AN INVESTIGATION INTO THE EFFECTS OF ACUTE ALCOHOL ON THE INHIBITORY MECHANISMS OF CONTROL INVOLVED IN VISUAL PERCEPTION

By

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ABSTRACT

The impairing effects of alcohol on attention are well documented, and there is reason to believe that inhibitory mechanisms may be involved although the specific nature of the impairment is unclear. Research suggests that intentional control mechanisms might be more vulnerable to alcohol, although the evidence is not conclusive. Ambiguous figures provide a novel way to assess these processes, as attention needs to be directed towards one interpretation and away from the alternate interpretation, which must be inhibited. The contribution of both intentional and automatic mechanisms can also be assessed by consciously controlling reversals or reporting them under passive viewing conditions. The results do not support the alcohol myopia model as alcohol had a facilitatory effect on reversals. Instead, the results seem to be broadly in line with an alcohol-induced impairment on intentional inhibitory processes, although the results are not straightforward. Alcohol does not result in more figure reversals being reported simply because inhibition is weakened. Its effect on reversals seems to depend upon the precise nature, the relative, and the absolute strengths of the two interpretations of the stimulus presented and is dependent upon the specific experimental conditions. These findings are clearly contrary to a simple account based on reduced inhibition.

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ALCOHOL PHARMACOLOGY, BEHAVIOURAL, AND COGNITIVE EFFECTS

1.1. INTRODUCTION

Alcohol is known to impair functioning in a variety of domains including memory, attention, behavioural control, and information processing and its effects have been extensively studied in laboratory research (e.g. Fillmore et al., 2009; Soderlund et al., 2005; Schulte et al., 2001; Lyvers, 2000; Sayette, 1999; Koelega, 1995; Hindmarch et al., 1992). Although it has been assumed that these varied effects arise from alcohol's impairment of cognitive functioning, research findings have been unclear whether these effects are due to a global impairment of cognition or rather some specific impairment of specific cognitive processes.

Moderate doses of alcohol have been shown to impair cognitive inhibitory mechanisms of behavioural control, with the suggestion that this impairment could underlie many negative behavioural effects associated with alcohol consumption (Casbon et al., 2003; Curtin & Fairchild, 2003; Vogel-Sprott et al., 2001; Steele & Southwick, 1985). The precise nature of this impairment is not understood, although impairment in behavioural control may be related to difficulties in maintaining attention on current tasks and/or deficits in inhibiting a prepotent response (Easdon et al., 2005; Abroms et al., 2003; Fillmore, 2003; Marczinski & Fillmore, 2003a; Fillmore & Vogel-Sprott, 1999).

The impairing effects of alcohol on attention are well documented (Koelega, 1995), with impaired performance on numerous aspects of attention including divided attention (Fillmore et al., 1998), selective attention (Fillmore et al., 2000a, 2000b; Tzambazis & Stough, 2000), and vigilance and sustained attention tasks (Vermeeran & O'Hanlon, 1998; Wilkinson, 1995). There is reason to believe that inhibitory mechanisms may be involved in these attentional deficits (Fox, 1995). For example, inhibitory mechanisms have been implicated in selective attention, especially in contexts in which cognitive resources are to be directed toward relevant stimuli and away from irrelevant stimuli (Houghton & Tipper, 1994). It has been proposed that alcohol impairs the inhibitory mechanism that directs attention away from irrelevant information (Abroms & Fillmore 2004; Fillmore et al. 2000a, 2000b). The suggestion is that alcohol leads to a narrower focus of attention, which lead to the proposal of the attention-allocation model (e.g. Steele & Josephs, 1990; Steele & Southwick, 1985), whereby intoxication restricts the focus of attention to the most salient cues, such that all available cues may not be fully processed (Sayette, 1999).

Although recent studies on alcohol and attention highlight the importance of inhibitory mechanisms, there remain many questions concerning the specific nature of the inhibitory mechanisms impaired by alcohol. A fundamental distinction among inhibitory mechanisms concerns whether the mechanism is intentionally controlled or is automatic (Marzi, 1999; Shimojo et al., 1999). Previous research has shown that behaviours that depend on intentional control might be more vulnerable to the impairing effects of alcohol than behaviours dependent upon automatic processes (Fillmore & Vogel-Sprott, 2006; Holloway, 1995). However, others have indicated that both

automatic and intentional inhibitory mechanisms of attention can be impaired under moderate doses of alcohol (Holloway, 1995).

Ambiguous figures provide a novel way to assess inhibitory and attentional processes that are known to be impaired following alcohol consumption (e.g. Meng & Tong, 2004; Toppino, 2003; Leopold & Logothetis, 1999; Girgus et al., 1977; Fisher, 1967). Also, these figures enable the combined assessment of inhibitory and attentional mechanisms outlined above without the need to use the different methodological approaches. For example, ambiguous figures can provide insights into the inhibitory mechanisms involved in the disambiguation of ambiguous figures (Girgus et al., 1977). In order for a figure to reverse, the observer has to suppress or inhibit the currently experienced interpretation. In addition, attentional processes have been strongly implicated in figure reversals, with existing research focussing on how the allocation of attentional resources to certain features can help to disambiguate ambiguous figures (Peterson & Gibson, 1994; Tsal & Kolbet, 1985; Bugelski & Alampay, 1961). Such results suggest an important role for the allocation of attention in the perception of ambiguous figures. Furthermore, the contribution of both intentional and automatic control involved in inhibitory mechanisms can also be assessed using ambiguous figures. Ambiguous figures, such as the Necker cube (see Figure 4.1a), are figures that have two or more interpretations. As neither interpretation is more plausible than the other, conscious perception of the figure alternates between the two interpretations. For the ambiguous figure task, observers report each perceptual alternation between the two interpretations of the ambiguous figure upon its presentation. Existing research has shown that an observer has a degree of control over reversals, the reversal rate can be

increased (e.g. Seth & Reddy, 1979; Pelton & Solley, 1968), or a given interpretation can be maintained (e.g. Liebert & Burk, 1985; Peterson & Hochberg, 1983).

The aim of this thesis was to assess the effects of a moderate dose of alcohol on the inhibitory and attentional processes involved in figure reversals. This chapter will discuss general effects of alcohol on cognitive processes, particularly those affecting attentional and inhibitory processes. The discussion will then focus on the existing literature on ambiguous figures and relate these findings to what is currently known about the effects of alcohol from the existing alcohol literature.

1.2. PHARMACOLOGY OF ALCOHOL

Alcohol has a widespread effect on the brain and behaviour. Knowledge about the time course of alcohol's action in the body as well as its interaction with receptors in the brain is essential for predicting the optimal doses required to reach a desired effect and maintaining the desired level for a certain period of time. Consequently, in discussing the effects of alcohol on cognition, it is important to understand its pharmacological effects and how they may mediate any effects. The following sections will discuss several such factors and their implications for the present thesis.

1.2.1. Pharmacokinetics

1.2.1.1. Alcohol absorption

The rate that the body absorbs alcohol has implications for the speed with which the drug exerts its effects on the central nervous system. After consumption, alcohol is rapidly distributed throughout the body in the blood stream, and due to its solubility

with both fat and water, it readily crosses important biological membranes, such as the blood brain barrier, to affect a large number of organs and biological processes in the body (Boggan, 2003). Consequently, the onset of action is relatively quick. The dose typically consumed by social drinkers can result in a peak BAC within 30 minutes (Kalant, 1996), although it can take around one hour for 90 percent of alcohol to be absorbed into the bloodstream.

The passage of ethyl alcohol across biological membranes occurs by a process of simple passive diffusion along concentration gradients. Ethyl alcohol taken in via ingestion passes from the mouth down the oesophagus and into the stomach and on into the small intestine. At each point along the way alcohol can be absorbed into the blood stream. However, the majority of the ethyl alcohol is absorbed from the stomach (approx. 20%) and the small intestine (approx. 80%) (Kalant, 1996). The rate of alcohol absorption is dependent on gastric emptying (Horowitz et al., 1989; Holt, 1981), so factors that modify gastric emptying will also modulate the rate of alcohol absorption (Horowitz et al., 1989; McFarlane et al., 1986; Finch et al., 1974). Humans vary widely in their ability to absorb and eliminate alcohol; after ingestion of an equivalent, weight adjusted dose of alcohol there is considerable inter-individual and intra-individual variation in the rate of absorption and peak blood alcohol concentrations (Fraser et al., 1995; Holt, 1981). In general, the faster the contents of the gastrointestinal tract can be emptied, the more rapid is the rate of absorption.

1.2.1.2. Metabolism of alcohol in the body

Alcohol metabolism is the process by which the body breaks down and eliminates alcohol from the body. Until all the alcohol consumed has been metabolised, it is distributed throughout the body affecting behavioural and cognitive processes. Therefore, understanding the processes involved in metabolism, including factors that speed up and slow down the process, has implications for studies that assess its effects.

The liver accounts for approximately 85% of the alcohol metabolised (Julien, 2008). The first stage of metabolism is oxidation by means of alcohol dehydrogenase (ADH) to form acetaldehyde. Acetaldehyde is rapidly converted by aldehyde dehydrogenase (ALDH) and nicotinamide adenine dinucleotide (NAD), into acetate. This is then released into the hepatic venous blood, where it combines with coenzyme A to form acetyl CoA, which enters the citric acid cycle. There it is oxidised to CO_2 and H_2O, which are then excreted from the body (Lieber, 1994). A small percentage of alcohol is not metabolised in this way and is excreted from the body via the lungs, or lost in urine or perspiration (Boggan, 2003).

Alcohol is metabolised at a steady rate immediately after the drug is absorbed into the bloodstream and begins to pass through the liver. The maximum amount of alcohol that can be metabolised in 24 hours is 170 grams, approximately 12 to 18 ml/hour (Ritchie, 1985). The ADH reaction is the rate-limiting step of alcohol metabolism, but the rate of this reaction is inhibited by elevated concentrations of acetaldehyde and NADH (Crabb et al., 1983). The rate-limiting factor is the availability of NAD, which requires the oxidation of the reduced form NADH. It is the balance between the absorption rate and

the metabolic rate of the individual that determines the effect of alcohol consumption. Alcohol is metabolised more slowly than it is absorbed. Since the metabolism of alcohol is slow, consumption needs to be controlled to prevent accumulation in the body and intoxication.

1.2.2. Pharmacodymanics

1.2.2.1. General effects

Previously, it was believed that alcohol acted through a general depressant action on nerve membranes and synapses (Julien, 2008). As alcohol is both water-soluble and lipid soluble, it dissolves into all body tissues. This led to the unitary hypothesis of action that alcohol dissolves in nerve membranes, distorting, and disorganising the membrane (Julien, 2008). The result is a non-specific and indirect depression of neuronal function. This would account for the non-specific and generalised depressant behavioural effects of the drug. However, it does not explain the evidence that alcohol may disturb both the synaptic activity of various neurotransmitters and various intracellular transduction processes.

More recently, specific interactions between alcohol and major neurotransmitter systems have been identified. The pharmacological effects of alcohol appear to be the result of its interaction with these multiple systems. However, these neurotransmitters result in differential contributions to the neurochemical basis of alcohol's behavioural effects. Thus, at a certain dose, a specific receptor system may be more prominent than others in contributing to a particular behavioural effect of ethanol.

1.2.2.2. GABA receptors

One of the targets of alcohol in the CNS is the $GABA_A$ receptor. GABA, the neurotransmitter that activates $GABA_A$ receptors, is the major inhibitory neurotransmitter in the CNS (Barnard et al., 1998). Alcohol has been shown to enhance $GABA_A$ receptor function by increasing the frequency and duration of opening the chloride channel and allowing chloride ions to enter the post-synaptic neuron (Tatebayashi, Motomura, & Narahashi, 1998), which, in turn, hyperpolarizes neurons by allowing more chloride ions to enter (Zorumski & Isenberg, 1991), or at least potentiating the hyperpolarization produced by GABA (Koob, 2004; Blair et al., 1988). This results in anesthesia, sedation, and anxiolysis and is thought to play a role in many of the known behavioural effects of the drug. Drugs that increase GABAergic function, such as GABA agonists, potentiate the effects of alcohol. Whereas, drugs that decrease GABAergic function, such as receptor antagonists, reduce alcohol behaviours (see Grobin et al., 1998). Some of the effects of alcohol may also be mediated by pre-synaptic GABA release (Ariwodola & Weiner, 2004; Criswell & Breese, 2005). While much work has shown that alcohol enhances $GABA_A$ receptor potentiation, the exact mechanism of alcohol actions on these receptors remains unclear. Individual $GABA_A$ receptor subunits have not yet been definitively linked with specific behavioural actions. Since there are multiple $GABA_A$ receptor subtypes, different subunits may account for distinct alcohol-induced behavioural effects.

1.2.2.3. Glutamate

The excitatory neurotransmitter, glutamate, increases the activity of signal-receiving neurons and plays a major role in controlling brain function. Glutamate exerts its effects

on cells in part through three types of receptors that, when activated, allow the flow of positively charged ions into the cell. Of these, the N-methyl-D-aspartate (NMDA) receptor plays a particularly important role in controlling the brain's ability to adapt to environmental and genetic influences. When activated by glutamate binding, NMDA causes excitation in the postsynaptic cell by allowing positively charged ions (e.g. sodium [Na+] or calcium [Ca2+]) to enter the cell. This rapid movement of positive ions into the cell reduces the voltage difference that normally exists between the cell's interior and exterior (i.e. across the cell membrane) adjacent to the receptors. Because each neuron carries thousands of glutamate receptors, the ion flow caused by an excitatory signal can result in a depolarization sufficient to generate another excitatory signal in the postsynaptic cell. Alcohol decreases the NMDA-induced Ca2+ flow into neurons (Leslie & Weaver 1993; Hoffman et al. 1989), even low concentrations (e.g. 0.3% BAC, Lovinger et al. 1989) can inhibit ion flow through the NMDA receptor. This indicates that alcohol concentrations commonly achieved during social drinking can have an inhibitory effect on the NMDA receptor. Alcohol disrupts glutaminergic neurotransmission by depressing the responsiveness of NMDA receptors to release glutamate (Chandler et al., 1998). The decreased electrical activity may help explain the reduced neurotransmitter release in response to NMDA. This reduction of glutamate responsiveness may be intensified by its known enhancement of inhibitory GABA neurotransmission. The inhibitory effects on NMDA contribute to the known sedative effects of alcohol (Tabakoff & Rothstein, 1983). With chronic alcohol intake and persistent glutaminergic suppression, there is a compensatory up regulation of NMDA receptors (Chen et al., 1997). On removal of alcohol’s inhibitory effect, excess excitatory receptors would result in alcohol withdrawal signs.

1.2.2.4. Dopamine

Extensive literature suggests that the rewarding and reinforcing properties of alcohol are related to the stimulation of dopaminergic transmission (Bardo, 1998; Koob & Nestler, 1997). The nucleus accumbens (NAc) is an important component of the reward system, and alcohol has been shown to increase dopamine levels in the NAc (Koob & Le Moal, 2001). By reducing tonic control over dopaminergic neurons in the ventral tegmental area, there is an increase in the release of dopamine in the nucleus accumbens (Tabakoff & Hoffman, 1996), which are actions shared by non-sedative drugs of abuse (for example, cocaine, nicotine, amphetamine). Thus the effect of ethanol is not always that of depressing neuronal function, the outcome may be release of dopamine but this is due to changes in inhibition. Furthermore, low concentrations of certain depressant drugs, such as alcohol, can induce excitatory effects (neuronal as well as behavioural), either due to a transient increase in the release of excitatory transmitters or to depression of inhibitory systems (Pohorecky, 1977).

1.2.2.5. Opioids

The endogenous opioids play a key role in the rewarding properties of ethanol (Froehlich, 1995; Swift, 1995). It has been suggested that alcohol might enhance opioid receptor activity either via stimulation of the synthesis, release or processing of opioid agonists and thus indirectly stimulate opioid receptors, or directly by enhanced sensitivity of the opioid receptors to endogenous opioids (Gianoulakis, 2004). It has been well established that ethanol stimulates the endogenous opioid system, which then serves to reinforce further ethanol drinking. Numerous studies have suggested that there

is a relationship between those motivational states underlying alcohol consumption and central opioid mechanisms (Herz, 1997; Ulm et al., 1995). Alcohol may affect the release of endogenous opioid peptides (Herz, 1997), and the initial sampling of alcohol may stimulate endogenous opioid receptors, an effect that is reinforcing. This, in turn, promotes additional alcohol consumption to achieve additional opioid stimulation (Reid et al., 1991). Gianoulakis (2004, 1996) suggested the vulnerability for increased ethanol consumption is determined by individual differences in sensitivity of the opioid system to ethanol. For example, low doses of the opioid agonist, morphine (1.0–2.5 mg/kg), have been shown to increase alcohol intake (Stromberg et al., 1997; Hubbell et al., 1988). On the other hand, the suppression of alcohol consumption by opioid receptor antagonists (naloxone and naltrexone) has been reported in mice (Middaugh et al., 1999), rats (Stromberg et al., 1998; Froehlich et al., 1990; Hubbell et al., 1986), and monkeys (Kornet et al., 1991; Altshuler et al., 1980).

1.2.2.6. Cannabinoids

The endogenous cannabinoid system has been implicated in the modulation of addictive behaviour and in the mechanism of action of different drugs of abuse (Gardner, 2005). There is evidence to indicate that the endocannabinoid system is involved in the pharmacological and behavioural effects of alcohol (Basavarajappa & Hungund, 2002). Evidence suggests that endocannabinoid signalling may be involved in the modulation of alcohol reinforcing effects and alcohol drinking behaviour. Hence, the CB1 receptor antagonist rimonabant (SR141716A) decreases alcohol intake in both alcohol-preferring rats (Colombo et al., 1998) and in C57BL/6 mice (Arnone et al., 1997) and the motivation to consume alcohol in rats (Gallate et al., 2004; Gallate & McGregor, 1999).

Lower cannabinoid function also appears to be related to greater vulnerability to alcohol consumption (Ortiz et al., 2004). It remains unclear exactly how CB1 triggers the rewarding effects of alcohol, one possibility is that activation of the CB1 receptor blocks the inhibitory signals for the production of dopamine, resulting in more dopamine being released and producing a pleasure/reward response (Thanos, 2005; Perra, 2005).

1.2.2.7. Acetylcholine

Alcohol stimulates the release of acetylcholine in the hippocampus area of the brain (ACh) (Henn, 1998). The release of ACh increases cortical arousal and is thought to influence attention and memory in humans (Warburton & Rusted, 1993). Importantly, acute alcohol administration is now known to have a biphasic effect on ACh release in the prefrontal cortex (Stancampiano et al., 2004; Henn, 1998). It has been shown that low-moderate doses (<0.5 g/kg) increase ACh release, while higher doses (>0.5 g/kg) decrease cortical ACh release (Stancampiano et al., 2004). At lower doses, ACh is known to facilitate memory and attentional processes; however high alcohol intake inhibits acetylcholine production and impairs performance on these tasks (Rossetti et al., 2002; Givens, 1995).

1.2.2.8. Serotonin

Serotonergic neurons influence brain functions related to attention, emotion, and motivation. The administration of alcohol has been shown to elevate serotonin levels within the brain (LeMarquand et al., 1994; McBride et al., 1993). The rise in serotonin levels has been linked to alcohol-induced changes in mood, at low doses the increase in

serotonin can improve mood, but in excess alcohol makes these serotonin levels fall and lowers mood, increasing depression (Chick, 1999). Serotonin appears to have the opposite effect of dopamine, and serotonin receptors are thought to modulate the activity of dopaminergic reward pathways and may lead to dependence (Rocha et al., 1998b). The emphasis is on the of serotonin 5-HT_2 and 5-HT_3 receptors in the central effects of alcohol, these receptors are located on dopaminergic neurons in the nucleus accumbens. These receptors are thought to play a role in the regulations of alcohol consumption and may thus contribute to its rewarding effects (Lovinger, 1999).

