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Cognitive Processes in Alcohol Binges: A Review and Research Agenda

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Abstract

Alcohol abuse is associated with a cluster of long-term changes in cognitive processes, as predicted by contemporary models of addiction. In this paper we review evidence which suggests that similar changes may occur during an alcohol binge, and as such they may play an important role in explaining the loss of control over alcohol consumption that occurs during alcohol binges. As a consequence of both acute alcohol intoxication (alcohol 'priming' effects) and exposure to environmental alcohol-related cues, we suggest that a number of changes in cognitive processes are likely. These include increased subjective craving for alcohol, increased positive and arousing outcome expectancies and implicit associations for alcohol use, increased attentional bias for alcohol-related cues, increased action tendencies to approach alcohol, increased impulsive decision-making, and impaired inhibitory control over drives and behaviour. Potential reciprocal relationships between these different aspects of cognition during an alcohol binge are discussed. Finally, we discuss the relationship between the current model and existing models of cognitive processes in substance abuse, and we speculate on the implications of the model for the reduction binge drinking and its consequences.

Keywords

Alcohol; binge drinking; priming; cues; craving; attentional bias; implicit cognition; impulsivity; inhibitory control; outcome expectancies

1. INTRODUCTION

Heavy episodic, or 'binge' drinking has been defined by US researchers as the consumption of at least 5 (males) or 4 (females) alcoholic drinks within a two hour period [1], and in the UK, as drinking more than half the recommended maximum weekly alcohol intake in a single session [2]. Other researchers have used the term to refer to 'excessive drinking over a single session resulting in self-reported drunkenness' [3], or as alcohol consumption resulting in a blood alcohol concentration in excess of the limits for drink-driving (see [4]). Irrespective of the exact definition, binge drinking has increased in recent years in the UK and other European countries, and in the USA [5], especially in youth [2, 6]. As the negative consequences of binge drinking for health, educational performance, relationships, and

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personal safety have been well documented [3, 7], its increased prevalence is an area of some concern for governments, particularly in Western Europe, the USA, and Australia. As such, the reduction of binge drinking and its adverse consequences has been highlighted as a priority by the governments in the UK [8], USA [9], and other Western nations.

It seems likely that individuals who binge drink often do so after making a conscious decision to get intoxicated. For example, the most frequently cited motivations for drinking among UK University students included pleasure, to increase confidence, the reduction of anxiety and stress, and social facilitation [2]. A qualitative study of underage drinkers revealed similar motives for engaging in binge drinking, particularly social facilitation, social norms and influences, and stress reduction [3]. In one study conducted in the USA, drinking to get drunk and meeting members of the opposite sex were important motivators for binge drinking (see [4]). However, it should be noted that although these self-reported retrospective reasons for binge drinking are of interest, the self-reported reasons for human behaviour may not always reflect the underlying factors which caused that behaviour [10], and binge drinking may be no exception.

The present paper is not concerned with the factors that lead to the *initiation* of an alcohol binge. For a recent and comprehensive review of the cognitive factors that are likely to lead to the initiation of an alcohol binge, see Oei and Morawska [11]. In this paper, we review evidence which suggests that elements of cognitive processing are likely to be influenced *during* the early stages of an alcohol binge, as a consequence of acute alcohol intoxication and the presence of alcohol-related cues. Importantly, these changes in cognitive processing are likely to lead to increased motivation to consume alcohol and a decreased ability to regulate this motivation and drinking behaviour. As such, we argue that these processes make an important contribution to excessive alcohol consumption during an alcohol binge.

In this paper, we briefly review evidence (section 2) which suggests that alcohol abuse and alcoholism are associated with a variety of changes in various cognitive processes (e.g., outcome expectancies and attentional bias for alcohol cues). These changes are consistent with current models of addiction, which generally suggest that although these processes may develop as a consequence of chronic alcohol consumption, they ultimately play a causal role in maintaining alcohol problems. In sections 3 and 4 we demonstrate how these processes are temporarily exacerbated in both alcoholics and social drinkers after exposure to two events which are common to an alcohol binge: the acute effects of alcohol itself, and exposure to environmental alcohol-related cues. In some instances, the absence of relevant research findings prevents us from reaching any firm conclusions, and in these cases we make some novel predictions, which should be tested in future research. In section 5 we outline our argument, namely that those changes in cognitive processes which occur during an alcohol binge as well as the inter-relationships between them, are at least partially responsible for the loss of control over alcohol consumption that occurs during the binge. Finally, in section 6 we speculate on the implications of this review for new interventions which may reduce the level of alcohol consumption during an alcohol binge, and the harms associated with binge drinking.

2. COGNITIVE PROCESSES IN ALCOHOL ABUSERS

Heavy drinking and alcohol abuse are associated with a cluster of changes in cognitive processes. We have structured this evidence into three subsections, which relate to (a) Self-report measures (outcome expectancies, motives and craving), (b) Indirect measures, which are thought to reflect automatic or ‘implicit’ cognitive processing (memory associations, approach tendencies, attentional bias), and (c) Impulsive decision-making and impaired inhibitory control. In this section, we briefly review this evidence and in the final subsection

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(d) we demonstrate how the different processes appear to be inter-related in ways predicted by current models of addiction, substance abuse, and craving.

a. Self-Report Measures

Individual differences in beliefs about the effects of alcohol (alcohol outcome expectancies) are associated with individual differences in alcohol consumption (for reviews see [12, 13]). These beliefs are usually assessed with self-report questionnaires, such as the Alcohol Expectancy Questionnaire (AEQ) [14, 15], in which participants are asked whether they agree or disagree with a number of statements describing the effects of alcohol (e.g., 'Alcohol makes me feel happy'). Later questionnaires also included negative expectancies (e.g., [16]) and dose-related expectancies [17, 18]. In general, heavier drinkers are more likely to expect positive and arousing outcomes, and less likely to expect negative outcomes, compared to lighter drinkers [12]. Note that for negative expectancies a reversal has been reported in alcoholics [13]: negative expectancies appear to decrease first, but after a threshold level of alcohol-related problems has passed, they increase again and these negative expectancies are related to motivation to decrease drinking. Furthermore, the degree to which positive and arousing alcohol outcome expectancies are endorsed is associated with the severity of alcohol-related problems [19] and alcohol abuse [20] and with family history of alcoholism, both in non-dependent adolescents [21] and in alcoholics [22].

Next to these two general types of expectancies (positive and negative), a third general type can be distinguished: negative reinforcement expectancies [12, 23, 24]. The crucial element in negative reinforcement expectancies is the belief that a negative emotional state is alleviated by drinking alcohol (e.g. "after a few drinks I feel less tense" or "after a few drinks I feel less shy"). Negative reinforcement expectancies can be categorized as a subgroup of positive expectancies, but it is important to distinguish them from positive reinforcement expectancies, because there is evidence that negative reinforcement expectancies are particularly important in the development of problem drinking [25-29].

A self-report construct that is very much related to outcome expectancies is 'reasons for drinking'. Unlike questionnaire measures of alcohol outcome expectancies, questionnaire measures of reasons for drinking require respondents to indicate the reasons why *they* drink, or the anticipated outcomes of drinking alcohol which motivate them to consume it [30, 31]. As might be expected based on the outcome expectancy literature, drinking motives can be separated into positive reinforcement motives (e.g. the desire to drink alcohol to elevate positive mood) and negative reinforcement motives (e.g. the desire to drink alcohol to alleviate negative mood). Although strong endorsement of any type of drinking motive is associated with alcohol consumption and alcohol problems, individuals who strongly endorse negative reinforcement motives in particular are more likely to drink heavily and be diagnosed with alcohol problems [30, 31]. One possible reason why negative reinforcement alcohol outcome expectancies and reasons for drinking are particularly closely associated with alcohol abuse problems is that even though alcohol may temporarily alleviate negative affect, chronic heavy alcohol use may result in more negative affect in the long run, which leads to more drinking to alleviate the negative affect, thus creating a vicious cycle [32].

