptive ability (measured via HRDT), relates to subjective craving and physiological indicators of craving. We had hypothesised that HRDT accuracy would change following alcohol cue presentation. No significant change in interoceptive accuracy as measured by HRDT followed the presentation of alcohol cues. This failed to support the interoceptive accuracy hypotheses of both AS (increased heart rate detection accuracy due to sensitivity) and EI (decreased heart rate detection accuracy due to interruption).

We had additionally hypothesised that HRDT accuracy would relate to later craving. This hypothesis was supported; completing the HRDT resulted in a reduction in craving, potentially due to the demand on cognitive resources. Additionally, although heart rate detection accuracy did not vary with alcohol cue presentation, salivation change was a significant predictor of heart rate detection accuracy. Participants with greater salivation change were more accurate and hence sensitive to their heart rate.

Greater HRDT accuracy following alcohol cues predicted decreased self-report cravings. Yet, this was independent of the individual's salivation in response to alcohol cues. Essentially, this indicates that while cravings measured by salivation do not interfere with subsequent tasks, additional cognitive load does interfere with cravings measured by self-report. The results produced here add to the body of research that shows inconsistent relationships between salivation as a physiological indicator of craving, and subjective cravings (cf. Carter & Tiffany, 1999). These findings indicate mixed support for the EI hypothesis.

Tentative further evidence for EI theory was found in the relationships between self-reported craving measured by the AUQ and the ACE Frequency of Imagery subscale. The absence of stronger relationships between the AUQ and the remaining ACE scales may have been due to the administration of the ACE prior to laboratory attendance. Administering the ACE prior to testing allowed for an estimation of what was usual for the individual, in which case those who regularly experienced imagery related to craving were more likely to crave in the present study.

The different relationships observed in this study indicate the importance of measuring different aspects of craving. Although our definition of craving was predominantly cognitive, physiological indicators of appetitive readiness for digestion (salivation), and arousal (HR, GSR) were included. Our findings support conceptualisations of multisensory craving processes. They further suggest that factors such as HRDT accuracy may contribute to explanations of relationships between subjective cravings and physiological craving indications such as salivation and GSR. This is particularly important given most previous research on craving processes has relied on self-report alone (e.g., Andrade et al., 2012; Panabokke, May, Eade, Andrade, & Kavanagh, 2005).

Previously, Andrade et al. (2012) argued that visual imagery interferes strongly with elaborations by loading the visuospatial working memory, a finding supported in varying degrees by studies of a range of substances (e.g., Andrade et al., 2012; Kemps & Tiggemann, 2007; Murray, 2008; Panabokke et al., 2005; Versland & Rosenberg, 2007). Comparatively, verbal tasks interfere with cravings by loading verbal working memory. Andrade et al. argued this second finding is due to allocation of cognitive resources to inhibiting automated substance-use schemata, as proposed by Tiffany (1990). In our study, the HRDT was neither



imagery- nor word-based, yet appears to have interrupted craving. Further research is required to determine the relationship between HRDT accuracy and craving.

Anecdotally, participants reported that the dental rolls were mildly unpleasant. The presence of the dental rolls may have distracted from the experience of craving; future research may circumvent this issue by using a different technique such as passive drool, which is less invasive (Poll et al., 2007). Considerations such as these are especially important given recent findings that completing a non-desire-related task using both manual and mental skills (forming shapes with clay without looking) can disrupt chocolate cravings (Andrade et al., 2012). This demonstrates the value in internally directed attention, supporting previous findings: May, Andrade, Batey, Berry, and Kavanagh (2010) assessed both externally- and internally-directed attentional tasks in relation to food craving and found that internally-directed attention (Body Scan) reduced food thoughts, where externally-directed attention (Guided Imagery) did not change them. Regardless of method, directing participants' attention internally in the present study produced a reduction in craving.

The present study contributes to understanding of anxiety sensitivity in craving. We had predicted that for those high on the ASI and SSAS, the HRDT would produce an increase in craving. Here, we found no evidence of this. Firstly, cravings decreased following the HRDT, and secondly, scores on the ASI neither significantly predicted craving nor shared significant relationships with any of the key alcohol-related variables such as the AUDIT. Similar results were obtained between the SSAS and alcohol-related variables; however, this is consistent with existing research (Barsky et al., 1990). Consequently, SSA models were not supported in the present study. Both the ASI and the SSAS had poor relationships with the craving and interoception variables. This finding is consistent with the finding of poor

construct validity reported by Aronson et al. (2001), where SSAS showed no relationship to internal sensitivity measured by HRDT. However, our results are in contrast to S. Stewart and Kushner (2001), who suggested that AS may be a mediator for withdrawal symptoms, whereby individuals high on AS would be least tolerant of such symptoms. The discrepancy may be due to the different populations under consideration; the present study's participants were not checked for withdrawal symptoms. They were unlikely to have been experiencing such symptoms given participants were predominantly social drinkers. Moreover, our findings may be attributable to a number of reasons. HRDT may be inadequate as a measure of AS and SSA; interoceptive sensitivity during craving may be qualitatively different to AS and SSA; or individual experiences of craving may have influenced the outcomes.

Our study contained a number of limitations. Firstly, we assumed that HRDT is the best representation of an individual's cravings to test our SSA/AS hypothesis (that focusing on a physiological indicator of craving would exacerbate the craving). Yet, self-report data to date (e.g., Szegedi et al., 2000) have suggested cravings may physiologically manifest in a highly individual way. Some people know they are craving when their mouth goes dry, or their hands become clammy, or by psychological phenomena such as being unable to concentrate. Consciously directing individuals' attention to their heart rate may not have provided the same internal focus that occurs during craving episodes. Our experiment may have been unintentionally skewed in favour of EI theory, by asking participants to focus on their heart rate instead of their most personally relevant craving symptom. For these individuals, HRDT may have provided a distraction, rather than a focal point for craving exacerbation required for a true test of the influence of SSA and AS.

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Secondly, our study used water as a control substance, yet failed to control for thirst adequately. Individuals who were thirsty upon arrival at the lab may have had increases in autonomic arousal upon presentation of water cues, and, as noted earlier, some regular drinkers may use alcohol to quench their thirst. This latter group may interpret thirst as cravings, potentially confounding our results. Future studies would benefit from controlling for thirst (e.g., by asking participants to consume a glass of water prior to the experiment beginning; Thomas, Bacon, Randall, Brady & See, 2011), or making other arrangements to circumvent this issue (de Wit, Söderpalm, Nikolayev, & Young, 2003).

Thirdly, this study may have experienced data collection location issues. The first of these is that participants completed trait-based questionnaires such as the SSAS and ASI at their leisure; we have no knowledge of whether another person helped them or if they were under the influence of a substance, among other possible confounding influences. The second data collection location issue is that the study may have experienced the same craving ceiling effect described in Chapter 2, whereby participants were exposed to alcohol stimuli upon arrival at the laboratory. All measures from baseline to post-experiment were conducted in the laboratory, providing no true baseline in a neutral environment. Related to this, all questionnaires were administered in the same order, potentially producing contrast effects in individuals' responses.

Finally, although the exclusion of individuals who met DSM-IV criteria for anxiety disorders was considered necessary to avoid recruiting too many participants with high interoceptive abilities, this may have resulted in an unrepresentative range of AS. Future research would benefit from including the ASI in screening measures to recruit individuals

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with a range of AS scores, while controlling for individuals whose AS is manifested in pathology, such as panic disorder.