1.2.2.9. Summary

Alcohol has been shown to produce many neurochemical effects, each of which is likely to contribute to the observable behavioural effects of alcohol. The combination of stimulatory and depressant effects of alcohol can determine the resulting psychological and behavioural effects associated with the drug. Furthermore, prolonged use of alcohol can alter the functioning of these neurotransmitters, which can lead to behavioural and physiological tolerance.

1.2.3. Individual differences in response to alcohol

There is considerable inter-individual and intra-individual variation in the response to alcohol (Fraser et al., 1995; Holt, 1981). Several factors can alter the rate at which alcohol is absorbed and metabolised, either increasing or decreasing the rate of elimination from the body (Lin et al., 2001). The following sections discuss some of these factors and their implications for studies that assess alcohol's effects.

1.2.3.1. Properties of the beverage

The alcohol concentration of the beverage can affect alcohol absorption. There is a curvilinear relationship between alcohol concentration and BAC. It has been shown that alcohol concentrations of 45% and 15% are absorbed at a slower rate than alcohol of 30% (Lolli & Rubin, 1943). Consequently, alcohol absorption is thought to be maximal at concentrations of 10–20% (Stark, 2005). This is because high concentrations of alcohol irritate the gastric mucosa, stimulating an increase in mucus secretion (Stark, 2005; Lolli & Rubin, 1943) and delaying gastric emptying (Lolli & Rubin, 1943), possibly due to an increase in stomach content volume caused by the excess mucus (Roine et al., 1993). This increases the amount of time the alcohol stays in the stomach, compared with a more dilute solution (Stark, 2005; Roine et al., 1991). The resulting increase in the duration of contact between the alcohol and gastric ADH results in an increased gastric metabolism and a lower peak BAC.

The form in which the alcohol is consumed is also likely to have an effect on alcohol absorption (Holt, 1981). For example, the presence of glucose in sweet drinks is known to reduce absorption rates, (Holt, 1981; Sedman et al., 1976). Alternatively, carbonated mixers have been found to increase alcohol absorption (Roberts & Robinson, 2007). This is due to the carbon dioxide releasing gas into the gastric lumen (Ploutz-Snyder et al., 1999), this causes the stomach to swell (Ploutz-Snyder et al., 1999; Ploutz-Snyder et al., 1997), resulting in increased gastric emptying (Ploutz-Snyder et al., 1999), which would consequently affect alcohol absorption rates.

1.2.3.2. Presence of food in the stomach

One of the most important factors in alcohol absorption is the presence of food. The rate of alcohol absorption is slower when a beverage containing alcohol is consumed with or after a meal rather than on an empty stomach (Horowitz et al., 1989). Drinking on a full stomach retains alcohol in the stomach, increasing its exposure to gastric alcohol dehydrogenase, which results in lower levels of alcohol in the blood. The empty stomach allows rapid passage of the alcohol into the small intestine, where absorption is most efficient (Roine, 2000; Roine et al., 1993; Roine et al., 1991), resulting in increased levels of alcohol in the blood. Furthermore, the type of food in the stomach also affects absorption, with foods higher in fat content requiring more time to leave the stomach which slows down alcohol absorption (Pohorecky & Brick, 1990).

1.2.3.3. Speed of Drinking

Another factor that influences the absorption of alcohol is the rate at which the beverage is consumed. When alcohol is consumed steadily in small amounts, the rate of metabolism of alcohol keeps pace with intake (Kalant, 2000). In general, drinking more alcohol within a certain period of time will result in increased blood alcohol concentrations (BAC) due to more ethyl alcohol being available to be absorbed into the blood (Gentry, 2000; Kalant, 2000). Rapid intake of alcohol results in more alcohol in the stomach and small intestine which produces a larger gradient of alcohol and greater absorption into the blood stream and distribution into the tissues including the brain. If alcohol is taken in more rapidly than it can be metabolised, the BAC will raise leading to intoxication (Boggan, 2003).

1.2.3.4. Use of other drugs

A further methodological consideration is the consumption of stimulants that can mask the impairing effects of alcohol on performance. If more than one drug is present in the body, the drugs may interact with each other either in a therapeutically beneficial way or in an adverse way. Some drugs can affect gastric emptying which has an effect on alcohol absorption. For instance, drugs that delay gastric emptying, such as caffeine (Siegers et al., 1972) and nicotine (Johnson, 1991), delay alcohol absorption, which results in lower than expected BACs. This is likely due to the drug being retained in the stomach longer, and subjected to increased metabolism by ADH.

Alternatively, drugs that increase gastric emptying, such as antibiotics (Edelbroek et al., 1993), aspirin (Roine et al., 1990), antihistamines (Palmer et al., 1991), ulcer medication (Caballaria et al., 1991), and heartburn remedies (Roine et al., 1990), increase alcohol absorption, resulting in higher than normal BACs. This is likely due to the drug being emptied from the stomach more quickly, and subjected to reduction in metabolism by ADH.

1.2.3.5. Gender and body size

Body size also has an effect upon alcohol distribution. Greater body weight provides a greater blood volume in which alcohol can be distributed (Kalant, 2000). This means a larger person will be less affected by a given amount of alcohol than a smaller person would be. Additionally, because fat is less vascularised than lean tissue, an increase in body fat results in a smaller blood volume. Therefore, a leaner person with a greater muscle mass (and less fat) provides a larger volume for alcohol to be distributed in

compared with a person who weighs the same but has a higher percentage of body fat (Kalant, 2000). The person with low body fat will be affected less than the person with a higher level of body fat.

Males tend to be larger than females and inherently have a higher ratio of muscle to fat. This in turn means that men have a proportionately greater vascular capacity because fat is less vascularised than muscle. Therefore, alcohol will be more diluted in the bloodstream of a man than in that of a woman (Frezza et al., 1990). In addition, because males and females generally differ in the distribution and proportion of muscle and fat in their bodies, the intensity and duration of alcohol effects may differ as a result. For example, female bodies have, on average, lower total water content (54% for females and 60% for males) and a higher total fat content (28% in females compared with 18% in males), and therefore, peak plasma levels of alcohol tend to be higher in women.

Frezza et al. (1990) reported that comparable levels of alcohol affect women more than men. Women have around half the gastric metabolism of alcohol than men due to lower levels of ADH. Since ADH metabolises around 15% of ingested alcohol, the BAC is increased by approximately 7% over that of males. Such differences occur even when alcohol consumption is comparable with respect to total body weight (Schenker & Speeg, 1990). Consequently, women may be more susceptible to the adverse effects of alcohol than men.

1.2.3.6. Genetic differences

It is now apparent that genetic variations may affect how people metabolise alcohol (Edenberg, 2007). The rate of ethanol absorption is partly determined by genetic factors (Kopun & Propping, 1977). Martin et al. (1985) calculated that 62% of the variability in peak BAC and 49% of the variability in elimination rate are genetically determined. Therefore, people with a family history of alcoholism may exhibit genetic differences in the response of their NMDA glutamate receptors as well as the ratios of $GABA_A$ in their brain (Gianoulakis et al., 1989), and opioid receptors (McBride et al., 1998). In addition, the rate of alcohol metabolism depends, in part, on the amount of ADH in the liver, which varies among individuals and appears to have genetic determinants (Benet et al., 1996).

1.2.3.7. Drinking history

Previous drinking histories can also an impact on the cognitive and behavioural effects of alcohol. The previous drinking history is influential in determining the effects of current alcohol consumption, as this can affect the amount of alcohol needed to produce a desired and/or expected effect of the drug. The reasons for this and the implication to the studies presented in this thesis are discussed below.

1.2.3.7.2. Pharmacodynamic tolerance

Receptors in the brain adapt to the continued presence of alcohol. Neurons adapt to the excess alcohol either by reducing the number of receptors available to alcohol or by reducing their sensitivity to alcohol. Long-term, or chronic, alcohol exposure can lead to adaptive changes within brain cells. For example, if alcohol exposure inhibits the function of a neurotransmitter receptor, the cells may attempt to compensate for

continuous inhibition by increasing the receptor numbers or by altering the molecular makeup of receptors or cell membranes so that alcohol no longer inhibits receptor function (Tabakoff et al., 1986).

1.2.3.7.3. Pharmacokinetic tolerance

Exposure to alcohol can change its rate of metabolism and alter its BAC levels. Alcohol induces greater synthesis of the P450 enzymes responsible for its metabolisation, thus enhancing the rate at which ethanol is metabolised (Ritchie, 1985). More enzymes are available to metabolise the alcohol and, as a result, more alcohol must be administered to maintain the same level of alcohol in the body.

1.2.3.7.4. Behavioural tolerance

Tolerance can develop when alcohol is administered in the context of usual pre-drug cues but not in the context of alternative cues. Poulos and Cappell (1991) proposed a homeostatic theory of drug tolerance. They found that testing in an environment in which tolerance had developed affected the manifestation of tolerance, and an environmental cue could maintain the tolerance. This effect has been called environment-dependent tolerance (Siegel, 1977). Such findings have an implication for the test environment that is used in alcohol studies, such that alcohol administered in rooms resembling a bar in appearance are more likely to perform tasks better (i.e. were more tolerant) compared with novel environments (McCusker & Brown, 1990).

1.2.3.7.3. Chronic exposure to alcohol

Although initial increased exposure to alcohol can enhance alcohol metabolism (Ritchie, 1985), in those individuals who use large doses of alcohol chronically and develop liver damage, the rate at which alcohol is metabolised can be dramatically reduced, which increases ethanol levels and prolongs its stay in the body. As a result, the efficiency with which the body metabolises alcohol is altered which affects the behavioural response to alcohol.

Chronic expose to alcohol also results in structural damage to the brain and resulting behavioural deficits, which can be observed in different ways. Results of autopsy show that patients with a history of chronic alcohol abuse have smaller and more shrunken brains than non-alcoholic adults of the same age and gender (Rosenbloom et al., 1995). Brain shrinking is especially extensive in the cortex of the frontal lobe (Pfefferbaum et al., 1997). Studies indicate that chronic alcohol use results in cognitive and motor deficits (Svanum & Schladenhauffen, 1986; Oscar-Berman, 1980). Stephens and Duka (2008) have presented evidence from animal and human studies for altered function of prefrontal cortex and amygdala as the result of repeated periods of alcohol exposure. Furthermore, a considerable amount of evidence suggests right-hemisphere functions (e.g. visual-spatial skills, visual-perceptual analysis) are more impaired than left-hemisphere functions (e.g. verbal-linguistic abilities) (Grilly, 1998).

In considering results of alcohol's effects, it is important to bear in mind the fact that, as mentioned in some published works (e.g. Buela-Casal, 1992), performance is a function not only of the blood-alcohol levels of participants, but also of the individual's tolerance

to alcohol. Therefore, the sample used in studies examining the effects of alcohol need to be selected carefully, such that participants have a moderate degree of tolerance to the alcohol dose used.

In summary, the above section demonstrates that there can be many individual differences in response to alcohol, each of which can be a factor in the behavioural effects of alcohol. These effects of alcohol are supported by measures of physiological arousal and psychomotor performance.

1.3. PHYSIOLOGICAL AND BEHAVIOURAL EFFECTS

This section reviews some of the existing literature that has examined the physiological, psychomotor and cognitive effects of alcohol. Although research has shown that alcohol has an effect on a variety of physiological processes, such as heart rate (Sher et al., 1994; Borg et al., 1990), and changes in skin conductance (Sher et al., 2007; Glautier & Drummond, 1994) and blood pressure (Minami et al., 2002), the following section will focus on the effects of alcohol on physiological arousal as this has a direct effect on the cognitive effects discussed in later sections. These studies provide information on the nature of alcohol's behavioural effects, and were used to guide the design of behavioural measures used throughout this thesis.

1.3.1. Physiological arousal

Arousal is the physiological and psychological state of being reactive to stimuli, and involves the activation of the reticular activating system in the brain stem, the autonomic nervous system and the endocrine system. Arousal is important in regulating

consciousness, attention, and information processing. Physiological data suggests that alcohol has a dose-related effect on levels of arousal (e.g. Glautier & Drummond, 1994; Finn & Pihl, 1987; Begleiter et al., 1984; Sher & Levenson, 1982). Such increases in arousal are observed at low doses of alcohol that are much lower than those commonly observed to cause behavioural impairment. In addition, alcohol has been associated with increased cortical activation in EEG studies (Kähkönen et al., 2003; Peterson et al., 1990). The Yerkes-Dodson Law (1908) states that there is a relationship between arousal and task performance, arguing that there is an optimal level of arousal for performance, and too little or too much arousal can adversely affect task performance. It has been proposed that different tasks may require different levels of arousal, therefore, in some circumstances alcohol may facilitate performance.

For simple tasks, the relationship can be considered linear with improvements in performance as arousal increases. For complex, unfamiliar, or difficult tasks, the relationship between arousal and performance becomes inverse, with a decline in performance as arousal increases. In support of this, alcohol is often found not to affect performance in situations with low arousal, such as pursuit tracking (Mangold et al., 1996), simple RT (Heishman et al., 1997), central flicker fusion (Azcona et al., 1995) when the demands of the task are simple. However, when the task demands are more complex, performance on these tasks shows impairment (Azcona et al., 1995; Fillmore & Vogel-Sprott, 1995; Hindmarch et al., 1992). Differing levels of arousal and task complexity may explain some of the variability in results following alcohol consumption.

1.3.2. Subjective effects

Alcohol related changes in mood have been reported extensively in the literature. For example, alcohol has been shown to increase excitement (Loke & Lim, 1992), pleasure (Lloyd & Rogers, 1997), relaxation (Gilman et al., 2008), and happiness (Gilman et al., 2008). It has also been reported to reduce tension (Roehrs et al., 1999; Vogel & Netter, 1989; Sher, 1985), vigour (Irwin et al., 2006) stress and anxiety (Gilman et al., 2008). Furthermore, some of these effects are observable not only through self-report measures, but also through physiological measures such as heart rate and skin conductance (Baum-Baicker, 1985).

A non-linear dose response relationship has been observed between alcohol and mood. Low doses of alcohol have been reported to improve subjective reports of positive mood (e.g., reduce tension and uncertainty) (Lloyd & Rogers, 1997). At higher doses, alcohol has been shown to produce negative reports of mood, such as increased anxiety and depression (Pohorecky, 1981). The negative effects can become evident at lower doses in those who have low mood before drinking (Boggan, 2003). Alternatively, positive mood can persist at higher doses in those who have positive moods prior to drinking (Boggan, 2003).

Understanding the relationship between alcohol and mood is important as emotions can affect the ability to perform certain tasks. The pre-test mood of individuals can affect performance on numerous tasks, with the general effects being that positive mood can have a beneficial effect on performance, whilst negative mood can hinder performance (Randall et al., 2004) due to increases in levels of anxiety, tension, and stress.

1.3.3. Psychomotor Performance

1.3.3.1. Simple (SRT) and Choice (CRT) Reaction Time

Reaction time (RT) tasks assess the ability to attend and respond to a target stimulus, with response time as the dependent variable. The SRT task typically comprises one target and one response (e.g. button press in response to a flash of light). The CRT task comprises multiple targets and/or responses (e.g. different button presses in responses to different targets), although the nature of the stimuli, responses and other features can differ widely across tasks.

Alcohol has been shown to increase RT in both SRT and CRT (e.g. Vermeeren & O'Hanlon, 1998; Jääskeläinen et al., 1996; Azcona et al., 1995). The impairing effects of alcohol on RT have been reported at various doses ranging from 0.2 g/kg (MacArthur & Sekuler, 1982) to 1 g/kg (Hindmarch et al., 1991), although these effects are generally less consistent at lower alcohol doses (Finnigan et al., 1995; Millar et al., 1992). Although these studies generally show impairment in performance, other studies have reported benefits. For example, McManus et al. (1983) reported an improvement in performance in a CRT following alcohol (0.7 g/kg). Improvements in performance are likely to be caused by behavioural stimulation due to an increase in arousal that results from the suppression of the inhibitory mechanisms (Martin & Siddle, 2003). As the alcohol dose increases, CNS depression increases and overcomes the disinhibition (Arif & Westermeyer, 1988) thus impairing and slowing cognition. RT has been shown to vary under different alcohol conditions depending on the criterion of speed or accuracy required by the particular task (Jennings et al., 1976).

At low doses of alcohol the effect is not consistent, although RT is generally slowed by the ingestion of alcohol indicating the slowing of mental processes (Liguori & Robinson, 2001), alcohol does not always produce impairment (Finnigan et al., 1995). Prior experience with alcohol of similar doses can produce tolerance effects, such that impairment would only be evident when the dose exceeds the level typically consumed by the participant (Hiltunen, 1997). Similarly, prior experience of impairment following similar doses of alcohol could also lead to compensatory effects. It is possible that participants attempt to compensate for their anticipated behavioural impairment by increasing levels of concentration and effort (Vogel-Sprott & Fillmore, 1999; Fillmore & Vogel-Sprott, 1995).

Reaction time responses involve a series of cognitive processes including attentional processes that identify the target stimulus, and psychomotor processes that underlie the motor response. However, it is often not possible to determine the extent to which alcohol affects the individual processes that underlie the response. The effects of alcohol on motor speed have been directly tested using tasks that isolate the motor response from cognitive processes that are involved in SRT and CRT tasks. For example, tests assessing body balance and body sway reveal an alcohol-induced deficit (e.g. Mattila et al., 1992; Lukas et al., 1989) that is unlikely to be due to impaired cognitive processes.

1.3.3.2. Further Measures of Psychomotor Performance

Alcohol has been shown to have some impairing effects on motor skills, whilst

apparently sparing others. Alcohol has been shown to decrease performance on tasks that require participants to maintain balance and tracking (e.g. Mangold et al., 1996), but not on basic motor skills such as finger tapping and hand steadiness (Lukas et al., 1989). Overall, alcohol-induced impairment on these tasks appears to be associated with the complexity of the psychomotor task (Hindmarch et al., 1991). Impairments are found on tasks that place greater demands on psychomotor functioning such as tracking and maintaining balance. Therefore, more demanding tasks show impaired performance following lower doses of alcohol, whereas simple tasks require greater doses of alcohol before impairment becomes evident.

Findings from other psychomotor tasks have also produced mixed results. The general finding on tracking tasks is that alcohol hinders performance. For example, relatively small BACs have impaired performance on adaptive tracking tasks (Cohen et al., 1987), but not pursuit tracking tasks with comparable BACs (Mangold et al., 1996). Adaptive tracking tasks require participants to match the movement of a stimulus with the task demands increasing in difficulty based on performance on the task. Whereas pursuit-tracking tasks requires participants to maintain their position in response to a moving stimulus held at a constant speed. The impairing effects of alcohol become more consistent following alcohol doses of around 0.6 g/kg, and these effects appear to be consistent across various types of tracking tasks. For instance, impairments have been found on adaptive tracking (Cohen et al., 1987), critical tracking (Vermeeren & O'Hanlon, 1998), pursuit rotor tracking (Fillmore & Vogel-Sprott, 1995) and compensatory tracking (Collins et al., 1987). This is not to say that all studies at these alcohol doses have produced such results, some still show null effects of alcohol on

tracking performance (e.g. Mangold et al., 1996; Hindmarch et al., 1992; Maylor et al., 1990).

Methodological differences, including task difficulty, alcohol dose, participant age and level of withdrawal, could explain the differing findings across studies. These findings suggest that alcohol's behavioural effects are mediated by several factors and that tasks that have been shown to be sensitive to the effects of alcohol in some circumstances may nevertheless be insensitive in other situations or consumer samples. Therefore, in addition to selecting tasks that are suitable for studying the effects of alcohol, consideration should be given also to factors such as task length, task difficulty, consumer status of participants, and alcohol dose, all of which may influence whether an effect of alcohol is observed.