Subjective 'craving' is another example of a self-report measure which has been implicated in alcohol abuse disorders [33]. Although the concept has proved difficult to define [34], here we refer to the subjectively experienced desire or urge to use a drug, which can be dissociated from intentions to use the drug, for example when attempting abstinence [34]. Craving for alcohol is clearly elevated in heavy and problem drinkers. For example, among non-dependent adult drinkers, various measures of alcohol craving are positively correlated with indices of drinking frequency and quantity, or elevated in problem drinkers compared

to light drinking controls [35, 36]. Similar associations (between the quantity / frequency of alcohol consumption, and subjective craving) have also been reported in adolescents [37].

It is important to consider that subjective craving for a variety of substances, including alcohol, [38-41] appears to be a multifactorial construct, and therefore single item measures of craving (e.g., 'Please rate your desire for a drink now') may assess only limited aspects of the experience of craving. For example, a factor analysis revealed four components of craving in the Alcohol Craving Questionnaire (ACQ): 'emotionality', 'purposefulness', 'compulsivity', and 'expectancy' [40, 42]. Likewise, the Desires for Alcohol Questionnaire (DAQ) differentiates between mild desires, strong desires, perceived control over drinking and reinforcement [39].

More recently, McEvoy *et al.* [36] stressed the importance of measuring subjective motivation to drink (i.e., 'craving' as generally conceptualised) in tandem with the subjective motivation to *not* drink. They developed the Approach and Avoidance of Alcohol Questionnaire (AAAQ) and found that high motivations to drink were not necessarily associated with low motivations to not drink. Further, the two types of motivation were found to *independently* predict variance in consumption and alcohol-related problems [36, 43]. McEvoy *et al.*'s concept of avoidance motivation may resemble the role of negative outcome expectancies [13], the strength of which appear to act as a 'brake' on alcohol consumption.

To briefly summarise, a considerable body of evidence indicates that heavy alcohol consumption and alcohol-related problems are associated with elevated subjective craving for alcohol. More recent research indicates the need to consider the multifactorial nature of craving, and within this, other studies emphasise the need to consider the motivation to drink as well as the motivation to not drink; the two appear to be partially independent and they are not reciprocal.

b. Indirect Measures

As recently reviewed by Wiers *et al.* [44], researchers have begun to use various measures to investigate spontaneous and relatively automatic ('implicit'; see [44, 45]) alcohol-related cognitive processes. As such, one way in which to conceptualise this evidence is as a complement to the research on self-report measures of alcohol cognitions, as described in the preceding section. The crucial difference is that indirect measures do not rely on participants' self-reports to make inferences about their cognitive processes. Instead, these measures rely on alternative responses, typically reaction time and spontaneous associations, to make inferences about the underlying cognitive processes [46].

For example, Stacy and colleagues [47-49] employed memory association tasks in which participants were asked to provide their first association to a variety of prime words that were ambiguously related to alcohol use (e.g., 'draft'). Findings indicated that the extent to which alcohol-related words were spontaneously generated in response to these ambiguous primes was a robust predictor of subsequent drinking, even when prior drinking and explicit measures were statistically controlled [48]. Other researchers have employed reaction time measures such as the implicit association test (IAT) to examine individual differences in associations between alcohol and various target concepts (e.g., 'positive' *vs* 'negative', or 'arousal' *vs* 'sedation', [50, 51]). In this task, participants are required to rapidly categorise visually presented words. For example, they are instructed to press the left response key when an alcohol related word or a positive word is presented, and the right response key for non-alcohol or negative words. The rationale for the task is that if participants implicitly associate 'alcohol' with 'positive', they should be quicker to respond when 'alcohol'

and 'positive' words shared the same response key (as in the example), compared to another part of the task where 'alcohol' and 'negative' words shared the same response key.

However, perhaps surprisingly, numerous studies have consistently shown that both heavy and light drinkers have strong implicit alcohol-negative associations, rather than alcohol-positive associations [50-52]. This possibly reflects general social norms concerning alcohol use (see [53] and [44], for discussion). In the unipolar version of the IAT, in which positive and negative associations are assessed separately, it was found that negative associations are stronger but unrelated to alcohol use, while positive associations are weaker but related to alcohol use [54-56]. The latter finding is also supported by studies using other varieties of reaction time paradigms to assess alcohol associations [57, 58].

Using the IAT to examine alcohol-arousal (versus sedation) associations, it was found that only heavy drinkers, but not light drinkers, showed strong associations between alcohol and arousal [51, 54]. Similarly, Palfai and Ostafin [59, 60], showed that the tendency to associate alcohol with approach (rather than avoidance) during the IAT was associated with binge drinking frequency and difficulties in controlling alcohol use in hazardous drinkers. Taken together, these findings suggest that heavy and problem drinkers might implicitly associate alcohol with arousal and behavioural approach. Other investigators have used alternative paradigms and demonstrated comparable findings. Field *et al.* [61] investigated approach (versus avoidance) responses elicited by alcohol-related pictorial cues using a different task, the Stimulus-Response Compatibility (SRC) Task. During the task, participants are required to move a manikin either towards (approach response) or away from (avoidance response) alcohol and matched neutral pictures. The relative speed with which participants make the approach response to alcohol-related pictures (compared to the avoidance response) indicates the extent to which alcohol stimuli are compatible with approach versus avoidance responses (hence the name of the task). Results indicated that rapid approach responses to alcohol pictures were seen only in heavy drinkers, but not in light drinkers. The results from this task then, appear to complement the findings from the studies that used the IAT, as they demonstrate that heavy drinkers tend to automatically associate alcohol with behavioural approach. Wiers *et al.* (under review) used a related task to assess automatic approach tendencies for alcohol, the Approach Avoidance Test (AAT, [62]). In the variety they used, participants reacted to an irrelevant feature of the stimulus (picture format: portrait or landscape) with a push or pull movement of a joystick. Upon the movement, the picture changed in size on the computer screen, so that the picture became larger upon a pull movement (approach) and smaller upon a push movement (avoidance). They found that heavy but not light drinkers were faster to pull (approach) the alcohol-pictures than to push (avoid) the alcohol pictures. Furthermore, this difference was specific for the alcohol pictures (it was not found for general positive or negative pictures).

Heavy alcohol consumption and alcohol abuse problems are also associated with 'attentional bias' for alcohol-related cues. That is, alcohol-related cues tend to 'capture the attention' in heavy drinkers. For example, studies using the 'alcohol Stroop' task have demonstrated that alcoholics and heavy social drinkers, but not light drinkers, are slow to name the colour in which alcohol-related stimuli are printed (e.g., [63-65]; for a review, see [66]), which suggests that alcohol-related stimuli grab their attention. Other studies have used the visual probe task, which provides a more direct measure of visuospatial attention, to demonstrate that heavy drinkers, but not light drinkers, are faster to respond to probes that appear in the location of alcohol-related pictures than control pictures, which is consistent with their attention being allocated to the spatial location of the alcohol cues [67, 68].

It is important to distinguish between relatively rapid, perhaps automatic biases in the *shifting* of attention, versus biases in the *maintenance* or *disengagement* of attention [69,

70]. Of the various paradigms used to measure attentional bias in alcohol abusers, it is likely that the visual probe task can measure biases in rapid, automatic attentional capture when alcohol-related stimuli are presented for brief durations such as 200 ms or less (see [71]). By contrast, when stimuli are presented for longer durations (e.g., 500 ms or longer) then any observed attentional biases are more likely to reflect biases in the maintenance or disengagement of attention from those stimuli [71]. It is interesting to note that heavy drinkers tend to show attentional biases for alcohol stimuli only when they are presented for relatively long exposure durations (500ms or more; see [67, 68]), but not when they are presented briefly (e.g., 200ms; see [67]). However, in alcohol-dependent inpatients (compared to non-alcoholic controls), attentional biases are seen for briefly-presented (50ms) alcohol-related stimuli, and the magnitude of this effect is related to the severity of alcohol dependence [72]. Perhaps unexpectedly, if the stimuli are presented for 500ms or longer, attentional avoidance of those stimuli is seen (see [72-74]). This might reflect strategic attempts to avoid extensive processing of alcohol-related stimuli in inpatient alcoholics who are attempting to remain abstinent. Taken together, the evidence would appear to suggest that severe alcohol dependence, but not heavy drinking *per se*, is associated with relatively rapid and automatic attentional capture by alcohol-related cues. However, heavy drinking in individuals who are not seeking treatment is associated with biases in the maintenance or delayed disengagement of attention from alcohol-related pictures; among alcoholics who are attempting to remain abstinent, this slower aspect of attentional bias is eliminated or even reversed, perhaps as a consequence of attempts to avoid thinking about alcohol.

c. Impulsive Decision-Making and Impaired Inhibitory Control

The psychological concept of ‘impulsivity’ has proved difficult to define, although it is thought to underlie behaviours that are risky, poorly planned, and result in undesirable consequences [75]. As such, it is implicated in a number of psychiatric disorders, including substance abuse and addiction, and attention-deficit and conduct disorders in children [75]. Impulsive cognition and behaviour have been directly studied using experimental procedures (see [76]). Several authors [77-79] have suggested that two relatively independent processes may be involved: (a) deficits in inhibitory control (or ‘motor impulsivity’, [78]), such that individuals find it difficult to inhibit impulses or reward-driven behaviour, and (b) impulsive decision-making (or ‘cognitive impulsivity’, [78]), such that individuals consistently choose rewards that are immediately available, despite negative consequences of those choices in the future. As we discuss below, both components of impulsivity appear to be associated with long-term alcohol abuse.