This study tested predictions drawn from AS, SSAS, and EI theories. This new investigation has demonstrated the relationships between subjective and appetitive state craving, and interoception. Anxiety sensitivity and somatosensory amplification were not sufficient explanations for interoception and craving as the elaborative process in craving was interrupted by directing individuals' attention internally. This has progressed knowledge of how craving is processed.

## **Chapter 6**

### **General Discussion**



## CHAPTER 6: GENERAL DISCUSSION

Cravings are a salient part of alcohol use disorders. They are affect-laden, intrusive thoughts of alcohol, which may be amplified by beliefs about craving and the way that these beliefs are processed. In metacognitive models, individuals perceive cues, which may be exteroceptive or interoceptive. These cues provoke cognitions and at times metacognitions directly. These metacognitions – beliefs and knowledge about cognitions and cognitive processes – are assumed to have arisen as a function of the content, strength or valence of the cognitions themselves. When applied to cravings, previous research has assumed that stronger or more frequent craving will produce more metacognitions that are unhelpful, yet this assumption had not been tested. Very little was known about the relationships between cravings and metacognitions, as few studies have tested for them and only highly specific relationships have been found (Nosen, 2012).

The present studies aimed to investigate the relationships between cravings, metacognitions and cognitive processes in cravings. I aimed to understand variables related to metacognitions, and the relationships between alcohol cravings and metacognitions.

Initially, I sought to determine the effect of cravings on craving metacognitions. In Chapter 2, these relationships were small and may have been due to problems with the measurement of craving metacognitions (MCQ-A; Hoyer et al., 2007; CBQ; Beck et al., 1993). Accordingly, I developed and conducted initial testing of a new measure, the Craving Metacognitions Scale (Chapter 3). This measure addresses a broader range of metacognitions and provides an internally consistent, single-factor measure of the perceived antecedents and consequences of craving.

## CHAPTER 6: GENERAL DISCUSSION

My third study further tested the CMS by investigating the effect of metacognitions on craving (Chapter 4). Limited results produced in Chapters 2 and 4 indicated that focusing on the content of metacognitions and cravings does not produce strong and consistent results. As such, I redirected the focus of this thesis to the processes of craving, investigating the effect of interoception on craving. Each of these studies will now be addressed in detail.

### Overview of Studies

#### **Chapter 2: Treatment Seekers' Cravings Activate Specific Metacognitions in a Placebo-Controlled Alcohol Consumption Task.**

In Chapter 2, I predicted cravings and metacognitions would be related. Participants received a control, placebo or alcohol beverage, enabling a comparison of the effects of alcohol and anticipated effects of alcohol (expectancies). Post-drink metacognitions were regressed by pre-drink metacognitions, beverage, cravings and the interaction between the beverage and cravings. Post-drink cravings were regressed by pre-drink cravings, beverage, metacognitions and the interaction between the beverage and metacognitions. Here, I found that the expectancy that alcohol was consumed predicted Subjective Utility metacognitions. Those who believed they had consumed alcohol were more likely to find their cravings helpful. Such specific interactions indicate the importance of examining craving processes, to capture such nuanced relationships.

The structure of this experiment, whereby each type of measure was included both before and after alcohol administration, enabled observation of changes in both cravings and metacognitions, yet relationships between constructs were not observed. In fact, despite



finding a significant change in cravings before and after administration of all beverages, along with some changes to metacognitions after beverage as outlined above, I did not find any significant relationships between cravings and metacognitions. Based on these findings, I concluded the relationships between cravings and metacognitions were highly complex and worthy of further investigation due to potential measurement limitations.

### **Chapter 3: Development and Initial Evaluation of the Craving Metacognitions Scale (CMS) .**

The study reported in Chapter 3 aimed to develop a more comprehensive measure of craving metacognitions. The lack of relationships between cravings and craving metacognitions found in Chapter 2 may have been due to the way the MCQ-A was developed. The MCQ-A items were deliberately generated to tap the clinically relevant dimensions of uncontrollability, unpleasantness, thought avoidance and thought-action fusion, resulting in three subscales of Thought-Action Fusion, Unpleasantness and Subjective Utility. This meant the MCQ-A was unable to address beliefs about craving antecedents, and only addressed a limited range of beliefs about craving consequences. Other measures of craving metacognitions are similarly limited by also only addressing craving antecedents (Lübeck Craving-Recurrence Risk questionnaire (Veltrup, 1994). The CMS was developed to include both antecedents and consequences of craving. Items included in the measure were developed following a process of reviewing existing craving and drinking measures, establishing a large pool of craving cognitions, extracting craving metacognitions based on the semantic content, and writing new items for the CMS. The development sample comprised two groups: regular drinks and those with severe dependence. This ensured a broad range of the phenomena was sampled. One hundred and fifteen individuals receiving inpatient treatment for alcohol-use

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disorders, and 93 university students who drank regularly participated. Items were selected by balancing internal consistency and relationship with MCQ-A. The final scale contained 13 items addressing a single factor that diverged from the MCQ-A. I successfully met the aim to produce a more comprehensive measure of craving metacognitions. With the possibility that the measurement issues were resolved, I commenced testing the measure in an experimental situation.

### **Chapter 4: Metacognitions Relate to Cravings Following Cue Reactivity.**

With improved measurement, I returned to test the relationship between metacognitions and cravings in Chapter 4. This study improved on the methods used in Chapter 2, by measuring metacognitions more comprehensively via the CMS developed in Chapter 3. In this study, participants smelled rather than consumed alcohol, targeting an earlier phase of the alcohol consumption process. This meant my participants avoided changes in cognition that may result from alcohol consumption. I further improved upon the study reported in Chapter 2 as I provided a consumable, appetitive cue as a neutral stimulus (water); measured psychophysiological responses; and used a larger sample of a broader range of drinkers. Neutral and alcohol cues were presented to participants while their heart rate and galvanic skin response were recorded. Participants completed measures about craving and metacognitions before and after each reactivity task.

The CMS showed a significant relationship with craving, providing evidence for a relationship between cravings and metacognitions, and supporting my assertion that a broader measure of metacognitions was needed, given existing measures of metacognitions were limited and did not show relationships with craving. Craving metacognitions did not differ

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between low- and high-risk drinking, providing further support for the role of metacognitions in addiction research. This finding indicates that metacognitions provide a unique contribution not addressed by measures of drinking riskiness. Through Chapter 4, I found a relationship between cravings and metacognitions, yet the effect sizes were small. Accordingly, in Chapter 5, I examined the ways in which cravings are processed.

### **Chapter 5: Interoceptive Accuracy Task Interferes With Rather Than Amplifies Craving in a Cue Reactivity Task.**

Chapter 5 focused on the process rather than content of cravings and craving metacognitions investigated in Chapters 2, 3 and 4. Two theories with differing predictions for individuals' responses to sensations were tested. Participants completed two cue reactivity tasks; one neutral and one alcohol cue reactivity task, during which I measured their heart rate, galvanic skin response (GSR) and salivation. Following each cue reactivity trial, participants completed measures of heart rate detection accuracy, along with measures of craving, intrusive thoughts and anxiety sensitivity. Elaborated Intrusion theory predicted that individuals who were craving would become more distracted and less able to focus on their heart rate, and additionally would experience a decrease in craving as the HRDT prevented them from elaborating on their craving. Anxiety sensitivity models predicted that individuals with high anxiety sensitivity and somatosensory amplification would crave more because they are attending to physiological symptoms and becoming more aware of their internal environment. Results provided partial support for the EI hypothesis; early craving did not influence HRDT accuracy, but HRDT accuracy was related to lower craving later in the experiment. Anxiety sensitivity and somatosensory amplification were not related to craving. These findings support existing findings that cravings are a resource-intensive cognitive

process; craving reduction can be achieved by interrupting that process, rather than focusing on craving content.