1.4. ALCOHOL EFFECTS ON COGNITIVE PROCESSING

1.4.1. Information processing

The disruptive effects of alcohol on activities that require information processing have been well documented (Koelega, 1995; Carpenter, 1962; Goldberg, 1943). For instance, alcohol has been reported to impair a wide range of information processing tasks such as visual tracking (Maylor et al., 1990), visual scanning (Roehrs et al., 1994a; Hindmarch et al., 1992), rapid visual information processing (Fillmore et al., 2009), delayed and immediate pattern recognition (Kennedy et al., 1993), delayed recall (Weissenborn & Duka, 2000), digit symbol substitution (Mattila et al., 1992), visual spatial attention (Post et al., 1996), inhibition tasks (Rose & Duka, 2008), simple visual and auditory RT (Lemon et al., 1993), and vigilance (Bartl et al., 1996).

Some of the observed impairments on task performance following alcohol consumption have been attributed to limited availability of resources for processing information. The resource limitation account suggests that the central stage of information processing for the first task must be completed before processing of the central stage of the second task can begin. This creates a "bottleneck" when two tasks are to be performed in very close succession because the processing of the central stage of the second task must wait for the completion of the central stage of the first task. The prediction is that any manipulation, such as task difficulty that increases the duration of the central stage of information processing on the first task should carry over to increase RT on a second task that is performed immediately afterward. As the resources used to process task information are limited following alcohol consumption, the time needed to adequately process information is increased (Fillmore et al., 1998; Fillmore & Vogel-Sprott, 1997; Mitchell, 1985; Moskowitz et al., 1985).

Given that most complex tasks require some form of information processing, an important question is whether the impairments are due to global deficits in information processing or are more stage-specific. It has been suggested that behavioural impairments following alcohol consumption is due to a drug-induced slowing in the rate at which information can be processed (e.g. Fillmore & Van Selst, 2002; Maylor & Rabbitt, 1993; Moskowitz et al., 1985). As a result, any reduction in the capacity or rate of which information can be processed would reduce the efficiency of performance on attention-based tasks. The global disruptive effects of alcohol on information processing are based on the findings that alcohol affects overall performance on dual-task

performance or by conditions that require divided attention among multiple activities (e.g. Fillmore & Van Selst, 2002; Maylor & Rabbitt, 1987; Moskowitz et al., 1985).

On the other hand, it is possible that alcohol selectively impairs a certain stage of information processing, rather than resulting in global impairment. Information processing is assumed to involve at least three stages: (1) stimulus identification, (2) response selection, and (3) response execution. The first and last stages engage perceptual and motor processes, respectively, whereas the second stage involves cognitive processes such as decision making and planning. Schweizer et al. (2005) found that alcohol impaired only the central cognitive stage of information processing. It was found that when completing a dual-task, performance on the second task was increasingly impaired as the gap between performing the first and second task was decreased (Schweizer et al., 2005). The conclusion was that alcohol affected performance on the second task as a result of carry over effects from the first task. In order to successfully complete the second task, the central cognitive stage of processing must be completed before the processing of the second task can begin. At longer delays, no alcohol-related impairment was evident which goes against the global slowing hypothesis which would predict impairment at all delay periods.

In summary, the results of the Schweizer et al. (2005) study suggest that the global slowing hypothesis may not be able to fully account for the deficits observed in many alcohol studies. In which case, a detailed examination of the performance on complex tasks is required for a more comprehensive understanding of how alcohol affects

cognitive processes. The following sections review some of the effects of alcohol on complex tasks in more detail.

1.4.2. Executive Functions

Executive function (EF) is a broadly defined cognitive construct originally used to describe deficits associated with frontal lobe lesions. Executive functions are thought to be driven primarily by the prefrontal cortex (Duncan, 1996). The executive function domain includes the many skills required to prepare for and execute complex behaviour, including planning, behavioural control, and cognitive flexibility. The individual components of EF each have an impact on the ability of an individual to perform certain tasks and goals, and so the following section reviews some of the literature on the effects of alcohol on each component in turn.

1.4.2.1. Working memory

Working memory (WM) requires the ability to monitor and code incoming information for relevance to the task at hand, and then revise the items held in WM by replacing old, no longer relevant information with newer, more relevant information (Morris & Jones, 1990). Implicit in this description is the notion that information is actively manipulated, rather than passively stored (Miyake et al., 2000).

WM is considered to be an important component of executive function (Saults et al., 2007). Understanding alcohol-related effects are important as WM is thought to play an important role in most complex behaviours (e.g. Baddeley, 2001; Cowan, 2001), such as attention (Cowan, 2001) and inhibition (Finn et al., 1999). However, results from

working memory studies often produce differing results, with some showing alcohol-related impairment and others not (Paulus et al., 2006; Schweizer et al., 2006; Grattan-Miscio & Vogel-Sprott, 2005; Weissenborn & Duka, 2003). It is now thought that alcohol selectively impairs certain aspects of WM (Saults et al., 2007), with impairment revealed when stimuli are presented sequentially, but not when presented in array. The implication is that alcohol does not impair the ability to retain multiple items, but affects mnemonic strategies required to retain sequences (e.g. rehearsal: Baddeley et al., 1984).

1.4.2.2. Behavioural control

Behavioural control refers to the ability to deliberately inhibit dominant, automatic, or pre-potent responses when necessary. Alcohol is commonly associated with states of behavioural disinhibition or dyscontrol that are characterised by impulsive and extreme actions (Lyvers, 2000; Jellinek, 1952). Research has focused on the processes that regulate behavioural control, suggesting that impairment in this system underlies many of the behavioural deficits associated with alcohol (e.g. Abroms et al., 2006; Fillmore et al., 2000a, 2000b; Logan, 1994).

Assessments of behavioural inhibition mechanisms have been used to study the effects of alcohol on the ability to inhibit inappropriate behavioural responses (Fillmore, 2003). Several studies have examined alcohol effects using stop-signal and cued go/no-go models that assess behavioural control as the ability to quickly activate and suddenly inhibit pre-potent responses (Fillmore et al., 2005; Abroms et al., 2003; Marczinski & Fillmore, 2003a; Vogel-Sprott et al., 2001; de Wit et al., 2000; Fillmore & Vogel-Sprott, 1999; Mulvihill et al., 1997). The results from such studies indicate that

moderate doses of alcohol impair the ability to inhibit a pre-potent response, resulting in greater numbers of response errors and increased reaction times attributable to alcohol-induced impairment of inhibitory processes (e.g. Schweizer et al., 2006; Fillmore & Vogel-Sprott, 1999; Mulvihill et al., 1997). Together, the evidence suggests that the impulsivity and under-controlled behaviours associated with alcohol use could be due to an impairment of inhibitory control over pre-potent behavioural actions (Fillmore, 2003).

1.4.2.3. Planning

The planning component of EF requires the ability to successfully choose, evaluate and adopt alternative courses of action in order to perform tasks (Welsh et al., 1999). The relationship between the ability to plan behaviour and frontal lobe function is well established (Morris et al., 1993; Owen et al., 1990; Shallice, 1982).

Many of the tasks used to measure EF impose a set of rules that constrain the manner in which the task can be performed (e.g. the Tower of Hanoi, Simon, 1975; Tower of London, Shallice, 1982). Based on the structure, rules, and demands of the task, successful performance requires that a sequence of moves is planned, executed, monitored, and revised in advance of action. Studies have shown that alcohol can reduce the thinking time prior to initiating a solution (Weissenborn & Duka, 2003). Alcohol also increases the number of moves required to complete the problem and by increasing the time spent thinking about moves once a solution had been initiated (Weissenborn & Duka, 2003). Despite the dissociation of components of EF described by Miyake et al. (2000), it is possible that the initial time taken before initiating the

solution on these tasks is related to the impulsive behaviours often found following alcohol consumption (Weissenborn & Duka, 2003). Additionally, the tasks used to measure planning may place demands on spatial WM (Weissenborn & Duka, 2003), as possible solutions to the problem need to be held in memory and continually updated according to the task goal.

1.4.2.4. Set shifting

Set-shifting involves the ability to alternate back and forth between multiple tasks, operations, or mental sets (Monsell, 1996). Previous studies have shown that shifting mental sets incurs a measurable temporal cost (e.g. Rogers & Monsell, 1995), possibly due to the time taken to disengage from one task and the subsequent active engagement on another. Impairment in the ability to shift mental set has been ascribed to impaired function in dorsolateral prefrontal cortex (DLPFC) or to disruption of the fronto-striatal circuitry (Purcell et al., 1997).

The Wisconsin Card Sorting Test (Grant & Berg, 1948) and the Intra-Extra Dimensional Set Shift (IED) (Cambridge Neuropsychological Test Automated Battery, CANTAB) are typical tests used to measure set shifting ability. Using these types of test, studies have shown that alcohol also impairs set-shifting abilities (e.g. Saraswat et al., 2006). Furthermore, deficits on these tasks have been found primarily when the demands of the task active areas of the frontal lobe exclusively. For example, Dias et al. (1996) found that moderate doses of alcohol impaired performance only on the extradimensional shift and extradimensional reversal stage. The former stage has been shown to activate the dorsolateral prefrontal cortex, whereas the latter activates the

orbitofrontal cortex. Therefore, it appears that alcohol does not result in a global impairment of set shifting ability, rather only those requiring prefrontal cortical function are impaired. At the cognitive level, shifting within a category does not appear to be impaired, whereas shifting between categories is deficient.

Interestingly, the inability to shift mental set has also been attributed to a perseverative error, such that impaired behavioural control affects the ability to disengage from a previously beneficial strategy (Heaton et al., 1993). Similarly, Miyake et al. (2000) noted that the shifting ability may not be a simple reflection of the ability to engage and disengage appropriate task sets per se, but may also involve the ability to perform a new operation in the face of interference or negative priming. For example, when a new operation must be performed on a set of stimuli, it may be necessary to overcome interference or negative priming due to having previously performed a different operation on the same type of stimuli (Allport & Wylie, 2000).

1.4.2.5. Summary

The studies examining the effects of alcohol on EF clearly indicate that alcohol affects the ability to execute complex behaviours, such as planning, behavioural control, and cognitive flexibility. It is also apparent that the components of EF may not be unitary, and that the combined contribution of several components could contribute to deficits on complex tasks, such as WM and behavioural control. However, the underlying processes responsible for the deficits are still not known.

1.4.3. Memory encoding, storage and retrieval

The literature reporting the effects of alcohol on memory shows considerable variability. Some of this variability may be due to the different components of memory that are measured, with alcohol having a selective effect on some components and not others. The existing literature on the effect of alcohol on memory is vast, covering aspects of memory processes that are beyond the scope of the present thesis. Consequently, the following review of the effect of alcohol on memory will concentrate on those processes most likely to be implicated in disambiguating ambiguous figures.

Alcohol has shown mixed results about the ability to encode, store, and retrieve information. For example, episodic memory is particularly impaired by alcohol (Curran & Hildebrandt, 1999; Nilsson et al., 1989), semantic retrieval is somewhat less so (Wendt & Risberg, 2001), whereas priming is regularly unaffected (Duka et al., 2001; Fillmore et al., 2000a, 2000b; Nilsson et al., 1989; Hashtroudi et al., 1984). Also, differences in performance on memory tasks can be attributable to different patterns of drinking. For instance, Weissenborn & Duka (2003) have shown that binge drinkers are more impaired than non-binge drinkers. Furthermore, research has shown that moderate doses of alcohol can impair performance on episodic memory tasks (Curran & Hildebrandt, 1999) as well as memory encoding tasks more so than tasks that require the retrieval of information (Soderlund et al., 2005). Moreover, Parker et al. (1974) found that the ability to recall words decreased as the alcohol dose increased. These impairments have been attributed to alcohol-related deficits in the ability to encode new information (Mueller et al., 1983; Parker et al., 1976) as the ability to retrieve information from long-term memory does not appear to be impaired by similar doses of

alcohol (e.g. Soderlund et al., 2005). Additionally, Weissenborn & Duka (2000) have identified encoding impairments on the ascending and descending limbs of the alcohol curve. The tolerance-related improvement in performance usually found on the descending limb was not evident. It was suggested that the impairment at encoding was so strong that acute tolerance could not be developed. In support, Bennett et al. (1993) found improvement in performance on the descending limb occurs only when there are sufficient mental resources available due either to a low BAC or to a task that is relatively unaffected by alcohol (e.g. retrieval). Furthermore, the context in which alcohol is administered can have an effect on the ability to retrieve information. For instance, Weissenborn & Duka (2000) have shown that when the context in which alcohol is given is similar at both encoding and at retrieval, it can have a facilitative effect on retrieval.

The effects of alcohol on the ability to recall information have also produced differing results. For instance, some studies have failed to show an alcohol-induced impairment on retrieval (Weissenborn & Duka, 2000), digit recall (Baker et al., 1985) and letter recognition (Hindmarch et al., 1992). Whereas, Heishman et al. (1997) found a significant effect of alcohol on number recognition and word recall following alcohol doses above 0.5 g/kg, but not below this level. However, no effects of alcohol were observed on auditory short-term memory or the Sternberg test following administration of comparable levels of alcohol. Null effects on auditory short-term memory have also been reported by Millar et al. (1992) from doses of 20 mg/100ml to 80 mg/100ml. However, in an earlier study using the same task materials, Millar et al. (1987) reported significant impairments. It should be noted that these tasks differed in complexity, with

the later study being less complex due to less inter-list interference, which is a major source of forgetting (Postman & Underwood, 1973).

The implication is that any adverse effect of alcohol is dependent upon the precise nature of the task, with alcohol seemingly affecting performance on more complex tasks. Similarly, Lister et al. (1991) found that similar doses of alcohol impaired the ability to explicitly remember words, but did not impair memory for the same material when assessed implicitly. It is generally agreed that explicit recall is more complex than implicit recall (Graf & Schacter, 1987), further highlighting the fact that alcohol impairment might be related to the specific nature of the task. Furthermore, it has been shown that alcohol had a selective effect for tasks that presented material sequentially rather than in an array (Saults et al., 2007). This was taken to suggest that alcohol has little effect on the ability to retain multiple concurrent items, but had a more substantial effect on the rehearsal strategies that are needed to retain sequences.

It appears that alcohol does not produce global impairment of memory functioning. Rather, deficits are restricted to tasks that place greater demands on cognitive resources and are typically found following moderate to high doses of alcohol.

1.4.4. Attention

Many studies have reported that the ingestion of alcohol impairs attention (e.g. Moskowitz & Burns, 1990; Moskowitz & Sharma, 1974). However, research into the effects of alcohol on attention is complicated by evidence suggesting that attention is not a unitary construct (Rosselló et al., 1999; Posner & Petersen, 1990; Sack & Rice,

1974). The existing literature indicates that alcohol produces different effects depending upon the subtype of attention being measured, with some showing more impairment than others (Koelega, 1995; Holloway, 1995; Mitchell, 1985). Furthermore, many of these effects occur at doses similar to those consumed by regular social drinkers and within the legal limit of alcohol intoxication. It is therefore important to fully understand the nature of the impairments caused by alcohol on attentional processes. However, the basic cognitive and perceptual mechanisms by which alcohol disrupts performance on these tasks remains unclear and requires further examination. With these issues in mind, the aim of the following section is to review the effects of alcohol on the subtypes of attention.

1.4.4.1. Sustained attention and vigilance

Sustained attention refers to the maintenance of focused attention over extended periods (Sher et al., 2007), whereas vigilance requires prolonged attention to enable the responder to detect and respond to changing stimuli (Broadbent, 1971). It is well established that alcohol intoxication results in impairments on tasks that require continuous levels of performance, such as driving and piloting (Vermeeran & O'Hanlon, 1998; Wilkinson, 1995). These impairments are usually ascribed to a reduction in attention capacity (Linnoila et al., 1986).

Existing data on alcohol's effects on the ability to sustain attention have often produced differing outcomes. For instance, the overall level of performance following alcohol on sustained attention tasks has sometimes been shown to be unaffected (Jääskeläinen et al., 1995; Millar et al., 1992; Hindmarch et al., 1991, 1992; Gustafson, 1986a; Miles et

al., 1986; Vogel-Sprott, 1976). In these cases, it is generally concluded that sustained attention and vigilance tasks are insensitive to the effects of alcohol, leading to the assumption that alcohol's effects on such tasks are minimal (e.g. Ritchie, 1980). This idea is further supported by the findings that similar doses of alcohol impair performance on other types of attention, such as divided attention and selective attention (e.g. Fillmore et al., 2000a, 2000b; Finnigan et al., 1995). Other studies, however, have found evidence that alcohol does impair performance on these tasks (e.g. Sher et al., 2007; Jansen et al., 1985; Tong et al., 1980; Erwin et al., 1978). Furthermore, some studies (e.g. Maylor et al., 1987; Shillito et al., 1974) have indicated that low doses may slightly improve rather than impair vigilance.

Some of this variability may be accounted for by differences in the doses of alcohol that were used in these studies. Generally, studies where impairments were not found administer relatively small doses of less than 0.3 g/kg (e.g. Jääskeläinen et al., 1995; Hindmarch et al., 1991, 1992; Millar et al., 1992). Whereas, studies where impairments are found administer doses of around 0.6-0.8 g/kg (e.g. Vermeeren & O'Hanlon, 1998; Wilkinson, 1995). Studies looking at dose-related effects have generally concluded that much of the variability in the results is attributable to the difference in alcohol dose that is administered in these studies (Koelega, 1995; Rohrbaugh et al., 1988; Erwin et al., 1978).

Not all the variability in results can be attributed to differences in the doses of alcohol that are administered in these tasks. Although studies typically report deficits occurring following doses of around 0.5 g/kg, not all studies do so. The factor influencing whether

a deficit will be found seems to be related to the complexity of the task. Such that, simple tasks that place fewer demands on resources do not appear to result in impaired performance. For instance, some studies have shown that moderate doses of alcohol do not impair performance on Central Flicker Fusion (Hindmarch et al., 1992; Kuitunen et al., 1990) and choice RT (Jääskeläinen et al., 1995). Alternatively, tasks that are considered to be more complex and demanding do reveal alcohol-related impairments on performance. For example, studies that measure the effect of moderate doses of alcohol on driving (Vermeeren & O'Hanlon, 1998) and flight simulator performance (Taylor et al., 1996) consistently reveal deficits in performance. This variability in findings cannot be attributed to dose-related differences as they all used similar doses of around 0.5 – 0.8 g/kg.

1.4.4.2. Divided attention

Divided attention tasks require simultaneously attending to two or more activities at the same time, measuring the ability to distribute attentional resources. A great deal of research has been carried out with dual tasks for determining the effects of alcohol on the capacity to divide attention (e.g. Curtin et al., 2001; Schulte et al., 2001; Erblich & Earleywine, 1995; Moskowitz & Burns, 1990). Evidence suggests that divided attention tasks are highly sensitive to the effects of alcohol (e.g. Roehrs et al., 1994a; Moskowitz & Robinson, 1987; Landauer & Howat, 1983), and some believe these tests are the most sensitive to the effects of alcohol (Canto-Pereira et al., 2007; Moskowitz et al., 1985).

Deterioration in performance is seen when two tasks are performed together, than when they are performed separately (Schulte et al., 2001; Perry & Hodges, 1999). Impairment

has been found following alcohol doses of around 0.3 – 0.4 g/kg (Rosselló et al., 1999; Roehrs et al., 1994b), with consistent effects at doses of around 0.5 – 0.8 g/kg (Wilkinson, 1995; Lex et al., 1994). The effects found at a range of doses further suggest that these tests are sensitive to the effects of alcohol. Furthermore, the degree of impairment often increases as a function of the dose of alcohol administered in these tests (Wilkinson, 1995; Hindmarch et al., 1991).