Inhibitory control deficits have been measured with ‘Go/No-Go’ and ‘Stop’ tasks, both of which measure the ability to inhibit inappropriate responses. For example, during the Go/No-Go task [80], participants are instructed to make a rapid manual response to certain discriminative (‘Go’) stimuli, but to withhold their response when a No-Go signal stimulus is presented. Commission errors (inappropriate responses to the No-Go stimulus) provide an index of inhibitory control. Very few published studies have investigated differences between alcohol abusers and controls on tasks that measure inhibitory control. However, Noel *et al.* [81] reported some evidence which suggests that alcoholics have compromised inhibitory control compared to non-alcoholics, and Kamarajan *et al.* [82] reported differential patterns of brain activity (event-related potentials) in alcoholics and controls whilst they performed a Go/No-Go task. In another study, among social drinkers, heavy alcohol consumption was associated with more failures of inhibitory control during a Go/No-Go task [83].

The delay discounting procedure has been widely used to measure impulsive decision-making, in both human and animal subjects. In the procedure (see [84]), subjects are

presented with choices between small rewards that are available immediately, versus larger rewards that are available after a delay. A preference for small rewards delivered immediately at the expense of larger rewards delivered after a delay can be understood as 'impulsive' responding (see [84]). Numerous studies [84-87] show that adult heavy drinkers and alcoholics display more pronounced delay discounting than nonor light-drinker controls; furthermore, even adolescent heavy drinkers show more pronounced delay discounting than their light drinker counterparts [37], and the magnitude of delay discounting is negatively correlated with the age of first alcohol use [88], which might indicate a linear positive relationship between the lifetime duration of alcohol use and delay discounting.

d. Theoretical Accounts of these Phenomena and their Inter-Relationships

Neurobiological and cognitive models of addictive behaviours can account for the phenomena described above. Firstly, with regard to alcohol outcome expectancies, cognitive models of substance abuse (e.g., [89]) and specific expectancy models (e.g., [90]) posit that beliefs about the effects of alcohol are important determinants of alcohol consumption; research showing that individual differences in explicit outcome expectancies can prospectively predict alcohol use are consistent with this proposition (e.g., [25]) although it should be noted that prospective prediction is much weaker, especially after controlling for previous alcohol use [13, 19, 91]. Regarding subjective craving, almost all theories of addiction, whether they are neurobiological (e.g., [92, 93]), or cognitive (e.g., [89], but see [94]) in origin, agree that subjective craving is a core component of substance dependence which maintains drug-seeking behaviour and can increase the risk of relapse in users attempting abstinence. Cross-sectional research such as that described in section 2a indicates that alcohol dependence and heavy drinking are associated with elevated craving; however, this type of research does not necessarily suggest that craving is the cause of excessive alcohol consumption, although many prospective studies have suggested that craving does play a causal role see [95]. Importantly, as predicted by Marlatt and Gordon's ([89]) model of the inter-relationships between different cognitive processes in addictive behaviours, alcohol cue-induced increases in alcohol outcome expectancies are accompanied by and associated with increases in subjective alcohol craving (e.g., [96]).

Turning to indirect measures, we note that some theorists [44, 97] have speculated that these measures are more likely to measure the automatic determinants of alcohol consumption than self-report measures, such as alcohol outcome expectancies. Research showing that performance on some of these indirect measures (e.g., the IAT; spontaneous memory associations) is a robust predictor of subsequent alcohol use, even when the role of explicit outcome expectancies is statistically controlled, is consistent with this position (e.g., [48, 51, 53]). With regard to attentional bias and automatic approach tendencies, numerous models [93, 98, 99] predict that such cognitive responses to alcohol cues should be a core feature of alcohol-related problems, and they may maintain the disorder. For example, Robinson and Berridge [93] suggest that repeated administration of any substance of abuse, including alcohol, produces a dopaminergic response that becomes sensitized (i.e., progressively larger) with each new substance administration. This process causes the substance to be perceived as particularly salient and to acquire strong motivational properties, so that obtaining and self-administering the substance becomes an important goal, and strong subjective cravings for the substance develop. Through classical conditioning, a substance-related cue ac-quires incentive-motivational properties, and as a consequence it "grabs attention, becomes attractive and 'wanted,' and thus guides behaviour to the incentive" [93, p. 261]. A recent extension to the theory [98] speculates that attentional bias, once established by this dopaminergic process, might act to maintain alcohol-seeking through its reciprocal relationship with subjective craving. In essence, once attentional bias for alcohol cues is established, this should increase subjective craving (see [100, 101], for supportive

evidence). Once craving increases, this provokes further increases in attentional bias, and so on, until ultimately the substance is sought out and self-administered. However, the association between subjective craving and indirect measures of cognitive processes is not always found [98] and recent findings by Wiers, Rinck *et al.* [102] suggest that approach action tendencies in response to alcohol-related cues can be 'primed', with subsequent effects on drinking behaviour, in the absence of effects on subjective craving. This suggests that appetitive responses to alcohol-related cues (attentional bias, approach action tendencies) and subjective craving may often be related and both appear to play a role in alcohol use and problems, but they are not identical. One possibility is that subjective craving is generated when one becomes subjectively aware of attentional bias or an approach tendency, for example because the execution of the approach tendency is undesirable or impossible (cf. [46, 94]). Note that according to this view, implicit appetitive motivation can also lead to alcohol use without subjective craving.

Finally, the observed deficits in inhibitory control, and increased impulsive decision making, in heavy drinkers can be explained by contemporary models of addiction which emphasize that substance abusers' executive cognitive functioning (ECF) is compromised, which is manifested as elevated impulsivity and poor inhibitory control over drives and behaviour [44, 78, 103-107]. For example, Jentsch and Taylor [105] and Goldstein and Volkow [104] argue that chronic substance use leads to damage to regions of the frontal cortex which are involved in the regulation or inhibition of behaviour. As a consequence, alcohol problems might develop because the motivation to consume alcohol increases, yet the ability to regulate or inhibit this motivation decreases.

Many of these models that depict a relationship between impaired ECF and heavy alcohol use also postulate that substance users with poor inhibitory control will have difficulty inhibiting their responses to substance-related stimuli with strong incentive-motivational properties. For example, Dawe *et al.* [103, p. 1397] discussed a "possible synergistic effect between the heightened … incentive salience of conditioned substance stimuli … and an inability to consciously inhibit impulsive behaviour, possibly due to prefrontal dysfunction". Thus, highly impulsive substance users and those with poor inhibitory control might be more sensitive to the attention-grabbing properties of substance-related stimuli than others. Results from some recent studies are consistent with this argument: in adolescents, heavy alcohol consumption, impulsive decision-making (as assessed with a delay discounting task), and attentional bias (as assessed with a modified Stroop task) are all inter-correlated [37]. In addition, two other recent studies found that poor executive function (as assessed with a working memory task) in adolescents moderated the associations between implicit associations and drinking behaviour [108, 109]. In both studies, the relationship between the appetitive alcohol associations (assessed with open ended memory associations in [108]; with varieties of the IAT in [109]) with drinking behaviour was stronger in adolescents with relatively poor scores on an unrelated working memory task.