### **Thesis Aims and Discussion**

This thesis aimed to investigate variables related to craving metacognitions. This aim was met throughout the thesis. Metacognitions about craving unpleasantness increase over time in a laboratory setting regardless of beverage type consumed, and metacognitions of subjective utility are related to expectation of alcohol effects (Chapter 2). I speculated that craving metacognitions may be due to other factors in drinking experiences, such as engaging in risky drinking; yet in Chapter 4, I found no differences in metacognitions held by low- versus high-risk drinkers.

I further aimed to explore metacognitions and cognitive processes as they relate to craving. Limited relationships observed in Chapter 2 prompted the development of the CMS in Chapter 3, to complement metacognitions addressed by the MCQ-A. The CMS demonstrated in Chapter 4 that it provides a valuable measure of craving metacognitions, as it was significantly related to craving induced by alcohol cue presentation, when no relationships were observed between cravings and other metacognition measures. Cognitive processes and cravings were explored in Chapter 5, where I observed few and small relationships between self-report craving, physiological indicators of craving and metacognitions. After examining the role of interoceptive ability, I found those with greater salivation showed higher heart rate detection accuracy, which in turn led to lower later craving.

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Collectively, the results of these studies suggest that craving metacognitions are trait-like in nature, as they are not easily influenced by the presentation of alcohol cues or changes in craving. The hypotheses developed for the present studies were substantially bolder than the findings I achieved. Nevertheless, the specific relationships I obtained highlight the importance of examining the role of process in craving and metacognition research, where considerable research to date has focused on content.

My work has contributed to the recent resurgence in craving research, brought about by the conceptualisation of cravings as cognitive experiences. Modern craving theories, in which craving is a cognitive feature of addiction and substance use disorders, have extended on earlier and almost discarded theories that supposed craving was merely epiphenomenal. As stated at the beginning of this thesis, if cravings are cognitive, then evaluations of cravings are by extension metacognitive. My research here has contributed to a growing body of evidence that metacognitions and cognitive processes of cravings are relevant to craving research. Small relationships between cravings and metacognitions shown here suggest that they are separate constructs; however, determining the boundaries between these phenomena is a complex issue.

Conceptualisation of craving as thoughts about which an individual holds metacognitions requires careful consideration of boundaries. The relationships between craving itself, the metacognitive response to craving, and even the schemata that may underpin metacognitions, are complex and intertwined. Craving processes are fluid; cues that activate craving may simultaneously activate metacognitions and schemata of substance use. This thesis has contributed to this understanding by reporting the first experimental research

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combining cue reactivity and craving metacognitions. Phases of craving may arise from and provoke these higher order functions, requiring close consideration of the boundaries of each construct. Poorly defined boundaries limit measurement of the relationships between these constructs and other parts of the addiction experience. Well-defined boundaries require comprehensive measurement of each construct.

In observing boundaries between craving and metacognition, I have aimed for comprehensive coverage of craving metacognitions as a construct. My studies suggest four components of problematic craving metacognitions, addressed by the three subscales of the MCQ-A, and the CMS (Chapter 3). These components have shown varied relationships with craving. Relationships observed between these components and craving were neither as strong nor as broad as I had anticipated. Yet, under certain conditions outlined previously, these components changed over time and showed significant relationships with beliefs about alcohol effects (Chapter 2), and produced small but significant relationships with craving (Chapter 4).

Accordingly, I moved to address craving processes by examining the role of interoceptive ability and cravings. This required comprehensive measurement of craving and physiological indicators of craving, along with consideration of non-craving appetitive states. Chapter 5 provided measurement of subjective craving along with physiological indicators of craving (heart rate, GSR and salivation), in conjunction with presentation of an appetitive but non-alcohol related cue: water. Such comprehensive measurement allowed me to observe changes in craving levels arising from cognitive load.

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The outcomes of the present studies may contribute to the development of a metacognitive model of craving. The key findings produced here of small and specific relationships between cravings and metacognitions is counterintuitive to assumptions based on general metacognitive theory (e.g., Wells & Matthews, 1996), which suggests that stronger relationships might have been found. This divergence from predictions by general metacognitive theory may be due to measurement issues, or may indicate the importance of a metacognitive model of craving. To an extent this is unsurprising given specific metacognitive models have been developed for other areas including problem drinking (Spada et al., 2012; Spada & Wells, 2009), anxiety (Cartwright-Hatton & Wells, 1997; Wells, 1995), and depression (Wells et al., 2009; Wells & Matthews, 1996). A metacognitive model of craving would likely differ to Spada and colleagues' model of problem drinking due to the focus on the craving experience, rather than the broader behaviours associated with actual consumption.

The contributions that have been made here have progressed knowledge of the ways in which individuals respond to their cravings for alcohol, particularly by measuring the relationship between cravings and craving metacognitions under controlled experimental conditions. However, some of my work indicates potential links with EI theory (Kavanagh et al., 2005). At times, metacognitions bear similarities to these elaborations of intrusive thoughts. For example, in EI theory an individual who experiences desire for alcohol may then elaborate on that desire with evaluation of whether the desire is good or bad. If desire is considered to have cognitive components, this evaluation is metacognitive. Moreover, my research (Chapter 5) showed disruptions in craving based on interoception, demonstrating that cognitive tasks that interrupt craving can be internally focused. These findings are consistent with EI predictions that simultaneous processes are often completed less successfully or may

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take longer to complete, or both. Conversely, tasks that interfere with the elaborative process can reduce the extent of the elaborations (Kavanagh et al., 2005). This has previously been shown with other attentional tasks, especially tasks involving imagery (e.g., Panabokke et al., 2005); however, this is the first known time that EI theory has been tested using interoceptive ability. These findings demonstrate the importance of attending to processes of cravings and related cognitions, and suggest further lines of enquiry for the relationships between craving metacognitions and elaborated intrusions using cue reactivity methods.

Since this research was conducted, issues have been identified for the clinical relevance of laboratory-based, cue-induced cravings (Sayette & Tiffany, 2013). An alternative methodology, peak provoked craving, is proposed to provide a more naturalistic craving variable. Peak provoked craving does not attempt to separate abstinence cravings from cue-induced cravings by controlling for baseline craving levels, because this does not occur in natural environments. Such separation may be the reason for poor relationships between cue exposure techniques and relapse. For this reason, stronger relationships may be found between peak provoked craving and metacognitions.

This thesis has contributed to metacognitive models of craving. It has tested relationships between cravings, craving metacognitions, and cognitive processes related to craving using a range of methods and measures. In utilising experimental designs including collecting data in a simulated laboratory, using alcohol administration tasks including placebo and control groups, and using cue reactivity tasks, the research presented here is among the most naturalistic as is ethically possible. This is an important feature of craving research as studies have shown that research participants are aware of differences in cravings induced in a



laboratory setting (Wilson, Sayette, Delgado, & Fiez, 2005). Nevertheless, these studies contained a number of limitations.