Although a moderate dose of alcohol impairs performance on divided-attention tasks, performance is relatively unaffected on those tasks considered to be the most salient (i.e. the primary task) while performance on secondary tasks is greatly impaired (e.g. Schulte et al., 2001; Fisk & Scerbo, 1987). Similarly, studies in which participants are told to attend to stimuli in one modality while ignoring stimuli in a different modality (distracters) show that intoxicated participants perform somewhat better than sober participants (e.g. Erblich & Earleywine, 1995; Patel, 1988), indicating that moderate doses of alcohol actually may improve the ability to screen out irrelevant information.

According to the alcohol myopia model (e.g. Steele & Josephs, 1988, 1990; Steele & Southwick, 1985), in complex situations, especially when the primary task presents a high level of difficulty, intoxicated subjects will allocate their limited attentional resources to the most important stimulus or the primary task. As a result, the processing of secondary tasks or the effect of distracters will be greatly impaired. On the other hand, when the primary task is simple and low demanding, more resources will be available for processing distracters and its disruptive effect on the primary task will become more evident. Consequently, fewer resources will be available for secondary

task or stimuli, which are not related with the central task, resulting in performance impairment. Consistent with this hypothesis, much research has demonstrated that tasks requiring subjects to divide their attention across distinct spatial locations or to more than one task are severely impaired by acute alcohol intoxication (Schulte et al., 2001; Rosselló et al., 1999; Koelega, 1995). Moreover, intoxicated subjects seem to give priority to processing of primary or central tasks, even in a situation where the secondary task has emotional significance (Curtin et al., 2001; Curtin et al., 1998). These deficits may be related to the impaired ability to select certain aspects within a visual scene for processing.

1.4.4.3. Selective attention

The term selective attention refers to the processes that control or facilitate the detection of a target in the presence of extraneous information. It has long been claimed that selective attention and related aspects of visual information processing are impaired under the influence of alcohol (e.g., Fillmore et al., 2000a, 2000b; Tzambazis & Stough, 2000; Post et al., 1996).

The ability to detect and direct attention towards relevant information is important when it is presented with irrelevant and distracting information (Chun & Wolfe, 2001). Selective attention performance has been studied using visual search tasks in which the numbers of items in the display is varied, and the latencies and/or accuracies for the detection of targets are measured as a function of the number of items in the display (e.g. Duncan & Humphreys, 1989). Results from these studies show that RT increases and accuracy declines as the number of items in the display increases. However, this

effect is true only when targets and distracters share similar features and are relatively difficult to distinguish from each other. Latencies and accuracies are largely unaffected when targets and distracters have distinct features (Wolfe, 2003). It is also known that the efficiency of selective attention diminishes as the distance from centre fixation spreads across the visual field (e.g. Scialfa & Joffe, 1998; Wolfe et al., 1998).

Research has shown that there are larger alcohol-induced deficits in the spatial distribution of attention as a function of task complexity and processing demands (e.g., Post et al., 1996; Moskowitz & Sharma, 1974). To assess the effects of alcohol on attentional selection, Hoyer et al. (2007) conducted two experiments to examine the effects of two doses of alcohol on RT and accuracy for both simple and complex controlled target detection. It was found that alcohol impaired target detection in the more difficult search task, when targets were located more peripherally. This effect was shown following administration of 0.7 g/kg of alcohol, but not at doses below 0.5 g/kg. Similarly, Canto-Pereira et al. (2007) found that alcohol resulted in attention being maintained near the point of gaze, causing impairment in orienting attention to peripheral regions. It is possible that alcohol might impair the ability to direct attention to relevant task stimuli in the first place (e.g., Fillmore et al., 2000a, 2000b; Houghton & Tipper, 1994). However, Hoyer et al. (2007) found no group differences in the ability to perform the easier search task at either a low or high dose.

It is possible that impairment in selective attention is due to the inability to select stimuli located at peripheral locations, and so the focus of attention remains in more central locations. However, studies have found no evidence to support this claim (Post

et al., 1996; Moskowitz & Sharma, 1974), suggesting that alcohol does not reduce the size of the visual field resulting in impaired performance at the periphery. For instance, Moskowitz and Sharma (1974) found that impaired ability to detect targets at the periphery were associated with the processing demands of the task. More complex tasks resulted in impaired ability to detect targets at the periphery, whereas performance was not affected on simple tasks. Similarly, Post et al. (1996) found that alcohol does not affect the ability to extract information from peripheral locations, but that alcohol seemed to interfere with the ability to shift the focus of attention from one target location to another. Furthermore, alcohol impaired performance under conditions that placed greater demands on the controlled aspects of visual spatial attention.

However, these findings raise further issues about how attention might be affected by alcohol. For instance, whether or not alcohol simply reduces the amount of information that can be processed at any one time or whether it affects the allocation of attention. These effects can be isolated using a procedure similar to that used by Kinchla et al. (1983). In their study, attention was biased towards the "global" aspects of stimuli, or to the "local" elements. The results show that as the probability of a target appearing at a given level increased, target detection at that level improved and target detection at the other level declined. It was concluded that when more capacity is allotted to one level, less is available for processing the other level. Within the Kinchla et al. (1983) paradigm, attentional allocation would be measured as relative performance at the local and global levels while attentional capacity would be measured as overall performance collapsed over these levels. Thus, this type of paradigm is especially useful because it

makes it possible to distinguish effects of alcohol on two separate aspects of attention, attentional capacity and attentional allocation.

Lamb and Robertson (1987) suggest that alcohol does not affect overall attentional capacity, but affects either how attentional capacity is allocated or the global processing mechanism itself. In their experiment, placebo participants were better at detecting letters at the global than the local level in the Global bias conditions, better at local than global in the Local biased conditions, and roughly equally good at both levels in the Neutral biased conditions (Lamb & Robertson, 1987). Alcohol had no effect on detection of letters in the local levels in any of the bias conditions but reduced detection of letters at the global level except under Global biased conditions. The question remains as to why performance at the global level deteriorated for alcohol subjects in the Neutral and Local Bias conditions. One possibility is that alcohol affects the mechanism that processes global information, but that the resulting deficit can be overcome by focused attention.

1.4.4.4. Inhibitory mechanisms and Attention

Recently, research has focused on the inhibitory mechanisms involved in the control of visual attention (Abroms et al., 2006). For instance, behavioural control contributes to selective attention by guiding cognitive resources towards relevant stimuli and away from irrelevant stimuli (Fox, 1995; May et al., 1995; Houghton & Tipper, 1994). The following sections review some of the literature examining the contribution of inhibitory mechanisms to attentional processing.

1.4.4.4.1. Negative priming

Recently, research has sought to identify processes that allow attention to be allocated to relevant stimuli in the presence of irrelevant information. The Stroop colour-naming test (Stroop, 1935) has commonly been used to study selective attention. The classic Stroop phenomenon involves presenting either colour words or control stimuli in different colours. When individuals are required to name the physical colour of the stimuli and ignore the carrier stimulus (colour words or Xs), their RTs are slower when the physical colours are presented on incongruent colour words than when presented on control stimuli.

Results from studies using the Stroop test have led to theories about the processes that underlie selective attention. One theory proposes that selective attention may operate by an inhibitory process that prevents the disruptive influence of irrelevant, distracter stimuli (Houghton & Tipper, 1994). This theory states that irrelevant information is internalised as neural representations associated with an active inhibitory process. The active suppression of irrelevant information by the inhibitory mechanism allows relevant stimuli to be processed free from distraction. Empirical support for an inhibitory process in selective attention has been obtained using a "negative priming" paradigm (e.g. Tipper, 1985). This paradigm uses a variation of the Stroop naming task in a repetition priming procedure (e.g. Neill, 1977). Priming is said to occur when responding to a "prime" stimulus affects the RT to a subsequent "probe" stimulus. Negative priming refers to the particular case in which responding to a prime slows the subsequent response to a probe. This tends to occur when the correct response on the probe trial was the response that had to be suppressed on the prime trial. This

combination of prime and probe trials leads to longer RTs than a combination using the identical prime, followed by an unrelated probe, in which neither the target nor the distracter appeared in the prime.

An inhibition-based explanation for negative priming suggests that the irrelevant distracter feature of the prime stimulus is internalised as a neural representation associated with an active inhibitory "gating" process. This gating process allows relevant features of the prime to be processed free from distraction. Because the internal representation of the ignored distracter feature is actively inhibited when responding to a prime, it takes longer to respond to a subsequent stimulus that features this same inhibited representation. The RT difference between negatively primed and unprimed probes is the negative priming effect (Houghton & Tipper, 1994).

Fillmore et al. (2000a) have shown that a moderate dose of alcohol (0.56 g/kg) suppresses negative priming. One interpretation of these findings is that alcohol prevented the distracter attribute of prime stimuli from being inhibited. This is because no reaction time cost was evident on probe trials in which distracter responses subsequently became target responses. Evidence that alcohol can suppress the negative priming effect is important because the effect is considered to reflect a basic cognitive process that allows attention to be selected away from irrelevant information (Houghton & Tipper, 1994).

Furthermore, the effect cannot be attributed to alcohol-induced impairment of memory processes. Fillmore et al. (2000b) showed that alcohol reduced the negative priming

effect at moderate doses of alcohol and also showed that the facilitating effect of positively primed probe responses was unaffected by alcohol (presenting identical prime-probe pairs). Positive priming was evident in both groups at baseline and remained unaltered during tests under alcohol and placebo. The differential effect of alcohol on negative and positive priming provides some important evidence about the specific mechanism by which alcohol might operate to suppress negative priming. Evidence for a selective effect of alcohol on negative, but not positive, priming suggests that the drug does not simply reduce negative priming by some general impairment of encoding or retrieval of prime stimuli. This suggests that there is some specific stimulus and/or response characteristic in the negative priming condition that makes it particularly sensitive to the effects of alcohol.

1.4.4.4.2. Visual search

Visual search tasks are further attentional processes that might be enhanced by inhibitory mechanisms (Klein, 1988, 2000; Klein & MacInnes, 1999). When scanning a visual environment, attention is directed over different locations until a target stimulus is detected. Once attention has been directed away from a location for a sufficient period, the time required for attention to return to that location actually increases relative to the time required to direct attention to a new, previously unattended location. The delay to return attention back to a previously attended location is taken to reflect the operation of an inhibitory effect on the search process (Klein, 2000). This finding is referred to as inhibition of return (IOR) (Posner & Cohen, 1984). IOR enhances the efficiency of information gathering by biasing attention toward new information in

unexplored locations and away from old information contained in previously searched locations (Klein, 1988).

IOR tasks present a visual cue in a peripheral location and following the cue, a target stimulus is presented at either the same or a different location. Participants are instructed to detect the target as quickly as possible. IOR is evident on trials in which the target appears at the same location as the cue. On these trials, IOR is demonstrated by a longer target detection time compared with trials in which the target and cue appear at different locations. Target detection time also depends on the delay interval between the cue and the target presentation (Samual & Kat, 2003; Klein, 2000; Posner & Cohen, 1984). Research shows that IOR requires a cue-target SOA of at least 300 ms, at brief cue-target SOAs, target detection times are shorter on same-cue-target trials than control trials. This effect is the result of the brief SOA not allowing time for attention to initially leave the cued location before the target is presented (Lupianez et al., 2001). Therefore, detection times are shorter at these SOAs because attention remains at the same location throughout the trial (Briand et al., 2000).

Based on research that has shown that alcohol reduces inhibitory influences on selective attention (e.g. Fillmore et al., 2000a, 2000b), it has been suggested that alcohol would also impair inhibitory mechanisms involved in visual search and thereby reduce the extent of the IOR effect observed. Abroms and Fillmore (2004) reported dose-dependent slowing effect on target detection time following moderate doses of alcohol, reflecting alcohol-induced diminished IOR. It was concluded that IOR was diminished under alcohol because the drug reduced the inhibitory influences on target detection. It

is argued that the time delay normally observed in this condition is due to the operation of an inhibitory influence on the visual search process that delays the return of attention to previously attended locations (Klein, 2000). The reduced detection delay under alcohol could implicate some impairment of this inhibitory process. Furthermore, the tendency to acquire redundant visual information could contribute to alcohol-related slowing effects on reported elsewhere in the literature (e.g. information processing).

Although the effect reported by Abroms and Fillmore (2004) was in the order of millisecond changes, it was suggested that subtle disturbances at these basic levels of attention could have a substantial impact on higher order cognitive and behavioural functions. Many fundamental cognitive and perceptual processes, such as inhibitory influences, are considered to operate in a bottom-up fashion to exert increasing influence at each stage of higher order attentional and cognitive functions (e.g. Barkley, 1997; McClelland & Rumelhart, 1981). Therefore, although alcohol might produce slight disruption on attention-based mechanisms, the disturbance might exert considerable influence on the higher order behavioural functions that rely on those mechanisms.

Even though recent studies on alcohol and attention highlight the importance of inhibitory mechanisms, there remain many questions concerning the specific nature of the inhibitory mechanisms that may be impaired by alcohol. The variability in the level of impairment observed in these tasks may be due to the amount of control an observer has on their behaviour.

1.4.4.4.3. Automatic versus controlled attention

A primary distinction among control mechanisms concerns whether the mechanism is intentionally controlled or is automatic (Marzi, 1999; Shimojo et al., 1999). Intentional control mechanisms are those that are under the volitional control of the individual, and operate at the level of awareness. By contrast, automatic inhibitory mechanisms occur without intention in a reflexive manner caused by the presence of irrelevant stimuli.

According to Fisk and Schneider (1981), some of the differences in the literature can be interpreted in terms of whether automatic or intentional control mechanisms are responsible for processing the task information. Fisk and Schneider (1981) demonstrated that many of the impairments found in the literature reflect impairments in the intentional control mechanisms responsible for processing information, whereas, automatic control mechanisms are relatively unaffected by alcohol. Automatic processes are relatively effortless, whereas intentional mechanisms are regarded to be effortful processes (Schneider & Shiffrin, 1977), suggesting that more complex tasks may result in greater impairment in performance.

These differences between the two control mechanisms are also supported by more recent work in this area (Abroms et al., 2006; Fillmore & Vogel-Sprott, 2006; Holloway, 1995). For example, Abroms et al. (2006) measured the ability to inhibit eye movements for automatic and controlled mechanisms separately. The results showed that moderate doses of alcohol impaired the intentional inhibitory mechanism, but not the automatic mechanism. However, one limitation that concerns most measures of visual attention that rely on eye movements is that the processes are not perfectly

correlated. Although eye movements have been taken to indicate shifts of visual attention (e.g. Godijn & Theeuwes, 2003; Hyona et al., 2003; Posner, 1980), there are conditions under which the eyes may be directed to one location while attention is directed to a different location.

Furthermore, the distinction between the two control mechanisms remains somewhat problematic as some previous reviews of alcohol's effect on attention have indicated that both control mechanisms can be impaired under moderate doses of alcohol (Holloway, 1995). Also, some studies have found that neither control mechanism is impaired following moderate doses of alcohol (Vorstius et al., 2008). Although, this study may be subject to the same problems as the Abroms et al. (2006) study as both used eye movements as the dependant measure.

Although a description of the decrement in terms of automatic and intentional processes seems to provide an explanation, it remains rather vague. A clear understanding of how these attentional mechanisms work, and which of the underlying processes are assumed to deteriorate is needed.

1.4.4.5. Summary

Although recent studies on alcohol and attention highlight the importance of inhibitory mechanisms, there remain many questions concerning the specific nature of the inhibitory mechanisms impaired by alcohol. The ability to efficiently extract and process information from complex visual displays is critical to performance on everyday tasks. Any alcohol-related reduction of inhibitory influences that normally

guide attention in these situations could represent a basic mechanism by which the drug impairs these skills. A novel way to assess the inhibitory processes involved in attention could involve the use of ambiguous figures. Fluctuations in the interpretation of these figures have been suggested to provide insights into underlying attentional mechanisms involved. For an ambiguous figure to alternate in appearance, the dominant percept must be inhibited for the alternate interpretation to then dominate. Also, manipulating the figures through biasing can affect which interpretation attention is initially directed to, thus determining which of the interpretations receive dominance in attention. The following sections review some of the existing literature on ambiguous figures, and highlight the valuable contribution in examining the role of inhibitory mechanisms in attentional processes.

1.5. PERCEPTUAL AMBIGUITY AND PERCEPTION

Ambiguous figures have played an important role in understanding attention because their multistability is thought to reveal critical sensory, motor, physiological, and cognitive processes (Long & Toppino, 2004). When viewing ambiguous figures, there is insufficient information in the retinal array to produce a single stable percept, and therefore, our perception of the image fluctuates between each of the possible interpretations. The following sections examine some of the suggested causes of perceptual fluctuations when viewing ambiguous figures.

1.5.1. The role of attention in figure reversals.

It has been suggested that figure reversals are the result of fluctuations of visual attention (Helmholtz, 1962). Leopold and Logothetis (1999) argued that spontaneous

and involuntary shifts of visual attention prompt figure reversals. More recently, several psychophysical studies have shown that phenomenal alternations can be influenced by controlled (van Ee et al., 2005; Meng & Tong, 2004; Toppino, 2003) as well as automatic attention (Chong et al., 2005; Mitchell et al., 2004). Moreover, functional imaging (Sterzer et al., 2002; Inui et al., 2000; Kleinschmidt et al., 1998; Lumer et al., 1998) and electro-physiological studies (Strüber et al., 2000) have shown that phenomenal reversals are associated with transient activations of right frontoparietal cortex, an area associated with attentional guidance and control (Nobre et al., 1997; Desimone & Duncan, 1995). Also, patients with lesions in this area have difficulties experiencing multiple aspects of complex ambiguous drawings (Meenan & Miller, 1994) and exhibit diminished voluntary control over phenomenal alternations (Windmann et al., 2006).

Attentional selection is known to enhance neural responses to the attended stimulus in the visual cortex (Reynolds & Chelazzi, 2004). Consequently, attention could initiate reversals either by shifting to the currently suppressed aspect of the ambiguous figure, boosting the associated neural activity, or by drawing away from the currently dominant aspect lowering the associated activity, or a combination of the two. Alternatively, a reversal itself could result in an automatic shift in attention to the ambiguous figure and the new interpretation of the figure. It has been documented that a sudden image change can trigger an automatic shift of attention to the changed location (e.g. Prinzmetal et al., 2005; Posner, 1980).

Consistent with this possibility, shifts of attention have been found to influence dominance durations (Meng & Tong, 2004; Mitchell et al., 2004) and to alter reversal rates (van Ee et al., 2005). The finding that reversals can be brought under volitional control suggests that cognitive resources can influence attentional mechanisms involved in figure reversals. Additionally, results showing that reversals can be brought about through automatic shifts in attention such as a sudden change in the image suggest the involvement of sensory-level resources in figure reversals. Inhibitory mechanisms appear important in the reversal process, regardless of whether reversals themselves are controlled or brought about by automatic processes (Meng & Tong, 2004). For instance, only one interpretation of an ambiguous figure can be dominant in awareness at a given time and so the alternate interpretation must be suppressed. Consequently, the importance of attentional resources in figure reversals appears to be in facilitating target selection (selection of the dominant interpretation) versus inhibiting distracters (suppression of alternate interpretation). Therefore, this alternation of dominance and suppression of figure reversals could be used to assess different types of inhibitory processes that could underlie the effects of alcohol on cognition. The following sections review some of the literature favouring both cognitive and sensory-level processes involved in the assumed facilitation and inhibition process.