In summary, the observed cognitive changes that characterise heavy drinkers are consistent with contemporary models of addiction. Recent models (e.g., [44, 104]) can also explain the inter-relationships between these cognitive changes, and as such they present a fairly comprehensive view of the cognitive processes that are involved in alcohol problems. In the next sections, we describe evidence which demonstrates how these cognitive processes are affected by administration of alcohol 'priming doses' in the laboratory (section 3), and how they are affected by exposure to alcohol-related cues (section 4).

3. EFFECTS OF BINGE DRINKING 1: ALCOHOL 'PRIMING' DOSES

a. Self-Report Measures

Given the large volume of research devoted to exploring the relationships between alcohol outcome expectancies and alcohol consumption and abuse [19] it is surprising that, to our knowledge, no published studies have looked at the effects of alcohol priming doses on these measures. Furthermore, one explanation for the effects of alcohol priming doses on subjective craving (see [110], and discussion below) is that alcohol priming doses may stimulate recall of (pleasant) past experiences when intoxicated, and these memories provoke the desire to consume more alcohol. The prediction that follows is that alcohol priming doses should increase the endorsement of expectancies about the positive outcomes of alcohol consumption (e.g., 'alcohol makes me more friendly'), while also perhaps decreasing the endorsement of expectancies relating to negative outcomes of alcohol consumption (e.g., 'alcohol makes me feel sick'). Similarly, given that alcohol priming doses can enhance positive mood and promote anxiolysis and feelings of relaxation [111], one might expect that as individuals begin to experience these effects during the early stages of an alcohol binge, their self-reported outcome expectancies and reasons for drinking would be expected to change – i.e. people may be more motivated to continue drinking in order to prolong and exacerbate the subjective effects which they are experiencing.

We are not aware of any single study which has investigated this research question by administering alcohol versus placebo drinks to participants before they complete questionnaire measures of alcohol outcome expectancies or drinking motives. As we describe in section 4, below, questionnaire measures of alcohol outcome expectancies can be modified so that they are sensitive to 'state' variables such as environmental context. We suggest that these questionnaire measures should be sensitive to alcohol priming effects; specifically, we predict that alcohol should increase the strength of positive outcome expectancies while decreasing the strength of negative outcome expectancies. Future research should test these predictions.

By contrast, the effects of acute alcohol intoxication on subjective craving have been extensively researched. Following early demonstrations that administration of 'priming' doses of alcohol could increase behavioural indices of drinking motivation (e.g., latency to drink) in alcoholics [112-114], several research groups have investigated if priming doses can increase subjective craving for alcohol. A considerable body of evidence now indicates that priming doses of alcohol can increase subjective craving in social drinkers and alcohol-dependent patients. For example, de Wit and Chutuape [115] demonstrated that alcohol preloads (0.25-0.5g/kg) dose-dependently increased subjective alcohol craving and choice for alcohol (in preference to monetary rewards). Numerous subsequent studies have demonstrated that, in social drinkers, alcohol preloads can increase subjective craving, and in those studies that administered multiple different doses, these effects were found to be dose-dependent [116-120]. However, it is important to note that some studies have not replicated these effects [121, 122].

In summary, the available evidence suggests that administration of an alcohol priming dose produces a robust increase in subjective craving among social drinkers. These effects appear to be dose-dependent, with large and robust increases in craving seen after administration of high doses of alcohol. However, many questions remain unanswered, and these issues pose important questions for further research. For example, if alcohol craving is a multifactorial construct, it is not clear which subcomponents of craving are most sensitive to alcohol priming doses. Given recent evidence which suggests that alcohol craving is not a unidirectional construct, with inclinations to drink being independent from inclinations to *not* drink [36] (see section 2a), a target for future research is to examine how these two

unrelated components of craving are affected by alcohol priming doses. For example, alcohol priming doses might increase the motivation to drink whilst leaving the motivation to not drink unaffected, or they may also inhibit or weaken the motivation to refrain from drinking.

More broadly, there is still much debate over the exact mechanisms which mediate the effects of alcohol primes on alcohol craving. As reviewed by de Wit [110], there are several possibilities, which are not mutually exclusive. Firstly, administration of a rewarding stimulus (such as alcohol) may prime the motivation for that stimulus through an unconditioned (unlearned) mechanism (e.g., [123]). Alternatively, priming effects may be learned. For example, given that alcohol craving may be, at least in part, a learned response [124, 125], it is possible that administration of alcohol may reinstate classically conditioned responses, including craving, and operant responses, including alcohol-seeking. A related account suggests that the interoceptive effects of alcohol may prime memory structures related to alcohol, such that when intoxicated, alcohol users may recall their previous positive experiences with alcohol and this may increase their motivation to drink. This account leads to the fairly straightforward prediction that alcohol priming doses should increase positive outcome expectancies, and perhaps also increase the accessibility of some alcohol associations; as we discussed earlier in this section, this prediction has not yet been investigated. Finally, it is possible that alcohol has disinhibitory effects which might impair the ability to inhibit intrusive subjective cravings. This will also be discussed in the following sections. It is likely that each of these possibilities can partially account for the effects of alcohol priming doses on alcohol craving, and for the psychological processes that occur during alcohol binges in general.

b. Indirect Measures

To our knowledge, very few published studies have examined the effects of an alcohol priming dose on indirect measures of alcohol-related cognitive processes. Palfai and Ostafin [60] had heavy drinkers complete an evaluative priming task which required them to rapidly respond to positive and negative alcohol-related outcome expectancy words which were presented after ‘prime’ words that were either alcohol-related or neutral. Half of the participants received an alcohol priming drink before completing the task, the remaining participants received a placebo. Results indicated that those participants who had consumed alcohol were faster to respond to positive alcohol-related outcome expectancy words, relative to negative alcohol-related outcome expectancy words, but this difference was not seen in participants who received placebo. However, in this study, the responses to the outcome expectancy words were not influenced by the type of prime word (alcohol-related *vs* neutral) which preceded them, in either group. The authors interpreted their results as indicating that alcohol priming doses may selectively increase the speed of activation of positive (but not negative) alcohol outcome expectancies; however, the alcohol priming dose did not appear to increase the ability of alcohol cues to facilitate responding to positive alcohol outcome expectancy words.

Schoenmakers *et al.* [120] reported that heavy drinkers were significantly faster on the stimulus-response compatibility task when required to categorise alcohol-related pictures by making a symbolic approach, rather than an avoidance movement (suggesting that alcohol-related cues elicit an automatic approach, rather than an avoidance response), thereby replicating previous results [61]. However, unexpectedly, this was unaffected by administration of 0.3g/kg of alcohol (compared to administration of a placebo). Therefore, at present there is no evidence to suggest that alcohol priming doses can increase automatic alcohol-approach associations. However, as only one study has investigated this issue, this remains an important topic for future research. For example, it is possible that other

measures of action approach tendencies (for example the AAT [102], or the approach-avoidance IAT), might be more sensitive to the effects of alcohol administration.

With regard to attentional biases, two studies investigated the effects of alcohol priming doses on attentional biases for alcohol-related cues. In the first study, Duka and Townshend [121] showed that, relative to placebo, a priming dose of 0.3g/kg of alcohol increased attentional bias for alcohol-related pictorial cues when assessed with a visual probe task, in a sample of heavy drinkers. However, attentional bias after a 0.6g/kg dose was not significantly different from the attentional bias observed in a group that received placebo. The authors also measured attentional bias for alcohol-related words using an alcohol Stroop task, and this measure revealed that participants who had received the high dose made more errors when colour-naming alcohol-related words, compared to participants who had received the low alcohol dose or the placebo; however, there were no effects of either alcohol dose on colour-naming times for alcohol-related words.

Schoenmakers *et al.* [120] compared the effects of a 0.3g/kg alcohol priming dose and a placebo, on attentional bias for alcohol-related cues. To measure attentional bias, we used a visual probe task with alcohol-related pictures, presented for 2000ms, with concurrent eye movement monitoring. As in previous studies [126, 127], our dependent measures included the conventional reaction time index of attentional bias (latencies to respond to probes that replaced alcohol and control pictures), and two eye movement measures: total ‘dwell time’, or time spent looking at alcohol *vs* control pictures over the duration of each trial, and ‘attentional orienting’, which was the percentage of trials in which the initial eye movement was directed at the alcohol-related picture (rather than the control picture).