### **Limitations and Future Directions**

#### **Conceptual issues**

This research is the most comprehensive testing of craving metacognitions to date. Many measures of cravings and drinking-related metacognitions were included (CBQ, MCQ-A, CMS, PAMS and NAMS; the Lübeck Craving-Recurrence Risk questionnaire, Veltrup, 1994; Appraisals of Cravings Questionnaire and Catastrophic Appraisals Index; Nosen & Woody, 2009; were not included). Yet, the studies reported here focused heavily on negative craving metacognitions. This was in part due to a paucity of measures addressing positive craving metacognitions. I know of no positively valenced craving metacognition measures, which may include cravings that build anticipation or excitement for a drink, or make drinks more satisfying or enjoyable. There is little to no information available regarding the breadth of metacognitions as a construct, and whether positive alcohol craving metacognitions are relevant or interesting to addiction research. No concerted effort was made to ensure positive craving metacognitions were included in the CMS during development. Therefore, I am unable to comment on the possibility of relationships between cravings and positive craving metacognitions.

The Subjective Utility subscale of the MCQ-A addresses cravings as warning signs indicating that they are adaptive, but not necessarily positively valenced. No information is available on how the Subjective Utility items were developed. Hoyer et al. (2007) stated that ‘warning signs’ described in the Subjective Utility subscale were warnings for the drinker to remain abstinent. This information was not conveyed to participants in the present studies; it

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is not clear if this definition was relayed to participants in Hoyer and colleagues' study. Moreover, 'warning signs' may be an ambiguous term, open to participants' interpretation of cravings as a warning that drinking has become too heavy or frequent over a period of days, weeks or months; or a warning that the individual should stop drinking within that session. Participants may even interpret the items to mean the opposite of Hoyer and colleagues' intent. Craving may be interpreted as a sign that the person should drink more to avoid experiencing withdrawal.

Including positive craving metacognitions may be particularly important given my study demonstrated that perceived alcohol consumption resulted in viewing cravings as more helpful. This suggests that craving metacognitions may play a role in maintenance of drinking episodes. Individuals with alcohol use disorder may consume amounts well beyond safe levels, even when accounting for severe physiological dependence where rapid total abstinence can cause death. The role of craving metacognitions in such heavy-drinking episodes is unknown, particularly given recent research has reported that drinkers are often unaware when their drinking goals have been met (Spada & Wells, 2006).

Positive metacognitions may be particularly pertinent for samples such as ours, who were predominantly social drinkers (Chapters 3, 4 and 5). For these participants, craving may not involve the same sense of deficit observed in older dependent drinkers, and may hold more positive valence. Although there are theoretical models outlining the role of positive beliefs about drinking in the areas of drinking metacognitions, elaborated intrusion and expectancies, positive craving metacognitions remain an area for future investigation.

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### **Measurement issues**

Early findings in this thesis may have been due to insufficient measures. Moderate to strong relationships between the JACQ-now as a measure of craving and the CMS as a measure of metacognitions were achieved under non-experimental conditions in Chapter 3. Additionally, significant relationships were found under experimental conditions with the CMS measure (Chapter 4). In contrast, in Chapter 2, only smaller relationships to the point of non-significance were achieved, when the AUQ was used under experimental conditions and variance from other measures was accounted for. This suggests the earlier findings may have been due to insufficient measures, and further raises questions of the susceptibility of metacognitions to experimental manipulation.

My studies showed further measurement issues arising from study procedures. Participants in three of the studies (Chapters 2, 4 and 5) completed some of their measures prior to attending the bar laboratory. This is problematic as there is no way of knowing the circumstances under which they completed the measures and confounding variables that may have contributed to the outcomes. For example, someone may have assisted or completed the measures on their behalf; they may have been under the influence of a substance while completing it, or may have been subjected to unknown distractions.

In these same three studies, participants were exposed to the bar environment early. Baseline measures of cravings and metacognitions, among other variables, were taken in the laboratory, limiting my ability to determine the effect of the environment on these variables. Baseline exposure to contextual cues may also have resulted in a ceiling effect for metacognitions and cravings, whereby limited changes were experienced once the participant

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was settled. This issue can be prevented by providing baseline questionnaires in a separate environment, or adopting measures and methods of peak provoked craving (Sayette & Tiffany, 2013). Nevertheless, significant changes were observed in craving, indicating that effects were strong enough to withstand this methodological issue.

Additionally, in all studies questionnaires were presented in the same order to all participants. The implications of this are greatest for Chapter 3, whereby individuals' responses to a new measure, the CMS, are otherwise unknown and may be readily influenced by other measures. The influence on findings in Chapters 2, 4 and 5 may also be serious; examining the effects of two variables on one another by measuring both using self-report in the same order each time may produce significant order effects.

Participants in some of my studies may have become fatigued by repeated questionnaires. One way to reduce this work in future research is via use of visual analogue scales, which typically measure only the intensity of craving at that present time. However, my research predominantly focused on craving content and breadth of craving experiences, I felt it more appropriate to use a questionnaire format.

Finally, self-report measures of metacognitions in the present studies relied heavily on questionnaires developed using a common factor model, which may not adequately capture unique and intense metacognitions experienced by individuals. While these measures had adequate psychometric development to show the items formed internally consistent measures, the role of individual, strong, intense metacognitions was not explicitly assessed here.

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Participants who experienced a range of metacognitions mildly would have received higher scores on the MCQ-A and CMS, compared to those who experienced fewer metacognitions with greater intensity. The latter group may have had stronger relationships between their cravings and metacognitions, yet this was unchecked. Studies by Spada and Wells (2006) and Hoyer et al. (2007) asked alcohol treatment patients to identify key drinking metacognitions; this method could be adopted and the intensity of salient metacognitions could be tracked during experimental manipulation.

### **Sampling issues**

Across each of the present studies, sample sizes limited my ability to conduct thorough analyses of the data. For example, a larger sample for the study described in Chapter 3 would allow exploratory factor analyses to be conducted and would improve chances of detecting effects. Yet, many of the effects observed in my studies were small; indicating that detection of genuine effects would have required substantially larger sample sizes.

Additionally, three of four studies reported here employed social drinkers. While some craving and metacognition research is conducted on non-dependent samples (e.g., May et al., 2004; Spada & Wells, 2008), this practice limits researchers' ability to draw generalised conclusions from the data. Social drinkers may have a qualitatively different experience of craving (e.g., Rosenberg & Mazzola, 2007), and may not experience a sense of deficit that many consider essential for craving. This issue is further compounded by the extensive use of young people in my research.

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Recent work by Slade et al. (2013) demonstrated that young people who technically meet criteria for alcohol use disorders based on DSM-5 criteria show a differential interpretation of alcohol use questions to those with established alcohol use disorder. Drinking levels in young individuals can be strongly tied to social pressures or non-compulsion-based reasons for consumption, even when alcohol use disorder criteria appear to have been met (Slade et al., 2013). As such, it may be difficult to generalise my findings to older, dependent or treatment-seeking populations.

This research has contributed to understanding the relationships between cravings and metacognitions. However, a number of questions remain unanswered. Firstly, the research produced here and elsewhere (e.g., Nosen & Woody, 2014) suggests that the relationships between cravings and metacognitions are highly specific and potentially inconsistent. Replication will confirm these relationships' veracity. Additional research is required to determine why some individuals may hold strong metacognitions in spite of relatively mild cravings, when an individual with comparable cravings makes no such evaluations. An individual's general metacognitive style may contribute to their craving metacognitions.