1.5.1.1. Evidence for cognitive processes involved in figure reversals

Evidence in favour of cognitive processes in figure reversals highlights the importance of intention and active processes to bring about reversals (Long & Toppino, 2004). As mentioned previously, reversals can be brought under volitional control (Strüber & Stadler, 1999). The observation that observers can maintain a particular interpretation

for some time without the alternate interpretation gaining dominance (e.g. Strüber & Stadler, 1999; Peterson & Gibson, 1991; Liebert & Burk, 1985) suggests that cognitive mechanisms such as inhibition might be an important factor by suppressing the unwanted percept (Meng & Tong, 2004).

The prefrontal cortex seems to be responsible for holding information and protecting this information against distracting input, which is a prerequisite for behaviour planning and goal-directed behaviour (e.g. Curtis & D'Esposito, 2003; Sakai et al., 2002; D'Esposito et al., 2000; Petrides, 2000). These functions may help to maintain and stabilise the dominant view of an ambiguous figure and to suppress competing representations, thereby reducing the reversal rate. It is well documented that alcohol can affect the prefrontal cortex (e.g. Medina et al., 2008; Kähkönen et al., 2003), as well as planning and goal direct behaviour (e.g. Weissenborn & Duka, 2003). Moderate doses of alcohol have also been associated with impulsivity and impaired inhibition (e.g. Abroms et al., 2006; Lyvers, 2000; Mulvihill et al., 1997). This suggests that if inhibitory mechanisms are implicated in figure reversals, then alcohol could impair performance.

The effects of expectancy, or set effects, are also well documented. For example, it has been shown that prior presentation of an unambiguous version of a traditional ambiguous figure reduces the ambiguity of the ambiguous figure (Long et al., 1992). Under such conditions, observers' typically report the interpretation of the ambiguous figure to be in the same configuration as the unambiguous figure presented beforehand. (Bernstein & Cooper, 1997; Long et al., 1992; McBeath et al., 1992; Fisher, 1967;

Botwinick, 1961; Bugelski & Alampay, 1961). Similarly, the context in which an ambiguous figure is presented can also affect how it is perceived. For instance, the figure *13* could be seen as the letter B or the numbers 1 and 3, depending on whether it was presented within other letters or other numbers (Bruner & Minturn, 1955). Similarly, prior presentation of pictures in the same category as one or the other interpretation of the ambiguous figure prior to viewing the ambiguous figure similarly biased the observer to report the primed interpretation of the ambiguous figure (Bugelski & Alampay, 1961).

In addition, the introduction of secondary tasks also affects the number of reversals that are reported. Reisberg (1983) and Reisberg and O'Shaughnessy (1984) found the introduction of a secondary task to increase the time until the first reversal of a reversible figure is reported as well as to decrease the rate with which subsequent reversals occur. They concluded that figural reversals required "perceptual judgments" that competed with the secondary task for working memory. Studies looking at the effects of alcohol on dual task performance have concluded that alcohol strongly impairs the ability to concurrently divide attention between two tasks (e.g. Curtin et al., 2001; Schulte et al., 2001; Erblich & Earleywine, 1995). The evidence suggests that divided attention tasks are highly sensitive to the effects of alcohol, therefore simultaneously performing another task whilst reporting figure reversals is likely to reveal alcohol-induced impairment.

Additional evidence in support of cognitive processes comes from studies using brain-injured patients. Research has shown that ambiguous figures activate the parietal and

prefrontal regions (Tong et al., 2002), and that multistablity is a function of the modification of activity in the visual area by association areas, specifically that perception of ambiguous figures involves a top-down feedback system from fronto-parietal areas to visual areas (Leopold & Logothetis, 1999; Blake, 1989). It has been found that patients with unilateral frontal damage are impaired on perceptual reversals (Cohen, 1959; Petrie, 1952). Teuber (1964) compared the effects of frontal and non-frontal lesions on the ability to reverse perspective of an ambiguous figure, finding that unilateral frontal lesions produced more impairment than posterior lesions. Although, no study has looked at the effects of alcohol on figure reversals, it is well documented that alcohol affects the frontal lobes (e.g. Kubota et al., 2001; Moselhy et al., 2001).

Ricci and Blundo (1990) also found that the ability to shift perception from one aspect of ambiguous figures to another is significantly impaired in those with lesions in frontal regions. Failure on such tasks was highly intercorrelated to performance on the Wisconsin Card Sorting Task (WCST), a test known for its sensitivity to frontal lobe damage (Miller & Cohen, 2001). This suggests an underlying perservative tendency seen in patients with frontal damage. Alcohol is also known to impair performance on set-shifting tasks (Lyvers & Malzman, 1991), indicating that moderate doses of alcohol might impair the ability to shift perception between the two interpretations.

Strüber and Stadler (1999) hypothesised that cognitive processes would act more effectively on semantically meaningful ambiguous figures (e.g. Duck-Rabbit illusion) rather than less semantically meaningful images (e.g. Necker cube illusion). Cognitive mechanisms were activated by the instruction to bring reversal rate under volitional

control. It was found that reversals of semantically meaningful figures could be controlled voluntarily to a much higher degree than for less semantically meaningful figures. It was concluded these results provided strong support for a distinct role of cognitive mechanisms for the processing of different types of ambiguous figures.

Taken together, the research cited above further highlights the involvement of cognitive processes in terms of the selective attention and inhibitory processes involved in figure reversals. Although there is evidence to strongly suggest that the attentional processes involved in figure reversals are modulated by cognitive mechanisms, there is also evidence to suggest that sensory-level factors might also play an important role.

1.5.1.2. Evidence for sensory-level processes involved in figure reversals

Evidence in favour of sensory-level processes in figure reversals highlights the importance of automatic processes outside of awareness and volitional control (Blake, 1989; Köhler & Wallach, 1944).

Early accounts of figure reversals claimed that the perceived interpretation depended upon the set of features within the ambiguous figure that were initially attended to and so received primary processing (Necker, 1832). Within this view, eye movements were thought to be critical because the foveated (the point of distinct vision of the retina) portion of the figure was believed to be in the foreground. In support, there is experimental work demonstrating that eye movements and eye position are related to figure reversals (e.g. Toppino, 2003; Ruggieri & Fernandez, 1994; Ellis & Stark, 1978). Several other studies have shown that fixation at different locations of a figure tend to

favour one or the other interpretation (e.g. Pomplun et al., 1996; Garcia-Perez, 1992; Peterson & Gibson, 1991; Hochberg & Peterson, 1987; Tsal & Kolbert, 1985). However, eye movements are not always necessary for a reversal to occur (Gale & Findlay, 1983). Although a strong positive correlation between eye movements and perceptual reversals has been found (van Dam & van Ee, 2006), it is not known whether it is the change of eye movement that results in a reversal or whether it is the reversal itself that causes a change in gaze. Most recent studies have shown that observers are still able to report figure reversals even when eye movement is controlled (Kornmeier et al., 2009; Kornmeier et al., 2007). The finding that reversals still occur even though eye movements are restricted strongly suggests that eye movements may not be a causal factor, or at least the only factor for reversals to occur.

Automatic shifts of attention have been mostly investigated using location-cueing tasks, in which a spatial cue presented in advance of the target directs the participant's attention to a location while fixation remains steady at another location (Posner, 1980). Although the impact of alcohol on automatic shifts of attention has received little attention, there is existing research to suggest that alcohol would impair performance on this aspect of attention. The allocation of spatial attention is controlled by parietal brain structures (Cohen et al., 1994; Rafal & Posner 1987; Posner & Cohen, 1984). In an fMRI study, Levin et al. (1998) showed that parietal brain structures became less activated following visual stimulation by a diffuse flash following alcohol. One of the few tests that have examined the effects of alcohol on automatic shifts in attention has shown that a moderate dose of alcohol (0.6 g/kg) impaired the ability to automatically shift attention (Schulte et al., 2001).

In addition, the number of reversals increases over time (Köhler, 1940), and if the figure is moved to a new location so that the image fell on different retinal regions caused the pattern of reversals to revert to the baseline level of reversals (Toppino & Long, 1987; Spitz & Lipman, 1962; Howard, 1961). In a closely related vein, Toppino and Long (1987) also demonstrated that adaptation to a figure of one size and then viewing a cube of a different size shows no carryover from the adaptation phase to the test phase. To many researchers, these demonstrations are especially strong evidence for the involvement of relatively localised processes.

Furthermore, the attributes of the figure itself can have an impact on the number of reversals that are reported. Such that more intense figures reverse more rapidly than less intense ones (Cipywynk, 1959; Lynn, 1961); complete figures reverse more rapidly than incomplete ones (Babich & Standing, 1981; Cornwell, 1976); and continuous viewing produces more reversals that intermittent viewing (Leopold et al., 2002). Each of these manipulations has been interpreted to affect relatively early cortical structures which analyse stimulus features and in which slowly building adaptation effects critically dependent on these stimulus characteristics are believed to occur.

Areas within the ambiguous figure that are attended to can also determine how the figure is interpreted. For example, Tsal and Kolbet (1994) identified “focal areas” in ambiguous figures that determined which perceptual interpretation was reported depending on where attention was directed. It was reported that if a letter had been processed in one of these areas just prior to the presentation of the ambiguous figure,

the interpretation associated with that focal area tended to be the one reported. In a similar study, Peterson and Gibson (1991, 1994) found that a region of the Rubin face/vase figure was perceived as a figure for longer when it was the fixated than when it was the non-fixated region. Similarly, Chastain and Burnham (1975) showed that the order in which individual features of the rat/man figure appeared governed its perception.

Furthermore, Georgiades and Harris (1997) found that the manipulation of individual features could strongly bias the perception of an ambiguous figure. Therefore, if the perception of ambiguous figures is determined by attention to these localised features, then the position of a fixation point within the figure should be important in determining perception. It was found that, in general, the dominance of the percepts varied with the relationship between critical features and the fixation point (Georgiades & Harris, 1997). When the fixation point was close to a critical feature, the tendency was for the figures to be less ambiguous.

General conclusions to be drawn from these studies is that automatic shifts of attention towards localised features is one determinant of the perception of ambiguous figures, and that manipulating the features within an ambiguous figure can affect its ambiguity and therefore the reversibility of the figure (see Table 1.1 for a summary of the sensory-level and cognitive processes associate with figure reversals). Furthermore, alcohol has been shown to impair performance on other types of tasks measuring automatic shifts in attention suggesting that similar impairment might also be revealed when reporting figure reversals.

Table 1.1. Summary of the sensory-level and cognitive processes associate with figure reversals

	Evidence	**Existing studies**
Cognitive processes	Volitional effects (e.g. consciously increasing, decreasing, or stabilising reversal rate)	Strüber & Stadler, 1999; Peterson & Gibson, 1991; Liebert & Burk, 1985
	Expectancy effects (e.g. positive bias, priming)	Bernstein & Cooper, 1997; Long et al., 1992; McBeath et al., 1992; Fisher, 1967; Botwinick, 1961; Bugelski & Alampay, 1961; Bruner & Minturn, 1955
	Cognitive load (e.g. dual-tasks)	Reisberg, 1983; Reisberg & O'Shaughnessy, 1984
Sensory-level processes	Increasing reversal rate over time (e.g. neuronal activation and fatigue)	Köhler, 1940
	Figure localisation effects on reversal rate (e.g. changing figure location)	Toppino & Long, 1987; Spitz & Lipman, 1962; Howard, 1961
	Adaptation effects (e.g. changing figure size)	Toppino & Long, 1987
	Intensity/luminance (e.g. altering the brightness or contrast of figures)	Cipywynk, 1959; Lynn, 1961
	Figural completeness (e.g. incomplete figure result in reduced reversals)	Babich & Standing, 1981; Cornwell, 1976
	Continuity of presentation (e.g. presenting figures in an on/off regime)	Leopold et al., 2002
	Eye movements/position (e.g. reversal rates following changes in eye movements)	Toppino, 2003; Ruggieri & Fernandez, 1994; Ellis & Stark, 1978
	Fixation location (e.g. focused attention on certain elements of the figure)	Pomplun et al., 1996; Garcia-Perez, 1992; Peterson & Gibson, 1991, 1994; Hochberg & Peterson, 1987; Tsal & Kolbert, 1985, 1994; Chastain & Burnham, 1975; Georgiades & Harris, 1997

1.5.1.3. Summary

Whilst much of the ambiguous figures literature has focussed either upon the cognitive factors influencing figure reversals, or the sensory-level factors that do so, the current opinion is that both processes can influence figure reversals depending upon the viewing conditions (Long & Toppino, 2004; Hochberg & Peterson, 1987). A good example of this comes from a study by Long et al. (1992) involving the effect of prior presentation of an unambiguous version of the typical reversible figure prior to the viewing of the ambiguous figure. Whilst demonstrating that brief presentation (e.g., less than 5 sec) of the unambiguous version produced a positive-bias effect favouring the same interpretation to the subsequently viewed ambiguous figure, they also demonstrated that longer presentations produced the opposite effect. As the duration of the adaptation period increased (e.g. 2–3 min), a reverse-bias effect was found. Thus, the effect of prior exposure to an unambiguous version of a figure depends on its duration, with short and long exposure periods seeming to affect perception via cognitive processes (i.e. set or expectancy) and sensory processes (i.e., neural adaptation), respectively.

Such a combined role hypothesis explicitly recognises the role of both sensory and cognitive process. And so varying the conditions under which figure reversals are reported can affect the degree of involvement from cognitive and sensory-level processes. The demonstration of the moderating influence of variables that favour either cognitive or sensory-level effects in figural reversal, depending on the particular viewing conditions can be used to determine how alcohol may affect these processes. Through identifying conditions where alcohol exerts impairment in performance as well

as conditions where impairment is not evident, this thesis provides evidence for the involvement of multiple processes in the perception of reversible figures and identifies those conditions whereby alcohol impairs performance.

1.6. Purpose of the present thesis

The interplay between alcohol, attention, and inhibition in the consequences of cognitive performance is at the centre of several problems that are addressed by this Thesis and will cover the following:

- Firstly, to address the specific nature of the alcohol-induced impairments of attention found in numerous studies to date.
- The studies presented within this Thesis make use of ambiguous figures to further explore the effect of alcohol upon attention. Ambiguous figures provide a novel way to assess mechanisms of attention.
- Previously, comparisons between the impairment of automatic and controlled mechanisms following alcohol consumption have been made across different experimental conditions and modalities.
- Here, ambiguous figures allow these mechanisms to be compared using a single stimulus type, eradicating the need for cross-modality comparisons.
- These studies are the first that have explored effects of alcohol on these attentional processes using ambiguous figures.

CHAPTER 2:

GENERAL METHODS

2.1. INTRODUCTION

The aim of this section is to provide a detailed description of the methods that were common to all experiments presented in this thesis. Descriptions of methods that are specific to a particular experiment are given in detail in the relevant chapters.

2.2. PARTICIPANTS

The number of participants used in these studies ranged from 24 to 30 to take into account the numbers used in studies of ambiguous figures (e.g. Nakatani & van Leeuwen, 2006; Kornmeier & Bach, 2004; Georgiades & Harris, 1997) and effects of alcohol on cognitive processes (Abroms et al., 2006; Abroms & Fillmore, 2004; Fillmore et al., 2000a, 2000b). Participants were students from the University of Birmingham who had normal or corrected-to-normal vision and who took part in return for £6 cash or 1 hour of course credits. To disguise the nature of the studies, they were advertised as "Investigations of the effects of alcohol on perception" and participants were told they might receive up to 5 units of alcohol. Participants were the first to volunteer who met the criteria outlined below, and were recruited through the Psychology departments' research participation scheme. This is an online system whereby researchers within the Psychology department advertise their study to prospective participants. A brief description of the study is given, including the inclusion and exclusion criteria. Participants choose which study they wish to participate in. All participants gave written consent and were debriefed at the end of the

study. All studies conformed to the BPS guidelines for conducting research with human participants. The participants used in the studies were tested in one study only, and were excluded from taking part in later studies.

2.2.1. Inclusion and exclusion criteria

To be eligible for participation, the volunteers had to be between 18-35 years old and were required to be self-reported regular consumers of alcohol who had drunk more than 7 units of alcohol on at least one occasion prior to testing, drank at least 8 units of alcohol per week, and were currently in good health. Participants were excluded if they reported any of the following: (1) past or present drug and/or alcohol dependency, (2) visual problems that could not be corrected with glasses or contact lenses, (3) were currently taking any prescribed and/or non-prescribed medication that would be affected by alcohol (excluding the contraceptive pill), (4) had ever suffered a serious head injury with loss of consciousness, (5) any neurological disorder such as epilepsy, (6) a history of psychiatric illness with or without treatment, (7) were pregnant or possibly pregnant, (8) failed to meet the criteria on the MAST (see section 2.3.3), (9) had eaten and/or drank and/or smoked within 1 hour prior to testing or had not eaten anything that day, (10) reported consumption of alcohol and/or recreational drugs within 12 hours prior to testing, (11) had a BrAL above zero upon arrival at the experiment. Saliva, blood, or urine samples were not taken to confirm whether participants had adhered to the exclusion criteria.

Participants gave verbal confirmation that they had not eaten within the hour prior to taking part, but they had eaten that day. BrAL was confirmed through a breath test (see

section 2.4. for more details). Weight was confirmed when participants were weighed as part of the alcohol administration procedure (see section 2.4. for more details). The eligibility criteria listed on the Research Participation Scheme stated that participants must have normal or corrected-to-normal vision, were not currently taking medications, had not suffered a serious head injury, were not pregnant, and must refrain from eating, drinking and smoking within 1 hour prior to the study participation. The inclusion and exclusion criteria were listed on the consent form, so by signing the form, participants gave written confirmation that they had adhered to these criteria. Information provided in a lifestyle questionnaire (see section 2.3.1) gave written confirmation that participants were regular consumers of alcohol, had no past or present drug and/or alcohol problems, not suffered a serious head injury, no known neurological disorders, no history of psychiatric illness, and had not consumed alcohol or recreational drugs within 12 hours prior to testing. Information provided in the MAST questionnaire (see section 2.3.3) gave written confirmation that participants had no alcohol misuse problems.

2.3. SCREENING TOOLS

2.3.1. Lifestyle Questionnaire

Participants completed a "Lifestyle Questionnaire" which was used to compile demographic information (see Appendix 1). This information was used to ensure there was no difference between the groups prior to testing that could affect performance on the experimental tasks, and to assess their suitability for testing based on the exclusion criteria outlined above. The questionnaire consisted of 16 questions on the participant's age, gender, highest level of educational qualification, number of years in full time education, amount of alcohol consumed (units per week), whether they smoked

cigarettes and if so, the number of cigarettes smoked per day and time since last use, the number of cups of tea and/or coffee consumed per day and time since last use, details of any prescribed and non-prescribed medication currently used, past or present psychiatric illnesses, any serious head injuries with loss of consciousness, any neurological disorders, and details of any recreational drugs used. Those who reported recreational drug use were asked for their age at first use of the drug, average number of days of current use per month, and the number of months at that frequency, typical amounts used per occasion, and the number of days since last use. All popular recreational drugs were listed, although participants were able to add other drugs if necessary. Any resulting group differences on any of these measures would be entered into statistical analyses as a covariate, details, if applicable, can be found in the Results sections of the studies presented in this thesis.