Results indicated that all three measures of attentional bias were significantly increased in participants after they had received the alcohol priming dose, compared to after they had received a placebo. That is, we replicated the findings from Duka and Townshend [121] by demonstrating elevated attentional bias for alcohol cues based on a manual reaction time measure, after 0.3g/kg compared to placebo. Furthermore, elevated attentional bias after alcohol compared to placebo was seen with the two eye movement measures, as participants looked at alcohol-related pictures for longer than control pictures after the alcohol prime, but not after placebo (the ‘dwell time’ measure of attentional maintenance), and participants directed a greater proportion of their initial fixations toward alcohol cues after the alcohol prime, but not after placebo (the measure of initial orienting of attention). These results tentatively suggest that alcohol priming doses increase the ‘attention grabbing’ properties of alcohol-related cues. However, further research is required to more clearly elucidate the psychological mechanisms involved. For example, is there an inverse-U relationship between alcohol priming dose and attentional bias, as Duka and Townshend [121] suggest, such that a low dose of alcohol can prime attentional bias but a higher dose of alcohol has no effect? Secondly, given that alcohol priming doses increase subjective craving, and subjective craving is thought to have a reciprocally excitatory relationship with attentional bias [98, 128], are the effects of alcohol priming on attentional bias mediated by priming-induced increases in alcohol craving? Finally, as we discuss in section 3c, alcohol priming doses seem to impair inhibitory control, particularly inhibitory control over attention. One possibility is that alcohol priming doses increase attentional biases because they remove inhibitory control over the ability of powerful incentive-motivational cues (such as alcohol cues) to ‘grab the attention’ (see [76], discussed in section 3c, below). If this is the case, one would expect priming-induced increases in attentional biases to be mediated by the effects of alcohol primes on measures of inhibitory control.

To date, no investigators have examined how alcohol priming doses influence other indirect measures of alcohol-related cognitive processes, and we suggest that this should be a

research priority. For example, given that responses on various versions of the IAT can fluctuate in response to food and nicotine deprivation, and mood states [129-133], it seems plausible to assume that they might also be influenced by an alcohol priming dose (note that none of the studies cited above used indirect measures to specifically assess alcohol-related memory associations). One relevant issue here is that the reliability and validity of indirect measures may be affected by acute alcohol use. For example, research indicates that aspects of executive function (switch ability) have an important influence on IAT performance [134]. Given that alcohol has marked effects on aspects of executive function (see following section), it is possible that acute intoxication may adversely affect the reliability of the IAT. In common with the themes identified throughout this section, it is also incumbent upon future researchers to investigate any dose-response relationships and temporal characteristics of the effects of alcohol on indirect measures of alcohol-related cognitions.

c. Impulsive Decision-Making and Impaired Inhibitory Control

Laboratory studies with humans and animals suggest that both inhibitory control and impulsive decision-making may be influenced by acute alcohol intoxication. As described in section 2c, inhibitory control deficits have been measured with ‘Go/No-Go’ and ‘Stop’ tasks, both of which measure the ability to inhibit inappropriate responses. In non-dependent participants, alcohol dose-dependently impairs inhibitory control in these tasks [135, 136] (see [76], for a review), yet at these doses alcohol does not impair the latency to initiate the behavioural ‘Go’ response. Results from a recent study are particularly relevant to the present review, as they suggest that these effects of acute alcohol are most pronounced in frequent binge drinkers [137]. Therefore, individuals who binge drink regularly may experience the greatest disruptions to inhibitory control when they are intoxicated. Similar effects of acute alcohol (i.e., selective disruption of inhibitory control) have been observed in rats (e.g., [138]; see [77], for a review). With regard to impulsive decision-making, as assessed with the delay discounting procedure, the evidence is more mixed. In rats, alcohol increases the preference for small immediate rewards over larger delayed rewards [139, 140], but in humans, the effects of acute alcohol on delay discounting are inconsistent across studies [141-143], which may be partly attributable to methodological differences between studies [142]. Given the paucity of research in this area, it is important to clarify the nature of the effects of acute alcohol on measures of inhibitory control and impulsive-decision making.

Fillmore [76] discussed the intriguing possibility that inhibitory control over selective attention might be particularly disrupted by priming doses of alcohol. For example, Fillmore *et al.* [144, 145] demonstrated that, after administration of a placebo, social drinkers exhibit a clear ‘negative priming’ effect, in that attempting to suppress attentional processing of a stimulus attribute (e.g., its colour) on a given trial can lead to impaired (i.e., slower) responding to that attribute on a subsequent trial. This effect is thought to depend on inhibitory control over attention – the participant is required to inhibit attention to a given stimulus attribute on one trial, and this inhibition of selective attention appears to ‘carry over’ to the subsequent trial. Importantly, in these two studies [144, 145], administration of an alcohol priming dose instead of a placebo led to the complete elimination of this ‘negative priming’ effect, indicating a failure of inhibition of attention. As we describe in section 5, this raises the possibility that alcohol may have a disinhibitory effect on the control of selective attention for salient environmental cues.

4. EFFECTS OF BINGE DRINKING 2: ALCOHOL CUES

a. Self-Report Measures

Numerous authors have suggested that exposure to discrete alcohol-related cues or alcohol-related contexts might increase the accessibility of positive alcohol outcome expectancies (e.g., [146]). For example, according to encoding specificity theory [147], alcohol outcome expectancies should be more readily retrieved from memory after exposure to cues that have previously been associated with alcohol consumption. With regard to discrete alcohol-related cues (e.g., exposure to alcohol-related beverages), results have been fairly mixed. In an early study, Cooney *et al.* [96] found that both alcoholics and social drinkers responded to an alcohol cue (holding and sniffing their preferred beverage) with increased expectations of pleasant alcohol effects, but decreased expectations of stimulation and behavioural impairment from drinking; these changes were accompanied by increased alcohol craving, and the changes in expectations of pleasant alcohol effects were significantly correlated with changes in craving. However, Schulze and Jones [148] tested social drinkers and found no effect of discrete cues (holding, sniffing and tasting alcohol cues) on expectancies, although the alcohol cues produced increases in subjective craving for alcohol.

However, the effects of cues on alcohol outcome expectancies are more consistent when broader *contextual* cues are considered, such as comparing the effects of testing participants in a bar setting (real or simulated) versus a laboratory or other alcohol-neutral setting. Wall *et al.* [149] found that social drinkers expected greater alcohol-related stimulation/perceived dominance and pleasurable disinhibition when assessed in a campus bar compared to when assessed in a laboratory; however, the context had no effect on negative alcohol outcome expectancies, in this case expectations of behavioural impairment from alcohol. In a second study, the effects of the bar context on positive outcome expectancies were largely replicated (positive outcome expectancies were increased when tested in a bar compared to an alcohol-neutral context). However, in this study, the effects of the bar context on negative outcome expectancies were variable: expectations of behavioural impairment and (negative) self-perception were reduced, but expectations of risk and aggression were unexpectedly increased when tested in the bar setting. In this study, the authors also assessed the speed of response to the different outcome expectancy items, which arguably provides a more indirect measure; these results are described in the following section.

Converging evidence comes from a study reported by MacLatchy-Gaudet and Stewart [150]. Rather than explore effects of *in vivo* exposure to different contexts on alcohol expectancies, those authors used a series of vignettes to explore the effects of participants (undergraduate females) imagining themselves in different contexts on different alcohol outcome expectancies. Participants were asked to imagine being either (a) with friends at a bar (social context), (b) involved in a date leading up to a possible sexual encounter (sexual context), and (c) at home alone at the end of a stressful day (tension context); after exposure to each vignette, participants completed an alcohol expectancy questionnaire that specifically assessed their beliefs about the effects of alcohol in each context. Results indicated that expectations of increased global positive affect were highest in the social context compared to the sexual and tension contexts; however, expectations of arousal were highest in the sexual context compared to the social and tension contexts.

Taking all of these studies together, we suggest that a general pattern can be seen. That is, testing social drinkers in an alcohol context such as a bar leads to changes in self-reported outcome expectancies, either increases in positive alcohol outcome expectancies or decreases in negative outcome expectancies. Comparisons across studies are hindered by the use of different manipulations and different measures of expectancies; the findings that some specific types of negative expectancies can be increased whereas others might be

decreased when tested in an alcohol context suggest the need for further research to clarify these issues.