As no measures of general metacognitive ability or style were included in the present research, I am unable to comment directly on the relationship between cravings, craving metacognitions and general metacognitions. Even so, individuals who hold beliefs about their cravings as uncontrollable, unpleasant or personally relevant may also hold these beliefs about other thoughts (Wells, 2009). Processes of general metacognitive style have not been thoroughly investigated in conjunction with cravings and craving metacognitions. Previous attempts to do so (Nosen, 2012) have used the subscales of the Metacognitions Questionnaire

## CHAPTER 6: GENERAL DISCUSSION

(MCQ; Wells & Cartwright-Hatton, 2004). This measure has been described as a measure of general metacognition (Nosen, 2012), yet three of the five subscales focus on the content of cognitions (positive beliefs about worry, uncontrollability/danger of worrying and cognitive confidence). Consequently, the assessed relationships were between cravings and worry, and cravings and memory; rather than thinking style and craving. The distinction here is important; individuals may experience perseverative thinking about craving (process) without being worried about their craving (content). As cognitions with specific content, neither worry nor memory constitutes a metacognitive processing style general enough to be applicable across a range of areas, including craving. Different measures with items addressing the underlying processes – that is, rumination/ perseverative thought patterns and confidence in cognitive ability – would better service these areas. For example, rather than ask about trust or confidence in memory, researchers may need to ask about trust or confidence in ability to process information and ‘think things through’. Feeling unable to remember things is potentially of little consequence to cravings, whereas an ability to follow thoughts through to a logical conclusion is more useful in learning to detach from cravings, and provides an antithesis to thought-action fusion metacognitions. This necessity illustrates a measurement challenge in this area; it is difficult to differentiate content versus process of latent constructs.

The finding that metacognitions are not influenced by cravings or alcohol (Chapter 2) provides a basis for further research into the factors that *do* affect metacognitions. Research into the development of expectancies may inform this. Factors influencing development of expectancies (e.g., cultural influences, modelled behaviours, personal experiences; Walters, 1998) may further influence alcohol craving metacognitions, especially given the effect of alcohol expectancies on craving metacognitions observed in Chapter 2. Future research may

benefit from consideration of aspects such as severe negative experiences of alcohol including those by proxy, such as having a family member hospitalised for alcohol poisoning, or an addiction that has required inpatient treatment; or perhaps consideration of personality types. Speculatively, it may be that those higher on neuroticism make more extreme evaluations about their cravings.

### **Concluding Comments**

The research presented here has demonstrated relationships between cravings and cognitive processes, including metacognitions, via a series of studies examining these constructs. Despite assumptions that cravings and metacognitions would be related, the phenomena had not been tested for a relationship. The existence of relationships between cravings and metacognitions provides support to cognitive models of craving, and highlights the importance of comprehensive measurement of related variables. These findings contribute to the body of knowledge about cravings and addiction, providing unique findings about craving metacognitions and processes.



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## Ethical approval for studies.



Kristen Tulloch &lt;kristen.tulloch@mq.edu.au&gt;

---

**Confirmation of Ethics approval**

**Ethics Secretariat** <ethics.secretariat@mq.edu.au>  
To: Kristen Tulloch <kristen.tulloch@mq.edu.au>

22 April 2013 10:10

Dear Sir/Madam,

This email is to confirm that the following ethics applications cited below received final approval from the Macquarie University Human Research Ethics Committee:

**1) Chief Investigator: Ms Kristen Jessica Tulloch**

**Ref: HE01MAY2009-D06444**

**Date Approved: 30/06/2009**

**Title: "An adaption of the three-pathway model: the investigation of cravings as a multifaceted, transitional construct".**

**1) Chief Investigator: Dr Lexine Stapinski**

**Ref: HE26JUN2009-R00026**

**Date Approved: 18/03/2010**

**Title: "Drinking over shyness: Understanding the vicious cycle of social anxiety and alcohol use problems".**

Following amendments for Ethics application Ref No: HE26JUN2009-R00026 were approved on 25/11/2010

1. The addition of the Iowa Gambling Task to the battery to aid assessment of compulsive impulsivity. The Iowa Gambling Task is a computerised cards game and participants will not be playing for real money.
2. An increase in the time taken to complete the research session. This will now take between 2-2.5 and 5 hours (participants who have consumed alcohol are asked to remain in the laboratory for an additional 2-3 hours while their blood alcohol content decreases).
3. The information and consent form and the debrief information and consent form have been amended to reflect the above changes.

Please do not hesitate to contact me if you have any questions.

Yours sincerely,

Dr Karolyn White  
Director, Research Ethics  
Chair, Macquarie University Human Research Ethics Committee

--

Office of the Deputy Vice Chancellor (Research)

Ethics Secretariat

Research Office  
Level 3, Research HUB, Building C5C  
Macquarie University  
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Ph: +61 2 9850 6848

Fax: +61 2 9850 4465

Email: [ethics.secretariat@mq.edu.au](mailto:ethics.secretariat@mq.edu.au)

**Ethics application reference-5201001453- Final approval**

Kristen Tulloch <kristen.tulloch@mq.edu.au>  
To: Kristen Tulloch <kristen.tulloch@mq.edu.au>

21 April 2013 20:16

----- Forwarded message -----

From: **Ethics Secretariat** <ethics.secretariat@mq.edu.au>  
Date: 20 December 2010 12:41  
Subject: Ethics application reference-5201001453- Final approval  
To: Dr Andrew James Baillie <andrew.baillie@mq.edu.au>  
Cc: Ms Kristen Tulloch <kristen.tulloch@mq.edu.au>, Miss Mirjana Subotic <mirjana.subotic@mq.edu.au>

Dear Dr Baillie

Re: "Alcohol cravings, impulsivity and cue reactivity: How do socially phobic and non-socially phobic individuals differ?"  
(Ethics Ref: 5201001453)

Thank you for your recent correspondence. Your response has addressed the issues raised by the Human Research Ethics Committee and you may now commence your research.

The following personnel are authorised to conduct this research:

Dr Andrew James Baillie- Chief Investigator/Supervisor  
Miss Mirjana Subotic & Ms Kristen Tulloch- Co-Investigators

NB. STUDENTS: IT IS YOUR RESPONSIBILITY TO KEEP A COPY OF THIS APPROVAL EMAIL TO SUBMIT WITH YOUR THESIS.

Please note the following standard requirements of approval:

1. The approval of this project is conditional upon your continuing compliance with the National Statement on Ethical Conduct in Human Research (2007).
2. Approval will be for a period of five (5) years subject to the provision of annual reports. Your first progress report is due on 20 December 2011.

If you complete the work earlier than you had planned you must submit a Final Report as soon as the work is completed. If the project has been discontinued or not commenced for any reason, you are also required to submit a Final Report for the project.

Progress reports and Final Reports are available at the following website:

[http://www.research.mq.edu.au/for/researchers/how\\_to\\_obtain\\_ethics\\_approval/human\\_research\\_ethics/forms](http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/forms)

3. If the project has run for more than five (5) years you cannot renew approval for the project. You will need to complete and submit a Final Report and submit a new application for the project. (The five year limit on renewal of approvals allows the Committee to fully re-review research in an environment where legislation, guidelines and requirements are continually changing, for example, new child protection and privacy laws).