2.3.2. Alcohol Use Questionnaire (AUQ)

AUQ Score. The AUQ (Mehrabian & Russell, 1978) asks questions about participants' habitual use of alcohol (see Appendix 2). Participants are asked to use the previous six-month period as a guide to help them answer the questions, rather than trying to remember the precise quantities of alcohol consumed. Generally, retrospective reports of alcohol consumption fall approximately 20% below the actual amount of alcohol consumed (Feunekes et al., 1999). The AUQ yields a score based on number of alcoholic drinks per week (wine, questions 1-3; beer, questions 4-6; spirits, questions 7-9), speed of drinking (drinks per hour, question 10), number of times intoxicated in the last 6 months (question 11), and the percentage of times intending to get intoxicated (question 12). The AUQ has previously been used to measure cognitive functioning in

social drinkers (Weissenborn & Duka, 2003), and attentional bias towards alcohol-related stimuli (Duka & Townshend, 2004). The AUQ has been found to be a reliable measure to assess habitual drinking and has a reliability of 0.73 (Townshend & Duka, 2002). There was no exclusion criterion for this measure; rather, the AUQ score was used to ensure that there was no difference between the two groups based on their habitual use of alcohol. The equation for the AUQ score is:

Item 3 + Item 6 + Item 9 + (4 x Item 10) + Item 11 + (0.2 x Item 12)

Binge Score. A binge score was calculated to assess the relationship between drinking patterns and alcohol intake. Information given in items 10 (drinks per hour), 11 (number of times intoxicated in the last 6 months), and 12 (the percentage of times intending to get intoxicated) of the AUQ were used for this calculation (Townshend & Duka, 2002). The Binge score is calculated in the same way as the AUQ score (Mehrabian & Russell, 1978), but without the items 1–9, which refer to quantity and type of alcohol intake. This gives an indication of the pattern of drinking, rather than a measure of alcohol intake. This binge score is derived from the relationship between alcohol intake and drinking patterns and thus provides an overall view of habitual alcohol use. For example, a participant with a high binge score may have a similar weekly intake of alcohol with those with a lower binge score but would consume the majority of the alcohol in a single session. Townshend and Duka (2005) gave a cut off score of $\geq$ 24 units per week to indicate binge drinking, although there was also no exclusion criterion for this measure; the Binge score was used to ensure that there was no difference between the two groups based on their pattern of drinking. Townshend & Duka, (2002)

found that the relationship between 'binge score' and alcohol intake showed little correlation (r = 0.23), whereas a highly positive correlation (r = 0.58) was seen between number of drinks/h and the amount of alcohol consumed, indicating that the binge score is unrelated to the amount of alcohol drunk. It is therefore important to distinguish between alcohol intake and patterns of drinking (Hartley et al., 2004), especially as bingeing behaviour is a key factor for cognitive impairment (Stephens, 1995).

2.3.3. Michigan Alcohol Screening Test (MAST)

The MAST (Selzer, 1971) consists of 22 questions to identify possible alcohol misuse and was devised to provide a consistent, structured interview for detection of alcohol problems that could be rapidly administered by a non-professional as well as professional personnel (see Appendix 3). The questions relate to self-appraisal of social, vocational, and family problems frequently associated with the consumption of alcohol. The questions require 'yes' or 'no' responses, which are scored by allocating 1 point to each 'yes' answer, and 0 points for each 'no' answer (apart from questions 1 and 4 where 1 point is allocated to a 'no' response and 0 points to a 'yes' response). The total score is used to assess possible drinking problems, a score of 0-2 indicating no apparent problem, 3-5 early or middle problem drinkers, and 6 or more indicating problem drinkers. Those with a score of 6 or above were excluded from any further testing. This was to ensure that the sample included social but not hazardous drinkers. Selzer (1971) originally established a cut-off score of 5 for a diagnosis of alcoholism. However, it was later decided that this be increased to 6 to lower the likelihood of obtaining false positives (Selzer, 1975). Benussi et al. (1982) reported that a higher cut off value should be used for the general population, as the prevalence of alcohol problems is lower in

normal populations than in the populations used to develop the MAST. The MAST is a valuable diagnostic instrument with a long clinical and research history (Gibbs, 1983). The MAST has previously been used as a diagnostic tool for alcohol disorders in general practice (Nicol & ford, 1986), Schizophrenia (Drake et al., 1990), and assessment of lifetime and recent problems (Zung, 1982). Studies indicate that the MAST possesses a good internal-consistency with a Cronbach's alpha coefficient of .86 (Conley, 2001) and a test-retest reliability of .84 (Skinner & Sheu, 1982).

2.4. ALCOHOL ADMINISTRATION

Upon arrival, participants were breathalysed (Lion alcometer breathalyser, model S-D2; Lion Industries, Barry, Wales) to determine sobriety. Any participant with a positive BrAC would have been excluded from further testing, but no participant was excluded for this reason in any of the experiments. Weight (in kilograms) was measured to determine the quantity of drinks to be consumed. Participants were fully clothed for this, but were asked to remove shoes or other heavy items (e.g. coats). Participants were then assigned to either the alcohol or placebo condition. Participants in the alcohol condition were given a drink containing one-part vodka (males: 0.8 g/kg, females 0.75 g/kg of vodka, 37.5 % concentration), one-part non-alcoholic apple schnapps, and three parts Indian tonic water. Vodka was chosen because it is a colourless, odourless, and flavourless vehicle. Pilot tests confirmed that this dose achieved the desired BrAC level, which has been shown to produce impairment of cognition (e.g. Brumback et al., 2007; Weissenborn & Duka, 2003). Participants in the placebo condition were given drinks containing one-part non-alcoholic apple schnapps, and four parts Indian tonic water which has been shown to be an effective placebo (Birak et al., 2007). This quantity was

divided equally between three glasses and participants were given 5 minutes to drink each drink, which pilot studies confirmed produced a peak BrAC after 25 minutes approximately. Drinking alcohol at a steady and controlled pace like this allowed for a predictable BrAC to be achieved after a certain period of time (see section 1.2 for further information about alcohol absorption and distribution). After this 15 minute drinking time, the empty glasses were removed and participants were given a 10-minute interval to allow for absorption after which they were breathalysed. Testing took place between 12-6 pm to allow time for participants to eat breakfast to control for differences in absorption rate depending on stomach content and also to coincide with the times participants were likely to consume alcohol in normal drinking situations.

2.5. MATERIALS

Unless stated, the experiments were run on a Toshiba laptop (2.00 GHz, 1015 MB), with the stimuli presented on the computer screen with a resolution 1280 x 800 pixels (60Hz) 32 bit. Viewing distance was approximately 60 cm in all studies. The programme DMDX (Forster & Forster, 2003) was used to run the experiments, including the presentation of the stimuli, recording the number of reversals reported, and recording the total viewing time (ms). DMDX is a Windows based display system used to measure reaction times to visual and auditory stimuli. Jonathan Forster at the University of Arizona wrote the software.

2.6. PROCEDURE

Participants were asked to eat breakfast before arriving to the experiment, but not to eat, drink or smoke within the 1 hour period before they were due to be tested. Upon arrival,

participants were breathalysed (Lion alcometer breathalyser, model S-D2; Lion Industries, Barry, Wales) to determine sobriety and then weighed to determine the quantity of drinks to be consumed, any participant with a BrAL above 0 would have been excluded from further testing, although no participant was excluded for this reason.

Participants then completed, in order, the Lifestyle Questionnaire, AUQ, and the MAST questionnaires. Based on the answers to these questionnaires, participants would have been excluded from the study if they failed to meet any of the criteria outlined in section 2.2.1. The drinks were divided equally between three glasses with the instruction to drink one glass every 5 minutes. They had 15 minutes to drink all three drinks to produce a peak BrAC after 25 minutes approximately. After which time the glasses were removed and participants were given a 10-minute rest period before being breathalysed again.

Participants then completed the experimental tasks, which are described in detail in the relevant sections of the following chapters. Participants were breathalysed again after all experimental tasks had been completed. Testing took place in the afternoon between 12 and 6; participants took part in one session lasting approximately 1 hour with one participant per session. Test sessions took place in a lab, which contained a desk, chair, and a laptop. At the end of the session, participants were debriefed and asked whether they had any questions about the study.

2.7. DATA ANALYSIS

Independent *t*-tests were used to analyse group differences based on demographic information and questionnaire responses provided by the Lifestyle, AUQ, and MAST questionnaires. In all studies where post hoc tests are conducted, Bonferroni corrections were used to deal with multiple comparisons. All statistical tests were performed using SPSS (Chicago, IL, USA).

CHAPTER 3:

EXPERIMENT 1: EFFECTS OF A MODERATE DOSE OF ALCOHOL ON SELECTED TASKS FROM THE CAMBRIDGE NEUROPSYCHOLOGICAL TEST AUTOMATED BATTERY (CANTAB)

3.1. INTRODUCTION

The aim of this thesis is to examine the effects of a moderate dose of alcohol on cognition using a model system (ambiguous figures). However, before this can be achieved, it is necessary to demonstrate established effects of alcohol on general cognitive tests. Although seemingly straightforward, this is complicated by the variability in the existing literature, and so several factors have to be considered when designing an experiment to test for cognitive effects of alcohol. The aim of the current chapter is therefore to demonstrate established effects of alcohol on cognitive tests whilst taking into account some methodological limitations of previous studies.

It is well known that alcohol has a disruptive effect on psychomotor functions (Kennedy et al., 1993), sensory processes (Pearson & Timney, 1998), auditory and visual reaction time (Tzambazis & Stough, 2000; Jääskeläinen et al., 1996), higher cognitive functions such as cognitive flexibility (Lyvers & Maltzman, 1991), divided attention (Roehrs et al., 1994b), inhibition (Finn et al., 1999), pattern recognition (Fillmore & Vogel-Sprott, 1997), and planning and spatial recognition (Weissenborn & Duka, 2003). In contrast, a number of studies have failed to show an effect of moderate doses of alcohol on spatial working memory, pattern recognition (Weissenborn & Duka, 2003), and decision-making tasks (Fein et al., 2006). Additionally, moderate doses of alcohol have been

reported to reduce drinkers' ability to inhibit their behaviour while leaving their ability to activate behaviour unaffected (e.g. de Wit et al., 2000; Fillmore & Vogel-Sprott, 1997, 1999), and other reports suggest improvements in performance following moderate doses of alcohol (Palva et al., 1979). There are several possibilities as to why these studies differ in their findings, and the aim of the following sections is to highlight some of these and suggest ways to resolve the variability in findings.

These variable findings may result from differing methodologies that are used in these studies. Some have criticised the lack of sophistication of many of the studies in this area (Maylor et al., 1987; Oborne & Rogers, 1983) pointing out poor measures and methods and lack of proper control group comparisons. For instance, an important methodological consideration is the time allowed for alcohol absorption. Knowledge of those factors that may affect absorption and distribution of alcohol is important as these play an important role assessing resulting cognitive deficits (Maisto, 1978). The absorption period in some of these studies may not allow for the peak BrACl to be achieved during the study, which could explain non-significant findings in some studies. Where a dose of around 0.8 g/kg is used, an absorption period of around 10 minutes should be used with the aim to achieve peak BrACl at around 25 minutes post drinking. It may be that inappropriate methodological procedures that have been used in previous studies are responsible for some of the variable findings reported to date. In order for future studies to test for alcohol effects on these tests, reliable and consistent results are needed. Without this, there is no basis from which predictions can be made about performance on similar tasks.

It is possible that the conflicting results are due to a failure to test appropriate doses of alcohol. For example, at low to moderate doses, alcohol does not seem to impair performance on psychomotor tasks such as body balance and finger tapping (e.g. Mangold et al., 1996; Lukas et al., 1989), but impaired performance is evident on similar tasks at higher doses (e.g. Kennedy et al., 1993; Mattila et al., 1992). Similarly, cognitive tests that use low alcohol doses tend not to produce impairments (e.g. Heishman et al., 1997; Hindmarch et al., 1991, 1992). However, moderate alcohol doses between 0.5 – 0.8 g/kg tend to show impairment on cognitive tests (e.g. Pickworth et al., 1997; Millar et al., 1995; Wilkinson, 1995), with consistent effects being reported at doses of around 0.8 g/kg (Hindmarch et al., 1991), which are representative of the levels typically consumed by regular social drinkers (Kerr et al., 1991). Few studies tend to assess the effects of alcohol at doses much beyond 1 g/kg, because the sensitivity to detect subtle effects at these doses is considerably reduced (Hindmarch et al., 1991). This implies that in order to reliably assess and detect subtle impairments in cognitive functions, the dose of alcohol used should be around 0.8 g/kg.

Differences in the drinking patterns of participants within a sample have also been shown to affect performance on cognitive tests (e.g. Townshend & Duka, 2005; Weissenborn & Duka, 2003; Owen et al., 1995). These studies have shown that binge drinkers, relative to non-binge drinkers, are impaired on tasks measuring spatial working memory (Weissenborn & Duka, 2003), and pattern recognition (Owen et al., 1995). In addition, there is evidence that high consumers of alcohol develop tolerance to aversive effects of the drug (Evans & Levin, 2004; Fillmore & Vogel-Sprott, 1996). This suggests high consumers may show no impairment at doses that would otherwise

produce impairments in those who consume less alcohol. Additionally, experienced social drinkers have been shown to exhibit less alcohol impairment than novice drinkers, as measured by the pursuit rotor task (Fillmore & Vogel-Sprott, 1995, 1996) and the Pegboard and Digit-Symbol Substitution Task (DSST) (Evans & Levin, 2004). It has been suggested that some of the impairments found in novice drinkers are due, in part, to expectancy of alcohol impairment following doses that are unusual to them (Fillmore et al., 1998). Therefore, the drinking histories of participants should be assessed to ensure that where either significant or non-significant results are found, these are unlikely to be due to tolerance effects or baseline differences. Consequently, the studies presented within this chapter will take into account participants drinking histories. To be eligible to participate, participants must drink more than 8 units per week (Brumback et al., 2007), but fewer than 25 units per week for males or 16 for females (Cox et al., 1999).

An important issue to consider in studying the effects of alcohol on cognition is the possible differential effects of the two limbs of the blood alcohol concentration (BAC) curve. Although alcohol is generally considered to be a depressant, alcohol follows a biphasic trajectory with different pharmacological effects on the ascending alcohol limb versus the descending alcohol limb. Studies have demonstrated that impairment is typically restricted to the ascending limb of the BAC curve, with disruptions to motor functioning (Holdstock & de Wit, 1998), immediate memory (Jones, 1973), abstract reasoning (Jones & Vega, 1972) and attention (Hurst & Bagley, 1972). Interestingly, performance has been reported to improve significantly on the descending limb. This reduction in impairment observed during declining BACs suggests some process of

adaptation or habituation may occur during physiological exposure to a dose of alcohol (Kalant et al., 1971).

Interestingly, impaired performance is not restricted to the ascending limb for all tasks. Some tasks have shown a fairly constant degree of impairment during both ascending and descending BACs in the 50–80 mg/dl range, such as performance on the WCST and Go-Stop paradigm (Logan, 1994; Fillmore & Vogel-Sprott, 1999; Mulvihill et al., 1997). It appears the differential affects on the two limbs may be the result of differences in the complexity of the tasks. For instance, Fogarty and Vogel-Sprott (2002) have shown that performance on a motor skill and an information processing task on the two alcohol curves revealed impairment on both tasks during ascending BACs, but only motor skill test revealed improved performance on the descending limb. Similarly, Bennett et al. (1993) have shown that tolerance on the descending limb developed following low alcohol doses compared to high alcohol doses (1.0 g/kg compared to 0.75 g/kg). Hence, improvement in performance on the descending limb compared to the ascending limb likely occurs when sufficient mental resources are available due either to a relatively low BAC or to a simple task or process that is relatively little impaired by alcohol (e.g. retrieval). Therefore, the order of testing is important given the differential effects of the two limbs on cognitive processes. Consequently, the studies presented within this chapter will take these effects into account. The order in which the tests are presented will be counterbalanced to ensure that they are presented on both the ascending and descending plasma curves.

Effects of alcohol on cognitive performance may also be influenced by the presence of other psychopharmacological agents. Some drugs are known to have agonistic effects with alcohol such as benzodiazepines, certain antidepressants, antihistamines, and narcotic analgesics (e.g. Hindmarch & Subhan, 1986; Subhan & Hindmarch, 1983). Also, caffeine is recognised as a CNS stimulant and may counteract some of alcohol's effects (Azcona et al., 1995). Therefore it is also important to accurately measure and control for any interaction effects with other drugs.

The aim of the experiments presented in this chapter was to test the effects of a moderate dose of alcohol (0.8 g/kg) on various cognitive tests that have previously produced variable results and are subject to some of the limitations listed above. A secondary aim was to begin to dissect effects of this dose on specific cognitive tasks. To do this, tests from the CANTAB battery were utilised to broadly characterise neuropsychological deficits following consumption of moderate doses of alcohol in a sample of regular social drinkers. In the present study, a selection of tests was used from the CANTAB Battery. The CANTAB computerised tests have been validated in a large number of publications (CANTABeclipse, 2009), and have been shown to be sensitive to the effects of drugs such as MDMA (Semple et al., 1999), nicotine (Spinelli et al., 2006), scopolamine (Robbins et al., 1997), caffeine (Durlach et al., 2002), and antidepressants (Murphy et al., 2008). Weissenborn and Duka (2003) have shown significant effects of alcohol on working memory and planning tests using the CANTAB battery.

The CANTAB test battery used in the present study comprised tests of: Stockings of Cambridge (SOC), Pattern Recognition Memory (PRM), Intra/Extradimensional Shift Test (IED), Affective Go/No-go (AGN), as well as simple motor tests (Big Little Circle (BLC) and Motor Screening Test (MOT). This enabled testing of multiple aspects of cognitive processes, which have been reported to be impaired following alcohol consumption (see sections 1.3 and 1.4). Simple motor screening tests were used in the present study (MOT and BLC) as previous studies indicate that simple psychomotor tests are not affected by alcohol at doses used in the present study (e.g. Liguori et al., 1999). Therefore, lack of alcohol-induced effects on these simple motor tests will be used to inform the design of further experiments within this thesis that involve similar simple motor responses. Other tests taken from the CANTAB battery include those designed to assess attentional shift (IED), a test sensitive to changes to the fronto-striatal areas of the brain in which previous studies have shown impaired performance in binge drinkers (e.g. Scaife & Duka, 2009). The PRM test is generally considered to be a measure of working memory (WM), and recent research suggests that certain components of WM are differentially sensitive to alcohol (Saults et al., 2007). The SOC test is a spatial planning test based on the 'Tower of London' test (Shallice, 1982) and gives a measure of frontal lobe function (Owen et al., 1990). The test has also been shown to be sensitive to the effects of alcohol (Hartley et al., 2004). The AGN test assesses response inhibition and cognitive control, which have been shown to be impaired following alcohol consumption (e.g. Easdon et al., 2005). The methodology used in the present study allows for a more rigorous assessment of the effects of alcohol, and where findings from previous research can be replicated, it is less likely to be due to other confounding variables.

In summary, the experiments within this chapter will directly compare the cognitive function of an alcohol group with that of a placebo group using tests from the CANTAB battery. The aim of this chapter is to assess the effects of a moderate dose of alcohol on cognitive tasks and attempt to uncover specific cognitive tasks that are impaired under this dose.

3.2 METHOD

3.2.1. Participants

30 young social drinkers from the University of Birmingham with normal or corrected-to-normal vision participated in the study. There were two experimental groups, an alcohol group (5 male and 10 female; mean = 19.87 years, sd = 3.07, average self reported consumption of 18.27 units of alcohol per week), and a placebo group (4 male and 11 female; mean = 19.27 years, sd =1.22, average self reported consumption of 11.87 units of alcohol per week). Participants were told they were taking part in a study looking at the effects of alcohol on cognition; they were not told which aspects of cognition the different tests assessed or the expected outcomes. Participants were the first to volunteer who met the criteria outlined in section 2.2.1 and were recruited through the research participation scheme (see section 2.2).