With regard to subjective craving, it is apparent that alcohol-related discrete and contextual cues reliably elicit increases in self-reported alcohol craving in alcoholics. For example, Carter and Tiffany [124] conducted a meta-analysis of the effects of cue exposure on alcohol craving among alcoholic participants and reported an average effect size of 0.53 (Cohen's *d*) for subjective craving, which, although classed as a 'medium' sized effect, was not as large as the effect size for cue-elicited craving among other substance using groups (e.g., tobacco smokers). Other investigators have suggested that individual differences in the ability of alcohol cues to elicit craving may confer an enhanced risk of relapse to drinking in alcoholics, although the evidence for this is inconsistent [95].

Among non-dependent social drinkers, similar effects of alcohol cues on craving have been reported. McCusker and Brown [151] found that, among social drinkers, craving was higher when in an alcohol-related context compared to when in a context unrelated to alcohol. Mucha *et al.* [152] found that pictures which depicted the preparation for or actual drinking, but not cues depicting the end of a drinking episode (e.g., an empty beer bottle), elicited robust increases in subjective craving in social drinkers. Willner *et al.* [153] reported that exposure to an alcohol cue (consumption of a non-alcoholic beer which participants were led to believe contained alcohol) produced increased craving in male, but not female, participants. Schulze and Jones [122, 148] found that alcohol cue exposure (looking at, sniffing, and sipping alcoholic beverages) increased scores on various subscales of the DAQ.

To briefly summarise, it appears that both alcoholics and social drinkers respond to presentation of alcohol-related cues with increases in subjective craving. However, the effects appear to be most robust for alcoholics; among social drinkers, the specific effects observed are inconsistent across studies (e.g., gender differences). Indeed, some studies have directly compared alcohol cue reactivity in alcoholics and controls and concluded that only the former group show a robust craving response [154], in some cases accompanied by distinctive patterns of brain activation in alcoholics but not social drinker controls [154-156].

b. Indirect Measures

A few published studies examined the effects of alcohol-related contextual cues on indirect measures of alcohol cognitions. Wall *et al.* [157] reported that social drinkers responded more rapidly to sociability related alcohol outcome expectancies when tested in a bar compared to when tested in a neutral context. Havermans *et al.* [158] report results of a study in which social drinkers were presented with ambiguous alcohol-related prime words (e.g., 'draft') and subsequently asked to generate sentences containing these prime words. Half of the participants were tested in an alcohol-related context (a simulated bar), and the remaining participants were tested in a neutral context (a simulated office). The number of alcohol-related sentences that were generated in response to these primes formed the measure of alcohol related memory-associations. As predicted, results indicated that those participants who were tested in the alcohol-related context (a simulated bar) generated more alcohol-related sentences compared to those participants who were tested in the neutral context.

To date, there is very little research regarding the issue of how alcohol-related environmental cues might influence performance on other indirect measures, such as the IAT or measures of action approach tendencies (for some preliminary results presented at a conference, see [159]). We predict that alcohol-related contextual cues should increase the

accessibility of associations between ‘alcohol’ and concepts such as ‘approach’, ‘arousal’, and positive valence; contextual alcohol cues should also increase the speed of action approach tendencies directed toward alcohol cues. These predictions should be tested in future research.

Regarding attentional bias, some studies suggest that alcohol-related contextual cues can increase the magnitude of attentional bias for discrete alcohol-related cues. In the first study [160], heavy and light social drinkers completed an alcohol Stroop task either in a context that was either alcohol-related (alcohol advertisements were placed on the walls of the testing environment) or not (photographs of guitars were placed on the walls of the testing environment). Results indicated that the magnitude of attentional bias for alcohol-related cues was significantly greater in heavy drinkers who completed the task in the alcohol-related context, compared to either heavy drinkers tested in the alcohol-unrelated context, and to light drinkers tested in either context. These results clearly suggest then, that attentional bias in heavy drinkers is most pronounced when those drinkers are tested in an alcohol-related context. A second study [161], used a different cue exposure manipulation and a within-subject rather than a between-subject experimental design, but demonstrated comparable effects of alcohol cues on attentional bias to those demonstrated in their 1999 study. In this latter study, individual differences in average weekly alcohol consumption were positively correlated with the magnitude of attentional bias for alcohol-related words (as assessed with a Stroop task), but only if participants had been exposed to an alcohol cue (involving holding and sniffing an alcoholic drink) before completing the Stroop task; among participants who were exposed to a non-alcoholic control cue before completing the Stroop task, there was no association between average weekly alcohol consumption and attentional bias. Although this latter study does not unequivocally demonstrate that alcohol cues increase attentional bias for alcohol-related words among heavy drinkers, the results are consistent with a more general argument advocating that attentional bias for alcohol-related cues is determined by the interaction of environmental context (alcohol-related *vs* alcohol-unrelated) and type of drinker (heavy versus light).

As with the research showing effects of alcohol priming doses on attentional bias for alcohol-related cues, these issues require further investigation in order to clarify the mechanisms by which alcohol-related cues and contexts can increase attentional bias for discrete alcohol-related cues, including words. For example, given that alcohol cues increase subjective craving, and subjective craving is thought to have a reciprocally excitatory relationship with attentional bias [98, 128], are the effects of alcohol cues on attentional bias mediated by cue-induced increases in alcohol craving? This issue cannot be addressed by the two studies discussed [160, 161] as the investigators did not measure subjective craving. Furthermore, as described previously, research on attentional biases in addiction can be separated into studies that show rapid and automatic attentional capture by addiction-related stimuli [162], versus studies that show the enhanced maintenance of attention, or delayed disengagement of attention from, addiction-related stimuli [127-128]. One question for future research is: do contextual alcohol cues selectively influence only one specific component of attentional bias for discrete alcohol-related cues?

c. Impulsive Decision-Making and Impaired Inhibitory Control

To our knowledge, no study has yet investigated whether heavy social drinkers or alcoholics make more impulsive decisions (e.g., on delay discounting tasks) or have particularly compromised inhibitory control (as assessed with the Stop task, for example), when tested in an alcohol-related context (e.g., a bar) compared to when tested in an alcohol-unrelated context. However, as we describe in section 5, below, numerous theoretical views would appear to converge on the common prediction that alcohol-related cues should increase impulsive decision-making or impair inhibitory control in heavy drinkers and alcoholics.

However, some very recent studies suggest that inhibitory control deficits might be exacerbated when discrete alcohol-related cues are embedded in tests of inhibitory control. Noel *et al.* [81] devised the 'Alcohol Shifting Task', a variant of the Go / No-Go procedure, to explore the ability of alcohol-related words to influence performance on an inhibitory control task in alcoholics and non-alcoholic controls. In the task, participants were required to respond to target words and to disregard distracter words; inappropriate responses to distracter words are taken as a measure of inhibitory control. In some blocks of the task, alcohol-related words served as targets and neutral words as distracters, whereas in other blocks, neutral words were targets and alcohol-related words were distracters.

Results indicated, firstly, that although alcoholics were generally slower than controls to detect targets, this difference was significantly smaller when alcohol-related words (rather than neutral words) served as targets, which perhaps indicates preferential processing of alcohol-related words in alcoholics, i.e., an attentional bias (see section 2). With regard to 'false alarms' (inappropriate responses to distracters), results indicated that alcoholics made more false alarms than controls, which is indicative of a general deficit in inhibitory control. More importantly, the authors used a signal detection analysis and demonstrated that the tendency of alcoholics to make more inappropriate responses than controls was particularly pronounced when alcohol-related words, rather than neutral words, were the targets. Therefore, these results can be understood as indicating heightened deficits in inhibitory control among alcoholics when they are required to respond to alcohol-related words and to suppress responses to neutral words.