4. All amendments to the project must be reviewed and approved by the Committee before implementation. Please complete and submit a Request for Amendment Form available at the following website:

[http://www.research.mq.edu.au/for/researchers/how\\_to\\_obtain\\_ethics\\_approval/human\\_research\\_ethics/forms](http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/forms)

5. Please notify the Committee immediately in the event of any adverse effects on participants or of any unforeseen events that affect the continued ethical acceptability of the project.

6. At all times you are responsible for the ethical conduct of your research in accordance with the guidelines established by the University. This information is available at the following websites:

<http://www.mq.edu.au/policy/>

[http://www.research.mq.edu.au/for/researchers/how\\_to\\_obtain\\_ethics\\_approval/human\\_research\\_ethics/policy](http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/human_research_ethics/policy)

## APPENDIX A

If you will be applying for or have applied for internal or external funding for the above project it is your responsibility to provide the Macquarie University's Research Grants Management Assistant with a copy of this email as soon as possible. Internal and External funding agencies will not be informed that you have final approval for your project and funds will not be released until the Research Grants Management Assistant has received a copy of this email.

If you need to provide a hard copy letter of Final Approval to an external organisation as evidence that you have Final Approval, please do not hesitate to contact the Ethics Secretariat at the address below.

Please retain a copy of this email as this is your official notification of final ethics approval.

Yours sincerely  
Dr Karolyn White  
Director of Research Ethics  
Chair, Human Research Ethics Committee

--

Ms Kristen Tulloch

Doctor of Philosophy (Psychology) Candidate  
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*I acknowledge the traditional owners of the land I work on – the Dharug people – and pay my respect to Elders past and present*

21/04/2013 8:16 PM



## Final Approval for Biosafety Ref 5201100016 LAB

1 message

**Biosafety@mq.edu.au** <Biosafety@mq.edu.au>

28 June 2011 09:09

To: A/Prof Andrew Baillie <andrew.baillie@mq.edu.au>

Cc: Ms Kristen Jessica Tulloch <kristen.tulloch@mq.edu.au>

Dear Dr Baillie,

Re: Alcohol cravings, impulsivity and cue reactivity: How do socially phobic and non-socially phobic individuals differ?

Thank you for your recent correspondence. The Committee has reviewed your responses and Final Approval of the above application is granted, effective 28 June 2011.

The following personnel are authorised to conduct this research:

Dr Andrew Baillie - Chief Investigator/Supervisor

Ms Kirsten Tulloch - Co-Investigator

Please note the following standard requirements of approval:

1. Approval will be for a period of 3 years subject to the provision of annual reports. If, at the end of this period the project has been completed, abandoned, discontinued or not commenced for any reason, you are required to submit a Final Report. If you complete the work earlier than you had planned you must submit a Final Report as soon as the work is completed. These reports are located at the following address:

[http://www.research.mq.edu.au/for/researchers/how\\_to\\_obtain\\_ethics\\_approval/biosafety\\_research\\_ethics/forms](http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/biosafety_research_ethics/forms)

A Progress/Final Report for this project will be due on: 28 June 2012.

2. Please remember to notify the Committee of any alteration to the project by completing a 'Request for Amendment' form and submitting it to [Biosafety@mq.edu.au](mailto:Biosafety@mq.edu.au) The 'Request for Amendment' form is located at the following address:

[http://www.research.mq.edu.au/for/researchers/how\\_to\\_obtain\\_ethics\\_approval/biosafety\\_research\\_ethics/forms](http://www.research.mq.edu.au/for/researchers/how_to_obtain_ethics_approval/biosafety_research_ethics/forms)

3. If you will be applying for or have applied for internal or external funding for the above project it is your responsibility to provide the Macquarie University's Research Grants Management Assistant with a copy of this email as soon as possible. Internal and External funding agencies will not be informed that you have final approval for your project and funds will not be released until the Research Grants Management Assistant has received a copy of this email.

If you need to provide a hard copy letter of Final Approval to an external organisation as evidence that you have Final Approval, please do not hesitate to contact the Committee Secretary at the address below.

Please retain a copy of this email as this is your formal notification of final Biosafety approval.

Yours Sincerely

Dr Sinan Ali

## Metacognitions Questionnaire for Alcohol Abusers (MCQ-A)

Listed below are a number of beliefs people sometimes have when thoughts of alcohol intrude on them. Click on the circle that best describes how much you would agree with each item when you have thoughts about alcohol, according to the scale on the right.		Strongly disagree	Disagree	Neither agree nor disagree	Agree	Strongly agree
Item #						
Thought-Action Fusion						
1	This thought is stronger than my will.					
4	I cannot stop this thought once I have it in mind.					
8	This thought has too much impact on me.					
9	I can control this thought.					
12	I cannot push away this thought.					
16	This thought increases my desire to drink.					
18	This thought stimulates craving for alcohol.					
19	This thought can really make me drink.					
Unpleasantness						
2	I feel bad when this thought comes up.					
5	This thought makes me lose my good mood.					
6	It is unpleasant to have this thought.					
11	I get annoyed at this thought.					
13	This thought disturbs me.					
17	I wish I could stop thinking this thought.					
20	I do not want to have this thought.					
21	It is annoying that this thought always returns.					
Subjective Utility						
3	This thought can be of help by waking me up.					
7	This thought serves as a warning signal for me.					
10	I can use this thought when I understand it as a warning sign.					
14	This thought can warn me.					
15	I can learn something through this thought.					

### Appendix C

#### CMS item sources

- Alcohol Abstinence Self Efficacy Scale (AASES; DiClemente, Carbonari, Montgomery, & Hughes, 1994)
- Alcohol Expectancies Questionnaire (AEQ; Brown, Christiansen, & Goldman, 1987)
- Alcohol Urge Questionnaire (AUQ; Bohn et al., 1995)
- Amsterdam Motives for Drinking Scale (AMDS; Ooteman et al., 2006a)
- Approach and Avoidance of Alcohol Questionnaire (AAAQ; McEvoy, Stritzke, French, Lang, & Ketterman, 2004)
- Craving Beliefs Questionnaire (CBQ; Beck et al., 1993)
- Drinker's Inventory of Consequences (DrInC; Miller et al., 1995)
- Jellinek Alcohol Craving Questionnaire-now (JACQ-now; Ooteman et al., 2006b)
- Lübeck Craving-Recurrence Risk Questionnaire (LCRR; Veltrup, 1994)
- Newcastle Alcohol Related Problems Scale (NARPS; Rydon, 1991)
- Obsessive Compulsive Drinking Scale (OCDS; Anton et al., 1995)
- Penn Alcohol Craving Scale (PACS; Flannery et al., 1999)
- Preoccupation with Alcohol Scale (PAS; Leonard, Harwood, & Blane, 1988)
- Temptation and Restraint Inventory (TRI; Collins & Lapp, 1992)

Two additional scales were used as they appeared in the publication by Love, James, and Willner (1998) due to difficulty in obtaining the original scales:

- Alcohol Craving Questionnaire (ACQ; Singleton et al., 1994; cited in Love, et al., 1998)
- Desires for Alcohol Questionnaire (DAQ; Clark, James, Petry, Exner, Williams & Norman, 1996; cited in Love et al., 1998)

Appendix D  
CMS Refined Items and Their Sources

Table D1.