3.2.2. Design

A between-subjects design was used with participants allocated pseudo-randomly to groups such that the groups were matched for age and gender. This was to account for the higher proportion of women within the Psychology department and any variations in age. All participants received the Motor Screening test first to familiarise them with the computer and the touch screen, and the order of presentation of tests was fully counter balanced thereafter to avoid confounding among the variables and to ensure tests were presented on both the ascending and descend alcohol plasma curves.

3.2.3. Inclusion and Exclusion criteria

Participants were excluded based on the criteria given in section 2.2.1.

3.2.4. Screening Tools

Details of the Lifestyle Questionnaire, Alcohol use questionnaire (AUQ; Mehrabian & Russell, 1978), Michigan Alcohol Screening Test (MAST; Selzer, 1971) that participants completed for this study are described in detail in section 2.3.

3.2.5. Alcohol Administration

A detailed description of the alcohol administration is given in section 2.4.

3.2.6. Materials and Tasks

All cognitive tasks were taken from the Cambridge Neuropsychological Test Automated Battery (CANTAB) task battery (Cambridge Neuropsychological Test Automated Battery; CeNeS Ltd, Cambridge, UK). Participants made their responses either via a specialised press-pad placed 15 cm in front of the computer screen, or by touching the screen itself, depending on the particular instructions of the task.

3.2.6.1. Psychomotor Tests

Motor Screening (MOT). Crosses were displayed one at a time at different locations on the touch-sensitive screen. Participants were required to touch the cross once it began to flash. This test screens for visual, movement and comprehension difficulties. This test has one outcome measure: speed of response.

Big/Little Circle (BLC). This is another training task and is used to prepare participants for the Intra-Extra Dimensional Set Shifting (IED) test, and is always given to

participants immediately prior to taking part on this task. This is designed to test attention, comprehension and the ability to learn and follow simple rules as well as rule reversals. Participants are presented with a series of pairs of circles, one big and one small. Participants are first required to touch the small circle for 20 trials followed by the large circle for the remaining 20 trials. This test has one outcome measure: speed of response.

3.2.6.2. Executive Functioning Tests

Intra-Extra Dimensional Set Shifting (IED). Feedback teaches the participant which stimulus is correct, and after six correct responses, the stimuli and/or rules change. Participants were presented with a series of multidimensional stimuli consisting of shapes and lines, see Figure 3.1. In stages 1 to 5 of the task (discrimination and learning stages), participants learn through trial and error to respond selectively to one specific shape, ignoring the other shape and the lines. In stage 6, the intradimensional shift, new shapes and lines are introduced, but shape continues to be the salient response dimension. In stage 7, the intradimensional reversal, the previously non-reinforced shape now becomes the correct response. The shifts at stages 6 and 7 are not thought to be primary measures of flexibility, as participants continue to respond to the same rule or set as in previous trials (Ozonoff et al., 2004). However, at stage 8, during the critical extradimensional shift, the correct rule now changes to the other dimension (e.g., the line) that has been irrelevant for the preceding dozens of trials. Finally, in stage 9, the extradimensional reversal, participants must respond to the previously non-reinforced line. To satisfy the set criterion of learning at each stage, participants must achieve 6 consecutive correct responses. If this criterion is met, participants progress through the

test. If at any stage the subject fails to reach this criterion after 50 trials, the test terminates and the maximum number of errors (25) was recorded for all subsequent stages not administered. The primary variables of interest were the number of errors committed and the number of trials taken to reach criterion at stages 6 (intradimensional shift), 7 (reversal of the intradimensional shift), 8 (extradimensional shift), and 9 (reversal of the extradimensional shift) of the task.

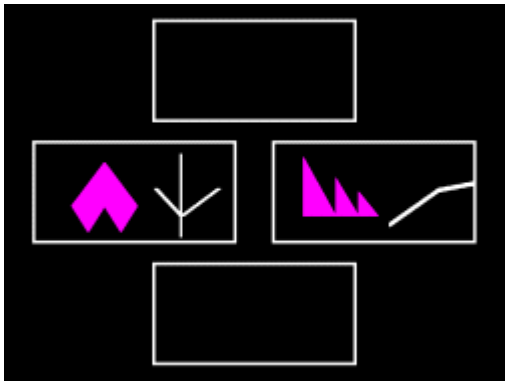


Figure 3.1. An example of the stimuli used in the Intra-Extra Dimensional Set Shifting (IED) test.

Pattern Recognition Memory (PRM). Participants are presented with 12 visual patterns one at a time in the centre of the screen. These patterns are designed so that they are difficult to be given verbal labels, which prevents rehearsal. For the recognition phase, participants are required to choose between a pattern they have already seen and a novel pattern. The test patterns are presented in the reverse order to the original order of presentation. This is repeated with a set of 12 new patterns to be remembered. This test has two outcome measures, the number of correct trials and reaction time.

Stockings of Cambridge (SOC). Three coloured balls are arranged at the top of the computer screen in a specific configuration set by the computer. In the 'plan and move' condition, participants see three identical balls, in a different configuration, displayed in the bottom half of the computer screen, which they need to match with the goal set, see Figure 3.2. The balls can be moved one at a time by touching the required ball, then touching the position where it is to be moved. They are told the minimum number of moves necessary to match the goal configuration (between two and five moves) and are instructed to use as few moves as possible. Participants are also instructed to wait to begin moving balls until they have planned their moves. Several problems requiring between two and five moves are then administered in a block. Following this phase, a second condition that controls for motor performance (the 'follow' condition) is administered. Participants are presented with their own solutions to problems in the 'plan and move' condition, seen move by move, at the top of the screen and are simply required to follow these moves on the lower half of the screen. By subtracting response times in the 'follow' condition from those in the 'plan and move' condition, it is possible to separately measure planning and movement times. Several performance variables are obtained. If participants make more than double the number of moves needed for the simplest solution, the problem is terminated. If three problems in a row are terminated, the entire test ends. The basic measure of planning efficiency is the 'Minimum Moves' variable, which is the total number of test problems completed in the fewest possible number of moves. The 'Mean Moves' variable describes the mean number of moves required by the subject to solve a test problem. The 'Initial Thinking Time' variable is the difference in time taken to select the first ball for the same problem under the 'plan and move' and 'follow' conditions. The 'Subsequent Thinking

Time' variable is obtained by taking the difference in time between selecting the first ball and completing the problem under the 'plan and move' and 'follow' conditions and dividing it by the number of moves made. This measure reflects the subject's speed of movement after the initial move has been made.

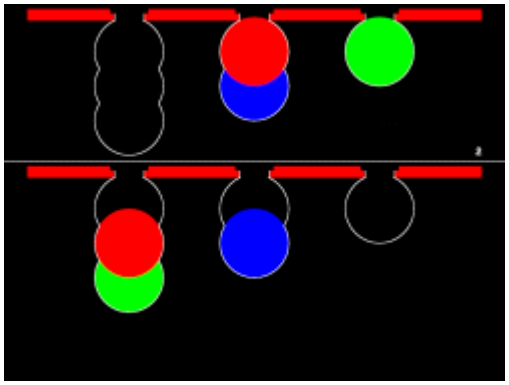


Figure 3.2. An example of the stimuli used in the Stockings of Cambridge (SOC) test.

Affective Go/No-go (AGN). The test consists of eight blocks, each of which presents 18 words from two different Affective categories: 9 Positive (for example, joyful), and 9 Negative (for example, hopeless). The subject is given a target category, and is asked to press the press pad when they see a word matching this category and to withhold a response to the other category. After two word blocks, the target category is changed so that the previous category is no longer the target. Conditions are alternated to create shift and non-shift response blocks. Words are displayed one at a time in the centre of the screen. Each word is displayed for 300 ms and there is an interval of 900 ms between the words. Variables extracted from this task were target (omission) errors (e.g., failing to respond to positive words during positive word blocks) and distracter (commission) errors (e.g., responding to positive words during negative word blocks)

during positive and negative word blocks, and during shift and non-shift blocks and reaction times of correct responses.

3.2.7. Procedure

The general procedure is outlined in detail in section 2.6. The CANTAB tasks were completed following the 10-minute rest period described in section 2.6. The Motor Screening test (MOT) was administered first for all participants, with the order of tests pre-randomised thereafter so that participants received the tests in different orders. All participants received the Big/Little Circle training test before the Intra-Extra Dimensional Set Shifting (IED).

3.2.8. Data Analysis

Reaction times below 100 ms were considered anticipatory (MOT, BLC, PRM, and AGN tests) and removed, and reaction times above 3 standard deviations above an individual's mean were deemed outliers (MOT, BLC, PRM, and AGN tests) and removed (MOT: 0% and 0%, BLC: 0%, and 0%, PRM: 0.3% and 0.5%, AGN: 0% and 1.2%, removed from data respectively).

The reaction time data for MOT, BLC, and PRM were analysed using independent t-tests, as was the error rate data for BLC and the number correctly remembered for the PRM test. Data for remaining tests were analysed using a mixed ANOVA, with Group (Alcohol and Placebo) as the between-subjects factor. For the AGN test, there was one within-subjects factor, Condition (Shift & No Shift). Separate ANOVAs were conducted on reaction time, omission errors (e.g., failing to respond to negative words

during negative word blocks), and commission errors (e.g., responding to positive words during negative word blocks). For the SOC test, there was one within-subjects factor, Stage (2, 3, 4, & 5 moves). Separate ANOVAs were conducted on the mean moves required per stage, initial thinking time per stage, and subsequent thinking time per stage. For the IED test, there was one within-subjects factor, Stage (ID shift, reversal of ID shift, ED shift, & reversal of ED shift). Separate ANOVAs were conducted on mean number of trials needed to reach criterion, and mean errors at each stage. For all tests, where a significant main effect and/or interaction was found, post-hoc independent and paired t-tests were used to determine the cause.

Demographic and questionnaire analysis followed the procedure outlined in section 2.7.

3.3. RESULTS

3.3.1. Demographics

Participant demographic and test results are summarised in Table 3.1. Independent *t*-tests and chi-squared tests revealed no age, gender, weight or education differences between the groups. There was no significant difference between the groups for units of alcohol per week. There were no group differences in caffeine use (ratio yes:no), and of those who reported caffeine consumption, there were no differences between the groups in caffeine consumption, or time since last use. There were no group differences in cigarette use (ratio yes:no), and of those who reported smoking, there were no group differences in the number of cigarettes smoked per day, or the time since last use. No significant differences were found between the groups for the MAST, and AUQ score and Binge scores. Of the recreational drugs that participants reported using, there were no group differences in the numbers that reported use compared with those who did not, and of those who did report use, there were no group differences in the number of days used per month (data not shown).

3.3.2. Breath Alcohol Concentration

All breath alcohol levels were 0 at the beginning of the session. There was a significant difference between the groups after drink consumption (alcohol group = 0.38 mg/l BRAlc) [$t(28) = 15.39$, $p = 0.01$], and at the end of the session (alcohol group = 0.13 mg/l BRAlc) [$t(28) = 6.90$, $p = 0.01$].

Table 3.1. Participant means, t-test and chi-squared results of between group comparisons (standard deviations in parentheses).

	Mean		Statistic	Value	Degrees of Freedom	p value
	Placebo Group (n=15)	Alcohol Group (n=15)				
Age (years)	19.27 (1.22)	19.87 (3.07)	t-test	0.70	28	0.49
Gender (male:female)	4:11	5:10	Fisher's Exact	n/a	n/a	1
Weight (kg)	67.07 (7.49)	71.64 (11.68)	t-test	1.27	28	0.21
Education (years)	14.00 (0.76)	14.13 (1.30)	t-test	0.34	28	0.73
Alcohol (units per week)	11.87 (5.08)	18.27 (24.49)	t-test	0.99	28	0.33
Caffeine (ratio yes:no)	10:5	9:6	Chi squared	0.14	1	0.71
(Cups per day)	3.40 (2.37)	2.56 (1.13)	t-test	-0.97	17	0.34
(Hours since use)	7.85 (7.23)	7.0 (7.04)	t-test	-0.26	17	0.80
Cigarettes (ration yes:no)	4:11	2:13	Fisher's Exact	n/a	n/a	0.65
(per day)	7.00 (3.56)	4.50 (3.54)	t-test	-0.81	4	0.46
(hours since use)	3.50 (3.11)	8.50 (0.71)	t-test	2.13	4	0.10
MAST	2.27 (1.33)	1.87 (1.36)	t-test	-0.81	28	0.42
AUQ	43.33 (23.55)	45.33 (42.58)	t-test	0.16	28	0.88
BINGE	31.13 (20.35)	29.40 (28.96)	t-test	-0.19	28	0.85

3.3.3. Task Performance

3.3.3.1. Motor Screening Test (MOT)

An independent t-test revealed no significant difference in reaction time (ms) between the alcohol group (M = 721.95, sd = 93.80) and the placebo group (M = 799.49, sd = 150.80) [t(28) = -1.69, p = 0.10].

3.3.3.2. Big/Little Circle Test (BLC)

An independent t-test revealed no significant differences in reaction time (ms) between the alcohol group (M = 598.41, sd = 119.84) and the placebo group (M = 646.40, sd = 90.44) [t(28) = -1.24, p = 0.23], or [t(28) = 0, p = 1].

3.3.3.3. Pattern Recognition Memory (PRM)

There were no differences in the number of patterns remembered correctly between the alcohol group (M = 21.33, sd = 3.31) and placebo group (M = 22.13, sd = 1.68) [t(28) = -0.83, p = 0.41], or the time taken to respond (ms) (alcohol group M = 1724.50, sd = 618.38; placebo group M = 1642.01, sd = 255.92) [t(28) = 0.47, p = 0.64].

3.3.3.4. Affective Go-No Go (AGN)

Reaction Time (RT): A repeated measure ANOVA was conducted on RT, with Group (Alcohol and Placebo) as the between-subjects factor and Valence (Positive and Negative) as the within-subjects factor. This analysis revealed a significant Group effect [F(1,28) = 25.28, p = 0.01], with the alcohol group having slower reaction times than the placebo group. There was no significant effect of Valence [F(1,28) = 0.33, p = 0.57],

and no significant Valence by Group interaction [F(1,28) = 0.14, p = 0.71], see Figure 3.3.

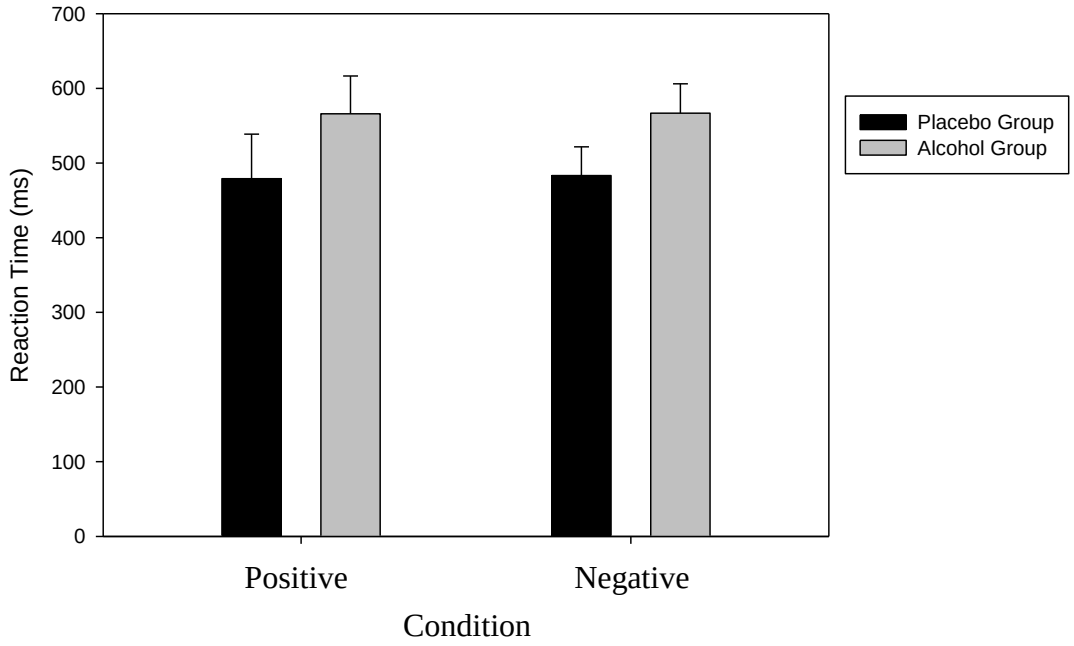


Figure 3.3. Mean Reaction Time (ms) for the alcohol and placebo groups for the Positive and Negative conditions. Error bars represent Standard Error mean.

Omission errors: A repeated measure ANOVA was conducted on target (omission) errors (e.g., failing to respond to negative words during negative word blocks), with Group (Alcohol and Placebo) as the between-subjects factor and Valence (positive and negative) as the within-subjects factors. This analysis revealed a significant Group effect [F(1,28) = 8.10, p = 0.01], with the alcohol group making more omission errors than the placebo group. There was a significant Valence effect [F(1,28) = 12.42, p = 0.01], and a significant Group by Valence interaction [F(1,28) = 28.50, p = 0.01]. Follow-up contrasts revealed that the alcohol group made more errors in response to

positive words than the placebo group [t(28) = 5.07, p = 0.01]. Whereas, there were no group differences in response to negative words [t(28) = -1.07, p = 0.29], see Figure 3.4.

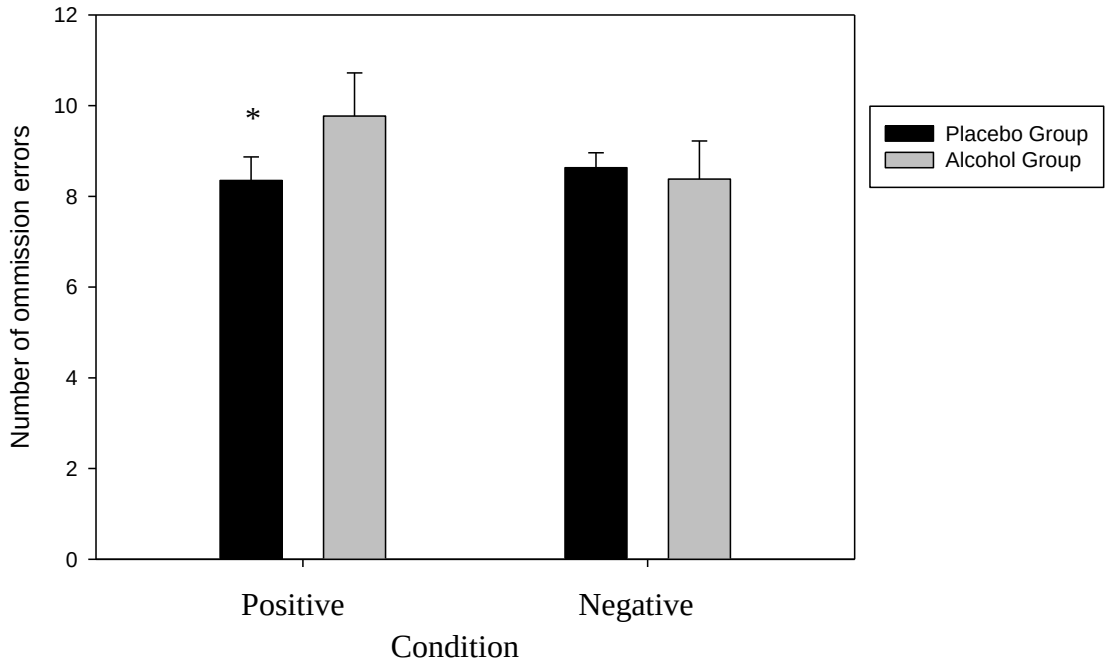


Figure 3.4. Mean target (omission) errors for the alcohol and placebo groups for the positive and negative word categories. * Indicates significant difference between alcohol and placebo groups ($p < 0.01$). Error bars represent Standard Error mean.