In a subsequent study, Noel *et al.* [163] repeated the basic procedure with a different group of alcoholic and control participants. Results indicated that, again, alcoholics made more inappropriate responses to distracters when alcohol-related words were targets and neutral words were distracters, although, unfortunately, the appropriate post-hoc statistical tests were not reported. These two studies are broadly consistent with Noel *et al.* [81, 163] argument that alcohol-related words promote inappropriate responding to distracter stimuli (or, deficient inhibitory control). However, as noted by Field and Cole [164], there are some problems with this interpretation of the data. Firstly, both studies appear to show inappropriate responding to neutral distracter stimuli among alcoholics when alcohol-related words serve as target stimuli. If alcohol-related words can exacerbate inhibitory control deficits, one might expect the opposite pattern of results, i.e., alcoholics should make more inappropriate responses to alcohol-related words when they serve as distracters and neutral words as the targets (but see [165], for a counter-argument). We suggest that, although these studies are broadly consistent with the notion that alcohol-related words can exacerbate inhibitory control deficits in alcoholics, the precise nature of these effects needs to be elucidated in future research.

5. THEORETICAL SYNTHESIS

In this manuscript we have reviewed evidence which suggests that heavy drinking and alcoholism are associated with a cluster of changes in cognitive processes, including self-report measures, indirect measures of alcohol-related cognitions, impulsive decision-making, and impaired inhibitory control. These changes, and their inter-relationships, are consistent with numerous models of addiction and craving. However, these are not stable 'trait' variables in alcoholics and heavy drinkers; instead, they appear to be particularly pronounced when provoked by (a) 'priming' effects of acute alcohol ingestion, and (b) exposure to discrete and contextual environmental cues. We do not want to state that these are the only possible factors to influence cognitive processes in binge drinking. For example, there are some indications that negative mood can influence cognitive processes in alcohol use [166, 167]. Our argument, which we outline in full below, is that alcohol binges involve

both alcohol consumption and exposure to alcohol-related cues. As such, during an alcohol binge, we suggest that individuals become more likely to engage in heavy drinking as a *consequence* of these changes in cognitive processing.

As we have discussed, subjective craving for alcohol is elevated after administration of alcohol, and these effects appear to be dose-dependent; subjective craving is also elevated after exposure to environmental alcohol-related cues. Similarly, subjectively-rated beliefs about the effects of alcohol become more positive when in an alcohol-related context, and we suggest that alcohol priming doses may produce similar increases in positive alcohol outcome expectancies, a prediction which should be tested in future research. With regard to indirect measures of alcohol-related memory associations, the available evidence is limited, but we suggest that further research is required to investigate our predictions that alcohol priming doses and exposure to alcohol-related cues should facilitate some implicit alcohol-related cognitions; specifically, ambiguous alcohol-related cues should more readily be able to prime alcohol-related memory structures, and implicit associations between alcohol and concepts such as positive valence, arousal, and approach tendencies should be facilitated. With regard to attentional bias for alcohol-related cues, there is some preliminary evidence to suggest that alcohol priming doses and exposure to alcohol-related cues can increase the ability of discrete alcohol-related cues to ‘grab the attention’, when assessed with numerous measures. However, further research is required to examine if these effects are dose-dependent and to explore the subcomponents of attention which are involved. Finally, there is evidence to suggest that alcohol intoxication dose-dependently impairs inhibitory control, and some suggestive evidence that alcohol may also increase impulsive decision-making; some recent studies also suggest that the presence of alcohol-related cues can produce further deficits on inhibitory control measures, although the effects of alcohol cues on impulsive decision-making have not yet been investigated.

To briefly summarise our argument: As a consequence of alcohol priming effects and exposure to alcohol-related cues during an alcohol binge, we would expect individuals to experience elevated craving for alcohol, increased positive outcome expectancies for alcohol use, altered implicit alcohol-related memory associations, increased action approach tendencies elicited by alcohol-related cues, increased attentional bias for alcohol cues, impaired inhibitory control over attention and behaviour, and increased impulsive decision-making. Some of the effects of acute alcohol are dose-dependent; therefore, as an alcohol binge progresses and more alcohol is consumed, we would expect alterations in these aspects of cognitive processing to become more pronounced. Importantly, these ‘temporary’ consequences of alcohol ingestion and exposure to alcohol-related cues act to make the intoxicated individual temporarily similar to an alcohol abuser in terms of these cognitive processes. For example, various models of addiction suggest that elevated craving and attentional bias [93, 98], increased positive outcome expectancies for alcohol use [89, 90], implicit alcohol-arousal associations [44], increased impulsive decision-making [84], and compromised inhibitory control over drives and behaviour [104] are all characteristics of substance abusers that occur as a consequence of experience with substances of abuse and which act to maintain substance-seeking in the future. As such, a social drinker who experiences these effects during an alcohol binge would be expected to lose control over their drinking in much the same way as someone diagnosed with alcohol dependence.

It is important to consider that these cognitive ‘symptoms’ that can be observed during a binge do not operate in isolation. We have presented the evidence for effects of alcohol priming doses and alcohol-related cues on these separable aspects of cognition in order to simplify our argument; however in reality there are numerous inter-relationships between these different aspects of cognition. For example, when attentional bias for alcohol-related cues is experimentally increased, subjective craving for alcohol increases as a result [100,

101]. As such, one might expect alcohol cues and priming doses to independently increase attentional bias and subjective craving, but once one of these variables starts to increase, this might provoke further increases in the other variables. Similarly, increases in positive alcohol outcome expectancies after alcohol cue exposure are correlated with increases in subjective alcohol craving [96]. Therefore, exposure to alcohol-related cues might increase both alcohol craving and positive outcome expectancies simultaneously, but changes in one might also produce subsequent changes in the other. Finally, some recent evidence suggests that, among adolescent heavy drinkers, impulsive decision-making and attentional bias are correlated with each other [37], and Fillmore [76] suggests that alcohol intoxication might specifically impair inhibitory control over selective attention. Taken together, these results may suggest that alcohol cues and priming doses could increase attentional bias and inhibitory control independently, but that impaired inhibitory control might confer a reduced resistance to the 'attention grabbing' properties of alcohol-related cues, thereby indirectly increasing attentional bias for those cues.

Finally, any discussion of inter-relationships between different aspects of 'implicit' and self-reported cognitive processes is reminiscent of a broader debate within psychology. That is, it is slowly becoming accepted that a variety of behaviours are influenced by both implicit and explicit cognition, and that implicit and explicit cognition might account for different aspects of behaviour [168]. In this broader context, there are numerous examples of implicit and explicit cognition having independent influences on behaviour, and other instances where their influences are multiplicative [169]. Returning to our discussion of binge drinking, it remains a target for future research to investigate which of these changes in cognition are the most important determinants of alcohol consumption during a binge, and which, if any, are 'primary' in the sense that they influence all of the other aspects of cognition which we identify here. Consideration of this final point is likely to lead to a better understanding of the psychological processes that lead to the loss of control over alcohol consumption seen during an alcohol binge, which in turn is likely to inform novel interventions for the management of binge drinking and its consequences.

6. IMPLICATIONS FOR THE MANAGEMENT OF BINGE DRINKING, AND HARM REDUCTION

The research findings and novel theoretical predictions discussed in this review have a number of implications for the reduction of alcohol consumption during an alcohol binge, and for reduction of the harms associated with binge drinking. We make two general points: Firstly, educating individuals about the changes in their subjective experiences which can occur during an alcohol binge can be helpful, but only up to a point. Many of the cognitive processes discussed may be relatively 'automatic', and neither consciously accessible or amenable to conscious control. With this in mind, our second general point is that education and standard psychological interventions may not be suitable for changing these automatic cognitive processes; however, behavioural interventions may have potential utility for changing these aspects of cognition directly.

a. Self-Report Measures

Some evidence provides clues as to how individuals may be able to counteract increases in their subjective states (specifically, craving and outcome expectancies) which occur during an alcohol binge. For example, cue exposure treatment, in which individuals are exposed to discrete alcohol-related cues whilst being prevented from consuming alcohol, does not appear to be particularly effective at reducing rates of relapse to heavy drinking in alcoholism [170]. However, those authors noted a number of factors which may well limit the effectiveness of cue exposure treatment, as traditionally applied, and at least two of those

are highly pertinent to the present review. Conklin and Tiffany [170] argue that after extinction, responses to discrete-alcohol related cues are likely to recur if the individual experiences those cues in an alcohol-related context (a *renewal* effect) and if the individual consumes alcohol (a *reinstatement* effect). As we have noted in this review, these two factors (alcohol-related contexts and alcohol priming effects) are the two factors that are common to an alcohol binge. As Conklin and Tiffany [170] themselves noted, the efficacy of cue exposure treatment – in terms of its ability to reduce subjective craving and actual consumption – might be increased if individuals experience cue exposure treatment whilst in an alcohol-related context. One possibility is that this approach – cue exposure treatment conducted in an alcohol-related context – might also be useful for reducing subjective craving and positive outcome expectancies during an alcohol binge. A related although more controversial option may be to extinguish these subjective responses while individuals are actually intoxicated, in a controlled setting – this may reduce the likelihood of reinstatement of subjective responses when the individual next consumes alcohol outside of the treatment setting.