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
Antecedents			
1	I believe that when I feel good I'm more likely to get cravings.	<b>When I feel ..., I'm more likely to get cravings</b> Happy Satisfied Relaxed A sense of achievement Excited / celebratory	LCRR LCRR LCRR LCRR AASES
3	How much I crave depends on how busy or active I am.	I craved more when I had nothing to do or I was bored My cravings happen more often / are more severe after physically strenuous activity. I deliberately occupied myself so I would not drink alcohol When I dream about taking a drink [I get cravings]	(Toneatto, 1999b) AEQ AAAQ AASES
4	When I want to change the way I think, I get cravings for alcohol.	<b>When I want to think ..., I'm more likely to get cravings</b> Less about a fight with my partner, family member or friend More productively Less pessimistically About more positive memories and thoughts	AMDS AMDS AMDS, Toneatto (1999b) Toneatto
6	My cravings occur frequently.	How frequently do these thoughts [about alcohol] occur? I craved constantly. I'll always have cravings for alcohol / have cravings for the rest of my life	OCDS PACS CBQ

## APPENDIX D

Table D1 (cont'd).

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
		Either I'm craving alcohol or I'm not; there is nothing in between	CBQ
8	I get cravings because I have no control over my drinking.	I drink more than I plan to	PAS
		When I get beyond a certain point, I don't stop drinking until all the liquor's gone or I pass out.	PAS
		I wanted to drink alcohol so much that if I started drinking I would have found it difficult to stop	AAAQ, ACQ
		When I want to test my willpower over drinking [I get cravings]	AASES
		When I want to try just one drink to see what happens [I get cravings]	AASES
10	There is nothing in my life that affects my cravings.	<i>Included to address metacognitions of those who do not consider their craving subject to influence</i>	
11	How much I crave depends on whether I am drinking or have had a drink recently.	I usually down the first couple of drinks fast to get a quicker effect.	PAS
		I try to get high and stay that way without getting real drunk [sic]	PAS
		Do you ever cut back on your drinking in an attempt to change your drinking habits?	TRI
14	When I want to change the way I feel I get cravings.	When I wanted to feel..., I got cravings	
		Excited/energetic	AMDS, AEQ
		Enthusiastic and elated / on top of the world / 'high'	AMDS, DAQ
		Relaxed / at ease	AMDS, AASES
		More social	AMDS, AEQ



## APPENDIX D

Table D1 (cont'd).

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
14	When I want to change the way I feel I get cravings (cont'd).	Fitter	AMDS
		Sensational	AMDS
		My muscles relax	AMDS, AEQ
		Enhanced sexual pleasure	AMDS, AEQ
		Nice things more intensely / like things were just perfect	AMDS, AUQ, DAQ
		More in control of my emotions	AMDS
		In a better mood / happy	ACQ, AEQ
		More confident	AEQ
		More optimistic	AEQ
		More compassionate	AEQ
		Carefree	AEQ
		Less stressed / less helpless / more in control in problematic situations	AMDS
		Like my stomach was calmer / less nauseous	AMDS
		Free of withdrawal symptoms	AMDS
		Less down/empty	AMDS, Toneatto(1999b), AEQ
		Less irritable	AMDS, ACQ
		Less anxious	AMDS, Toneatto(1999b), AEQ
		Less angry	AMDS, Toneatto (1999b)
		Less restless	AMDS, ACQ
		Less bored / like my life was boring and monotonous / an escape from my daily routine	AMDS, Toneatto (1999b), AEQ
		Less ashamed	AMDS
		Less afraid of failing	AMDS

## APPENDIX D

Table D1 (cont'd).

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
		Less lethargic / sleepy / tired	AMDS
		My heart beats more slowly / A reduction in the pressure in my chest	AMDS
		Calmer / Less trembling or jittery	AMDS, ACQ
		Less fearful of social situations (e.g., public speaking, failing in front of others)	AMDS
		Less sweaty	AMDS
		Less tense	AMDS, ACQ, DAQ
		Fewer negative memories and thoughts	Toneatto (1999b)
		Drinking helps me feel whatever I want to feel	AEQ
17	My cravings are the result of alcohol still being processed by my body.	My body has a strong need for alcohol	JACQ
		The craving is a physical reaction; therefore, I can't do anything about it	CBQ
		I am in agony because of stopping or withdrawing from alcohol use	AASES
		Sometimes I feel a physical need for alcohol	AASES
		I feel less bothered by physical ills after a few drinks	AEQ
		After a drink or two, things like muscle aches and pains do not hurt as much.	AEQ
		Alcohol makes me feel better physically	AEQ
		The craving is my punishment for using alcohol	CBQ
19	When I want to change the way I act I get cravings for alcohol.	<b>When I wanted to be/act...., I got cravings</b>	
		More active	AMDS
		More talkative	AMDS

## APPENDIX D

Table D1 (cont'd).

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
19	When I want to change the way I act I get cravings for alcohol. (cont'd)	More peppy More self-confident in a group More assertive / less submissive More productive	AEQ AMDS AMDS, AEQ AMDS
21	I believe my cravings are the result of people, places or things that remind me of alcohol.	My cravings happened when I wanted to settle in after I'd finished work If I had been at a pub or club I would have wanted a drink I avoided people who were likely to offer me a drink I avoided places in which I might have been tempted to drink alcohol When people I used to drink with encourage me to drink [I would get cravings] When I see others drinking at a bar or at a party [I would get cravings] When I was at a party where alcohol was consumed, I got cravings	LCRR AAAQ AAAQ AAAQ, TRI AASES, LCRR AASES, TRI LCRR, AAAQ
22	I believe that when I feel upset I'm more likely to get cravings.	<b>I believe that when I feel ... I'm more likely to get cravings. (negative)</b>  Troubled by my work  Belittled Lonely Depressed / down Scared Angry	LCRR  LCRR LCRR, TRI LCRR, AASES LCRR LCRR, AASES, AEQ

## APPENDIX D

Table D1 (cont'd).

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
		Indecisive	LCRR
		Like I wanted to switch off or numb myself	LCRR
		Helpless and weak	LCRR
		That I was having financial difficulties	LCRR
		That I was having problems with my loved ones	LCRR
		Like I couldn't sleep	LCRR
		Tense/Anxious	LCRR, TRI, AASES
		When I am concerned about someone	AASES
		When I have a headache	AASES
		Annoyed / When I feel like blowing up because of frustration	LCRR, AASES
		When I sense everything is going wrong for me	AASES
<b>Consequences</b>			
2	My cravings lead to relapses.	When I've craved, I've found it very easy to resist drinking.	PACS
		When I had cravings, I tried not to think about alcohol.	AAAQ, TRI
		I tried to make the cravings go away by distracting myself so I didn't drink.	AAAQ, TRI
		The craving makes me use alcohol	CBQ
		It would be difficult for me not to drink if I saw or smelled alcohol	JACQ
		I would not be able to stop if I tasted a little alcohol	JACQ
		It would be difficult to resist the temptation to drink if I saw or smelled alcohol	JACQ
		I would drink immediately if I was at home or in a bar.	JACQ
		If I had been at a pub or club I would have wanted a drink	AAAQ
		It would be hard for me to turn down a drink	JACQ

## APPENDIX D

Table D1 (cont'd).

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
		Drinking alcohol appeals as something very delicious to me	JACQ
		The cravings make me use alcohol.	CBQ
		Since I'll have the cravings my whole life, I might as well go ahead and use alcohol.	CBQ
		If the craving gets too intense, using alcohol is the only way to cope with the feeling.	CBQ
		When craving alcohol it's ok to use another substance to cope.	CBQ, DrInC
		If I drank a little alcohol right now I would not be able to stop using it	ACQ, AAAQ, JACQ
5	My cravings make me feel different physically.	My heart would beat faster if I saw or smell alcohol	JACQ
		I sweated more than usual if I smelled or saw alcohol	JACQ
		I produced more saliva if I saw or smelled alcohol	JACQ
		My hands and fingers would tremble if I saw or smelled alcohol	JACQ
		I got a dry mouth if I saw or smelled alcohol	JACQ
		My muscle tension increased if I saw or smelled alcohol	JACQ
		Have you been physically sick after craving?	NARPS
		Craving makes me feel flushed	AEQ
		Muscle aches and pains seemed to hurt more when I was craving.	AEQ
		I can't stand the physical symptoms I have while craving alcohol.	CBQ
		I want a drink so bad I can almost taste it	ACQ, AUQ

## APPENDIX D

Table D1 (cont'd).

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
7	Cravings make alcohol seem almost irresistible.	When I've craved, it felt like I would do almost anything for a drink. My desire to drink seemed overpowering / overwhelming I would have been drawn towards alcohol if there was a drink in front of me The craving is stronger than my willpower. I could not stop myself from drinking if I had some alcohol here Do you find that once you start drinking it is difficult for you to stop?	ACQ, DAQ ACQ, JACQ, AAAQ, DAQ JACQ, PACS CBQ ACQ, AUQ, AAAQ, PACS TRI
9	My cravings will make me feel negative and aroused (e.g., angry, anxious, stressed, out of control).	<b>When I had cravings...</b> ...I felt anxious ...It has made me feel angry ...I felt out of control The craving makes me so nervous I can't stand it.	AMDS, OCDS, Toneatto (1999b), DAQ AMDS CBQ CBQ, DAQ
12	I don't cope with social situations as well if I am craving.	Craving made me less tolerant of people I did not enjoy My family or friends have worried or complained about my craving. While craving I have said or done embarrassing things. While craving, I have said harsh or cruel things to someone. Craving interferes with my social functioning	AEQ DrInC, NARPS DrInC DrInC OCDS
13	Cravings change the way I act for the worse.	When I craved I became more impulsive. I have smoked tobacco more when I am craving.	AEQ, DrInC DrInC

## APPENDIX D

Table D1 (cont'd).

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
13	Cravings change the way I act for the worse.(cont'd)	I have lost interest in activities and hobbies because of my craving.	DrInC
		Because of my craving, I have not had the kind of life that I want.	DrInC
		My spiritual or moral life has been harmed by my craving.	DrInC
		My craving has gotten in the way of my growth as a person.	DrInC
		When I have craved I've neglected myself physically.	NARPS
13	Cravings change the way I act for the worse (cont'd).	Cravings can transform my personality	AEQ
		Craving increases aggressiveness	AEQ
		Cravings increase assertiveness	AEQ
		Cravings can drive you crazy.	CBQ
		Once the cravings start, I have no control over my behaviour.	CBQ
15	My cravings last a long time.	I hardly craved at all.	PACS
		My cravings have lasted for hours.	PACS
		My cravings lasted less than 20 minutes	PACS
		Much of my day was taken up by craving.	OCDS, PACS
		Time passes quickly when I'm craving	AEQ
16	I can't control my cravings.	It has been impossible for me to endure my cravings without distress	OCDS
		At their worst, my cravings seemed totally uncontrollable	OCDS, CBQ
		I have not been able to stop or turn away my thought(s) about alcohol	JACQ
		How much difficulty do you have in controlling your craving?	TRI

## APPENDIX D

Table D1 (cont'd).

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
18	My cravings interfere with my life.	It was very important for me to able to control my cravings	OCDS
		Cravings are something a person can choose to pay attention to	OCDS
		All I want to do right now is drink	ACQ, AUQ
		The images/thoughts I have while craving alcohol are out of my control.	CBQ
		If you have been addicted to alcohol then you have no idea what the craving is like (and you can't expect me to resist).	CBQ
		I'll never be prepared to handle the craving	CBQ
		I don't have any control over the craving	CBQ
		The quality of my work has suffered because of my craving.	DrInC, NARPS
		My ability to be a good parent has been harmed by my craving.	DrInC
		Because of my craving, I have not eaten properly.	DrInC
		I have missed days of work or school because of my craving.	DrInC
		I have found my work less interesting because of my cravings	NARPS
		When I craved, I couldn't concentrate on anything else, including work or home responsibilities	OCDS, TRI, JACQ
		It is hard to distract myself from cravings	TRI
		Do thoughts about drinking intrude into your daily activities?	TRI
		When I was craving, I did not consider myself totally accountable or responsible for my behaviour	AEQ
		When I'm really craving alcohol, I can't function.	CBQ
		I am bothered by recurring images about drinking alcohol	JACQ
		My personal relationships have suffered because of my craving	DrInC



## APPENDIX D

Table D1 (cont'd).

*CMS Refined Items and Their Sources.*

Item #	CMS item	Source item	Source measure/s
18	My cravings interfere with my life.(cont'd)	My reputation has suffered because of my craving	DrInC
		The thought of alcohol keeps me so busy I cannot think of anything else	JACQ
		I am thinking of nothing else but drinking alcohol	JACQ
		My thoughts about alcohol keep me out of my concentration	JACQ
20	My cravings will make me feel down.	I have felt bad about myself because of my cravings	DrInC
		My cravings reminded me of other times when I have craved	Toneatto (1999b)
		When I craved, I remembered bad or unpleasant things much more easily	AEQ
		Cravings made the bad things seem worse	DAQ
		When I craved, daily hassles seemed more important	DAQ
		If I don't stop the cravings, they will get worse.	CBQ
		I have felt guilty or ashamed because of my drinking.	DrInC
		<b>When I had cravings...</b>	
		...they made me feel sad or depressed	NARPS, DrInC
		...I felt less sure of myself	AMDS
		...My emotions became amplified	AMDS
		...I felt hopeless	CBQ

## Appendix E

## 13-item Craving Metacognitions Scale (CMS)

Original item number	Listed below are a number of beliefs people sometimes have about cravings. Please think about the times you have had cravings for alcohol and click for the response that best describes how much you agree with each item according to the scale on the right.	Strongly disagree	Disagree	Neither agree nor disagree	Agree	Strongly agree
2	My cravings lead to relapses.	1	2	3	4	5
4	When I want to change the way I think, I get cravings for alcohol.	1	2	3	4	5
5	My cravings make me feel different physically.	1	2	3	4	5
6	My cravings occur frequently.	1	2	3	4	5
7	Cravings make alcohol seem almost irresistible.	1	2	3	4	5
8	I get cravings because I have no control over my drinking.	1	2	3	4	5
9	My cravings will make me feel negative and aroused (e.g., angry, anxious, stressed, out of control).	1	2	3	4	5
11	How much I crave depends on whether I am drinking or have had a drink recently.	1	2	3	4	5
14	When I want to change the way I feel I get cravings.	1	2	3	4	5
16	I can't control my cravings.	1	2	3	4	5
17	My cravings are the result of alcohol still being processed by my body.	1	2	3	4	5
19	When I want to change the way I act I get cravings for alcohol.	1	2	3	4	5
22	I believe that when I feel upset I'm more likely to get cravings.	1	2	3	4	5