With regard to alcohol outcome expectancies, numerous investigators have investigated the utility of a manipulation known as ‘expectancy challenge’, which aims to alter individuals’ outcome expectancies for alcohol use. The basic expectancy challenge procedure [171] involves a group of participants interacting with each other, after consumption of alcohol or placebo beverages. After these interactions, participants are required to identify which of the other participants had received alcohol, and which placebo, based on their behaviour; participants are also required to guess if they had received alcohol or placebo themselves. Participants are generally very poor at correctly detecting if they had received alcohol or placebo, and similarly poor at making decisions about whether others have received alcohol, based on their behaviour. Therefore, this procedure provides a concrete example of how behaviours attributed to the pharmacological effects of alcohol can actually be a consequence of beliefs or expectancies about alcohol effects; this demonstration of expectancy effects is subsequently reinforced by a lecture on alcohol expectancy effects and their relationship to drinking behaviour.

Although the initial expectancy challenge studies [171, 172] yielded promising findings – participants who were exposed to the expectancy challenge showed significant reductions in their alcohol consumption, compared with participants who received a control manipulation – these effects have not been replicated in subsequent research (see [13], for a review). Nonetheless, the procedure might hold promise for altering expectancies and alcohol consumption in subgroups of heavy drinkers [50, 173, 174]. Of particular relevance here, research has demonstrated that young binge drinkers hold particularly positive expectancies for a high dose of alcohol [18, 173, 174]. Expectancy challenge treatments which specifically aim to manipulate outcome expectancies for high doses of alcohol might be particularly relevant for heavy binge drinkers. Challenging heavy drinkers’ outcome expectancies for high doses of alcohol (rather than for undetermined doses of alcohol) might be expected to reduce the quantity of alcohol consumed during an alcohol binge, but such procedures are difficult to implement in heavy drinking participants [173, 174].

b. Indirect Measures

Experimental manipulations which aim to influence indirect measures of alcohol-related cognitive processes have only recently been studied. An important issue is that these cognitive processes, which may occur spontaneously and may be difficult for participants to introspectively report on or consciously control, may need to be targeted directly, rather than using explicit psychological therapies (such as instructing people to suppress their attentional bias for alcohol). For example, Wiers *et al.* [102] used a modified version of the Alcohol Approach Action Tendency (alcohol AAT) task to ‘train’ heavy drinkers to direct

either approach, or avoidance responses to alcohol cues. Results indicated that those participants who had been trained to move away from alcohol cues subsequently drank less beer during a laboratory 'taste test' compared to participants who had been trained to move toward those cues. Similarly, we [100, 101, 175] have used 'attentional training' procedures to attempt to directly reduce the magnitude of attentional biases for alcohol cues, and we have examined the effects of these procedures on subsequent alcohol consumption. Results from all three studies show that it is possible to reduce attentional biases in heavy drinkers. However, contrary to predictions, none of these studies found that this reduction in attentional bias led to a reduction in alcohol consumption during a taste test. On a more positive note, in the first clinical study on attentional retraining in alcohol-dependent patients, the experimental reduction of attentional bias produced some improvements in clinical outcome [176].

It is promising to note that alcohol-related cognitive processes, as assessed with indirect measures, can be successfully modified (see also [177]). Future researchers may wish to extend this research by examining if performance on other indirect measures (e.g., spontaneous memory associations, the alcohol IAT) can also be successfully 'trained' with direct experimental manipulations. The recent research findings suggest that there are no consistent effects of altered performance on indirect measures on subsequent alcohol consumption. However, we stress that this type of research is in its infancy, and future research findings may shed some light on which specific aspects of implicit alcohol-related cognitive processes should be manipulated in order to reduce subsequent alcohol consumption. A further question for future researchers is to examine if any effects of training on subsequent cognitive processing are maintained when participants are intoxicated and in the presence of environmental alcohol-related cues (i.e., during an alcohol binge).

c. Impulsive Decision-Making and Impaired Inhibitory Control

It is possible that future interventions may be able to partially ameliorate the increased impulsive decision-making and impaired inhibitory control which occur during an alcohol binge; it is also possible that these interventions may have beneficial effects even during an alcohol binge. Regarding impulsive decision-making, some researchers (e.g., [178]) have suggested that impulsive choice can be reduced in individuals if they are encouraged to 'bundle' series of small immediate and larger delayed rewards together. That is, individuals tend to prefer an immediate reward (in this context, alcohol intoxication) in preference to a delayed reward (in this context, feeling well and being able to go in to work or college on the following day). However, if they are encouraged to 'bundle' these immediate and delayed rewards over a longer time period – for example, if they have to imagine drinking every day during the coming week versus *avoiding* an alcohol hangover during every day in the coming week, then this may reduce their impulsive choice – it may increase the likelihood that they will choose not to drink excessive amounts of alcohol during an alcohol binge, even when faced with a highly salient immediate reward (the possibility of starting an alcohol binge).

Alternatively, in a delay discounting paradigm, the immediate reward is always more salient to the individual. During acute alcohol intoxication, alcohol-induced myopia might increase the salience of the immediate reward, which may explain why organisms respond impulsively when intoxicated [179]. Results from a recent experimental study [179] are consistent with this argument. Rats were exposed to a delay discounting procedure in which either the immediate or the delayed reward was made more salient by signalling its presence with a light. Results indicated that alcohol increased impulsive choice (choice for the immediate reward in preference to the delayed reward) but, most importantly, alcohol also increased the choice for the most salient reward (the reward signalled by the light),

regardless of whether that reward was the immediate or the delayed choice. One possible implication of this finding is that it may be possible to reduce impulsive choice in humans by somehow increasing the salience of the delayed reward (e.g. being free of a hangover during the following day) when individuals are in the early stages of an alcohol binge. We appreciate that this may be difficult to accomplish, but theoretically, it should decrease impulsive choice during an alcohol binge.

With regard to inhibitory control deficits, some investigators have used training to improve the executive functions of children with attention deficit hyperactivity disorder, and observed effects of this training on impulsivity symptoms (e.g., [180]). It therefore remains a possibility that those individuals with compromised inhibitory control might benefit from similar training. These benefits may, in principle, remain whilst participants are engaged in an alcohol binge, so this may in turn ameliorate the inhibitory control deficits which occur during the binge.

7. SUMMARY AND CONCLUSIONS

In this paper, we have reviewed evidence which suggests that various aspects of cognitive processing are likely to be altered during an alcohol binge. We can distinguish three broad categories of cognitive processes which are altered: self-report measures, indirect measures, and 'impulsivity', which includes impulsive decision-making and impaired inhibitory control. In some instances, the available evidence for these cognitive changes is limited, and in these cases we make clear predictions which should be tested in future research. We feel that this model provides a useful heuristic for understanding the psychological processes that are involved during an alcohol binge, and future research should aim to clarify the model and hopefully derive useful interventions from it; these may, in turn, reduce the level of alcohol consumption during a binge, and reduce the harms associated with binge drinking.

Key Learning Objectives

1. What aspects of cognitive processing are likely to change during an alcohol binge?
2. How do alcohol cues and acute alcohol effects lead to these changes in cognitive processes?
3. How might these cognitive processes lead to loss of control over alcohol consumption during an alcohol binge?
4. How can this knowledge be applied to limit alcohol consumption during an alcohol binge, or to reduce the harms associated with binge drinking?

Future Research Questions

1. Are the effects of acute alcohol on cognitive processing dose-dependent?
2. We know that alcohol cues influence craving, but how might they influence other cognitive processes?
3. How do these different cognitive processes interact during an alcohol binge?
4. Which specific aspects of cognitive processing should be targeted in order to limit alcohol consumption during an alcohol binge, or to reduce the harms associated with binge drinking?

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