Distracter errors: A repeated measures ANOVA was conducted on distracter errors (e.g., responding to positive words during negative word blocks), with Group (Alcohol and Placebo) as the between-subjects factor and Valence (positive and negative) as the within-subjects factors. This analysis revealed a significant Group effect [F(1,28) = 11.10, p = 0.02], with the alcohol group making more distracter errors. There was a significant Valence effect [F(1,28) = 5.80, p = 0.02], and a significant Group by Valence interaction [F(1,28) = 6.42, p = 0.02]. Follow-up contrasts revealed that the alcohol group made more errors in response to positive words than the placebo group

[t(28) = 3.04, p = 0.01]. Whereas, there were no group differences in response to negative words [t(28) = 1.19, p = 0.25], see Figure 3.5.

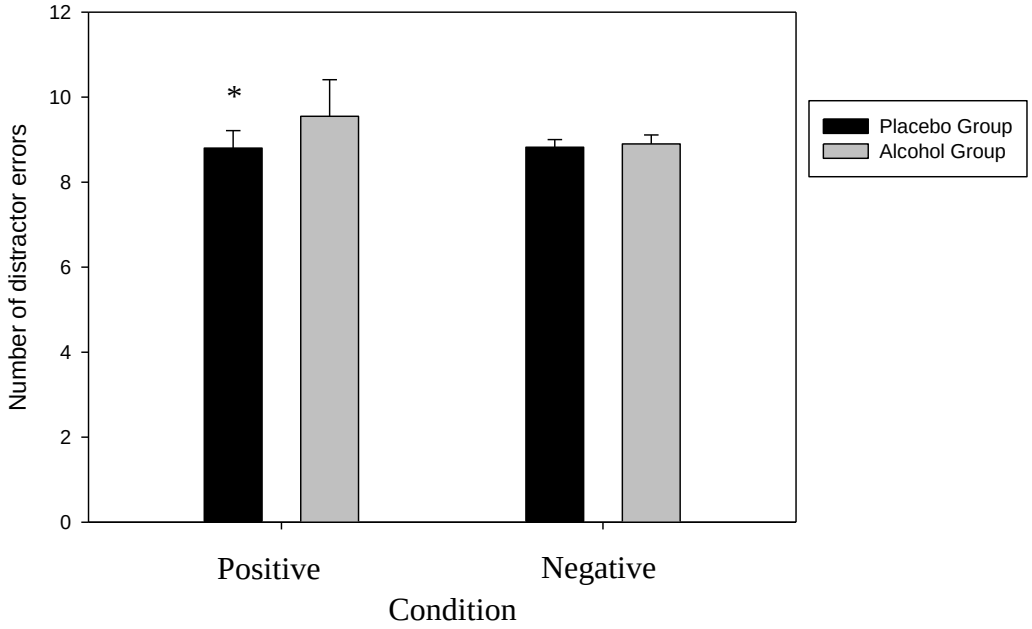


Figure 3.5. Mean Distracter errors for the alcohol and placebo group for Positive and Negative words. * Significantly more errors than Placebo (p < 0.01). Error bars represent Standard Error mean.

3.3.3.5. Intradimensional/Extradimensional Shift Task

Number of trials to reach criterion: The primary variables of interest were the number of trials taken to reach criterion at stages 6 (intradimensional shift), 7 (reversal of the intradimensional shift), 8 (extradimensional shift), and 9 (reversal of the extradimensional shift) of the task, see Table 3.2 for mean scores and standard deviations. A repeated-measures analysis of variance was conducted, with Stage as the within-subjects factor and Group as the between-subjects factor. This analysis revealed a significant Group effect [F(1,28) = 5.00, p = 0.03], with the placebo group needing fewer trials to reach criterion, a significant Stage effect [F(3,84) = 9.76, p = 0.01], with

more trials needed to reach criterion at stage 8, followed stages 7, 6, and 9, and a significant group by stage interaction effect [$F(3,84) = 4.20$, $p = 0.05$]. Follow-up contrasts to explore the source of the interaction effect revealed a lack of significant group differences in performance at stages 6, 7, and 9 (p's > 0.05), but significant group differences at stage 8 [$t(28) = 2.11$, $p = 0.04$]. At stage 8, the Alcohol group needed significantly more trials to reach criterion than the placebo group.

Number of errors: A similar analysis was undertaken to explore group differences in the number of errors made at stages 6 through 9, see Table 3.2 for mean scores and standard deviations. The repeated measures ANOVA again revealed a significant effect of both Group [$F(1,28) = 3.92$, $p = 0.05$], with the alcohol group making more errors, and Stage [$F(3,84) = 8.73$, $p = 0.01$], with the most errors made at stage 8, followed by stage 9, 7, and 6, as well as a marginally significant Group by Stage interaction [$F(3,84) = 3.48$, $p = 0.06$]. The interaction was again caused by a lack of significant group differences at stages 6, 7, and 8 (p's $> .05$), but significantly different performance at stage 9 [$t(28) = 2.08$, $p = 0.05$]. At stage 9, the Alcohol group committed significantly more errors than the placebo group.

Table 3.2. Mean performance on ID/ED Shift variables as a function of Group (standard deviations in parentheses). * Indicates significant differences between the alcohol and placebo groups ($p < 0.05$).

	Placebo Group (n = 15)	Alcohol Group (n = 15)
Trials to criterion		
Intradimensional shift	6.67 (0.49)	6.53 (0.52)
Intradimensional reversal	7.00 (0)	7.00 (0)
Extradimensional shift	9.53 (4.32)	19.07 (16.99)*
Extradimensional reversal	7.00 (0)	5.80 (3.55)
Mean	7.55 (1.20)	9.60 (5.27)*
Number of errors		
Intradimensional shift	0.67 (0.49)	0.53 (0.52)
Intradimensional reversal	1.0 (0)	1.00 (0)
Extradimensional shift	3.13 (2.90)	7.60 (9.36)
Extradimensional reversal	1.00 (0)	6.20 (9.74)*
Mean	1.45 (0.85)	3.83 (4.91)*

3.3.3.6. Stockings of Cambridge (SOC)

Minimum moves: An independent samples t-test was conducted on the Minimum Moves variable. This analysis revealed a significant difference in the number of minimum moves required, [$t(28) = -2.36$, $p = 0.03$], with the Alcohol group solving significantly fewer problems overall in the minimum moves required than the placebo group, see Table 3.3.

Mean moves: A repeated measures analysis of variance was conducted on the Mean Moves at each stage, with Stage as the within-subjects factor and Group as the between-subjects factor. This analysis revealed a significant Group effect [$F(1,28) = 7.72$, $p = 0.01$], with the alcohol group solving fewer problems in the minimum number of moves required. There was also a significant Stage effect [$F(3,84) = 260.20$, $p = 0.01$], with most moves required for 5 move problems, followed by 4, 3, and 2 move problems. There was no significant group by stage interaction effect [$F(3,84) = 1.94$, $p = 0.16$], see Table 3.3.

Initial thinking time: A similar analysis was conducted on the initial thinking time required, with Stage as the within-subjects factor and Group as the between-subjects factor. This analysis revealed a significant Group effect [$F(1,28) = 4.32$, $p = 0.05$], with the alcohol group having more initial thinking time than the placebo group. There was a significant Stage effect [$F(3,84) = 7.24$, $p = 0.01$], with most initial thinking time required for 5 move problems, followed by 4 move, 3 move, and 2 move problems. However, there was no significant stage by group interaction [$F(3,84) = 0.22$, $p = 0.74$], see Table 3.3.

Subsequent thinking time: Another analysis was conducted on the subsequent thinking time required, with Stage as the within-subjects factor and Group as the between-subjects factor. This analysis revealed a significant Group effect [$F(1,28) = 5.06$, $p = 0.03$], with the alcohol group having the greater subsequent thinking time. There was a significant effect of stage [$F(3,84) = 4.27$, $p = 0.02$], with most subsequent thinking time

required for 5 move problems, followed by 4 move, 3, and 2 move problems. There was no significant stage by group interaction [$F(3,84) = 1.15$, $p = 0.33$], see Table 3.3.

Table 3.3. Mean performance on SOC variables as a function of group (standard deviations in parentheses). * Indicates a significant difference between the alcohol and placebo group ($p < 0.05$).

	Placebo Group (n = 15)	Alcohol Group (n = 15)
Problems solved in minimum moves	8.93 (2.05)	7.40 (1.45)*
Mean moves		
(2 move problems)	2.00 (0)	2.07 (0.26)
(3 move problems)	3.23 (0.53)	3.47 (0.55)
(4 move problems)	5.28 (0.72)	5.48 (1.02)
(5 move problems)	6.87 (1.82)	7.43 (1.20)
Mean	4.61 (0.77)	4.35 (0.76)*
Mean initial thinking time (ms)		
(2 move problems)	625.83 (625.00)	1241.07 (1724.03)
(3 move problems)	2144.60 (1394.59)	2495.47 (2258.92)
(4 move problems)	3932.45 (2699.18)	3184.18 (3119.06)
(5 move problems)	4543.82 (2002.61)	5171.08 (6894.91)
Mean	2811.68 (1689.35)	3022.95 (3499.23)*
Mean subsequent thinking time (ms)		
(2 move problems)	345.38 (1157.45)	12.82 (49.64)
(3 move problems)	710.01 (1149.84)	485.87 (1081.57)
(4 move problems)	1165.08 (2429.66)	1684.32 (3611.43)
(5 move problems)	640.57 (905.75)	759.08 (909.07)
Mean	715.26 (1410.68)	735.52 (1412.93)*

3.4. DISCUSSION

The experiments presented within this chapter demonstrate several differences between the alcohol and placebo groups in terms of cognitive performance. These results are similar to previous studies that report alcohol-induced impairment on tests measuring inhibition, flexibility and planning (e.g. Scaife & Duka, 2009; Easdon et al., 2005; Hartley et al., 2004). Here, alcohol-induced impairments in performance following 0.8 g/kg (0.75 g/kg for women) of alcohol relative to placebo administration were observed for the AGN, IED, and SOC tests. However, there were no significant effects of alcohol on the MOT, BLC or PRM tests.

These findings are in agreement with many studies that have reported impaired performance following alcohol administration (e.g. Weissenborn & Duka, 2003; Tzambazis & Stough, 2000; Finn et al., 1999; Fillmore & Vogel-Sprott, 1997; Kennedy et al., 1993; Lyvers & Maltzman, 1991). Dose-related impairments on such tests have been reported at 0.5 g/kg, however, these impairing effects have been more reliably reported at doses of around 0.8 g/kg (Hindmarch et al., 1991), a dose comparable to the level consumed by regular social drinkers. The participants recruited to the present study were relatively moderate alcohol consumers, compared with some studies that do not adequately describe the drinking habits of their sample. This raises the question that those studies where effects were not found may have been due to tolerance to some effects of alcohol (Evans & Levin, 2004; Fillmore & Vogel-Sprott, 1996).

Some studies have demonstrated an alcohol-induced deficit in various psychomotor tasks (e.g. Jääskeläinen et al., 1996; Kennedy et al., 1993; Hindmarch et al., 1991), but

findings across these studies have been somewhat variable (e.g. Azcona et al., 1995; Finnigan et al., 1995). It has been suggested that impairments on simple motor tasks is spared under alcohol, and only the complex tasks reveal a deficit (Hindmarch et al., 1991). In the present study, no group differences in reaction times for the MOT and the BLC tests were found. These two psychomotor tasks are generally considered to measure simple motor tasks, and so support the notion that alcohol has a selective effect on only those psychomotor tasks that are more complex in nature. It can therefore be assumed that deficits found on the cognitive tasks in this study can be attributed primarily to differences in cognitive performance rather than slow motor speed per se.

In the present study, no group differences in performance were found on the PRM test. The PRM is considered to measure working memory (WM), which is considered to be an important component of cognition (Saults et al., 2007). Understanding alcohol-related effects are important, inasmuch as WM is thought to be critically involved in most complex behaviours (e.g. Baddeley, 2001; Cowan, 2001), such as attention (Cowan, 2001) and inhibition (Finn et al., 1999). However, studies examining alcohol's effects on WM have had mixed results (Paulus et al., 2006; Schweizer et al., 2006; Grattan-Miscio & Vogel-Sprott, 2005; Weissenborn & Duka, 2003; Finn et al., 1999). Research suggests that components of WM are differentially sensitive to alcohol (Saults et al., 2007), revealing impairments for tasks where stimuli are presented sequentially rather than in an array. This suggests that alcohol does not affect the ability to retain multiple concurrent items (e.g. the scope of attention: Cowan et al., 2005), but affects mnemonic strategies required to retain sequences (e.g. rehearsal: Baddeley et al., 1984). The results from the PRM test add to our understanding of alcohol's effects on WM,

showing that alcohol does not impair performance on sequential tasks when rehearsal is not possible. Both reaction time and the number of items remembered were not affected by alcohol, which supports previous findings reported by Weissenborn and Duka (2003). These results suggest that alcohol selectively impairs WM tasks that involve sequential presentation of material, but only when the information can be verbally encoded or recoded and maintained using rehearsal.

The SOC test is similar to the Tower of London test (Shallice, 1982), and both are used to measure planning ability. In addition, the SOC tests require an active search of possible solutions that have to be held in memory and transformed into sequences of motor movements, in this way placing a significant load on spatial working memory. Alcohol has been shown to impair planning abilities in similar tests, and these effects can be understood as a result of an increase in impulsivity due to an impairment of the underlying inhibitory mechanisms (Weissenborn & Duka, 2003). The results of the SOC test show that alcohol decreased the thinking time before initiating a move, which may also indicate an alcohol-induced impairment in impulsivity. The alcohol group was also found to have impaired planning ability, which can be seen by them completing fewer trials within the minimum moves required. Furthermore, the increase in subsequent thinking time once a solution had been initiated also suggests an alcohol-induced impairment in planning ability. The relationship between planning ability and frontal lobe function is well established within the literature (Shallice, 1982), although the underlying mechanisms that are responsible for accurate planning ability are as yet unknown. As previously mentioned, WM is an important component of cognition and it is reasonable to assume that it is involved in the SOC task as it requires an active search

of the possible solutions that need to be held in memory. However, the present study did not find a significant effect of alcohol on the PRM test, which would suggest that the impairments on the SOC test were not due to an effect of alcohol on working memory (Owen et al., 1990).

Alcohol is also understood to impair set-shifting abilities (e.g. Saraswat et al., 2006) and has been ascribed to impaired function in dorsolateral prefrontal cortex (DLPFC) or to disruption of the fronto-striatal circuitry (Purcell et al., 1997). The IED test is a measure of shifting and flexibility functioning, similar to the Wisconsin Card Sorting Test. The present study revealed significant differences between the alcohol and placebo groups on this particular test. The alcohol group performed worse on virtually all the dependent measures. However, this appears to be due to the alcohol group performance on two stages of the test. These two stages have previously been shown to implicate prefrontal cortex performance more than the other stages. For example, Dias et al. (1996) found that the extradimensional shift (stage 8) activates the dorsolateral prefrontal cortex, whereas the extradimensional reversal (stage 9), activates the orbitofrontal cortex. The results of the current study show that the alcohol group experienced significant difficulties compared to the placebo group at both these stages, but not at the earlier stages requiring discrimination learning and intradimensional shifting. This indicates that not all types of attention shifting are impaired by moderate doses of alcohol. It appears that only those requiring prefrontal cortical function are impaired. At the cognitive level, shifting within a category does not appear to be impaired, whereas shifting between categories is deficient. These results are consistent with the perseverative errors found in MDMA users (Fox et al., 2001b). Increased perseverative

behaviour on the WCST and in other task switching measures are linked to impaired adaptive control. Both perseverative behaviour and switch costs result from failure to shift set or to adjust a cognitive strategy in order to successfully complete a new task. The effect of alcohol on cognitive flexibility in the absence of effects on WM warrants the inference that alcohol consumption results in a selective impairment on cognitive flexibility.

The AGN test measures inhibition, which has previously been shown to be sensitive to the effects of alcohol (e.g. Fillmore et al., 2005; Vogel-Sprott et al., 2001). In the present study, a group difference in performance on the Affective Go/No-Go Task was found. Overall, the results replicate previous studies showing that alcohol consumption results in greater number of response errors and increased reaction times attributable to alcohol-induced impairment of inhibitory processes (e.g. Schweizer et al., 2006; Fillmore & Vogel-Sprott, 1999; Mulvihill et al., 1997). Although they may be mediated by different underlying brain structures, both omission and commission errors are predictable consequences of diminished cognitive control (Casbon et al., 2003). Following alcohol, one might anticipate alcohol-induced general impairment (i.e. increases in both commission and omission errors) due to alcohol-induced decreases in sensitivity (Casbon et al., 2003). It should be noted in the present study that alcohol did not produce a general impairment of task performance (i.e. increases in both commission and omission errors across all task conditions) or a selective impairment in inhibitory capacity (i.e. increases in commission errors only across all task conditions). Rather, the effects of alcohol occurred under specific conditions (i.e. in response to positive words) but were evident for both types of errors. The alcohol group made more

errors when responding to positive words indicating an information processing bias. On the other hand, the control group showed the opposite pattern for errors, suggesting that a positive bias is normal in normal populations. Additionally, the error rate results also suggest a mood-congruent attentional bias impairment of inhibitory processes following alcohol consumption. For example, previous studies have taken large numbers of omission errors in a Rapid Visual Information Processing tests to suggest a generalised deficit in attention (Erickson et al., 2005); and therefore, the bias found here may also reflect an alcohol-induced attention deficit. Equivalent performance by alcohol and placebo groups in response to negative words but impaired performance in response to positive words by the alcohol group indicates that salient stimuli affect attentional processing following moderate doses of alcohol, and so this mood-congruent attentional bias might occur within the context of an attention deficit.

Alcohol in the doses administered throughout this thesis has been shown to induce positive mood (deWit et al., 1987; Pohorecky, 1977). Consequently, the positive-response bias shown by the alcohol group could be interpreted in a similar way as the positive-response bias reported by patients in the manic phase of bi-polar disorder (Murphy et al., 1999). These patients have been found to exhibit attentional biases for positive emotional stimuli that are congruent with their current positive mood. Similarly, evidence has shown that healthy controls with induced elated mood exhibit a positive bias for remembering past experiences (Teasdale & Fogarty, 1979). The findings from the current study suggest that the alcohol-induced positive-response bias might be related to a general mood-congruent attentional bias, as both commission and omission errors were elevated. This interpretation is consistent with existing research

indicating that alcohol enhances positive expectancies and memory (Cooper et al., 1995) and such enhancement of positive memories is thought to underlie the reinforcing properties of alcohol (Koob & Nestler, 1997). However, the larger number of errors cannot be attributed completely to a generalised attention deficit, as errors were not comparable across affective valence. The results of the present study revealed equivalent performance by the alcohol group and control group in response to negative words, but impaired performance in response to positive words by the alcohol group. This indicates a mood-congruency effect in attentional processing following consumption of moderate doses of alcohol, supporting previous work showing impaired emotional modulation of inhibitory control (Loeber & Duka, 2009; Murphy et al., 1999). Although the alcohol-induced positive-response bias cannot readily explain the full range of impairments found on other tests in the present study and elsewhere in the literature, a narrowing of attentional focus to positive-related stimuli may contribute to widespread problems with focusing attention on different types of cognitive tasks.

The results of the studies within this chapter replicate those reported in previous studies using a rigorous methodology that controlled for some confounding factors (see Table 3.4 for a summary of the alcohol effects reported). Rather than being purely descriptive, these results will be used to inform the design of suitable experiments to assess some of the proposed underlying mechanisms involved in these effects. For instance, the results suggest that moderate doses of around 0.8 g/kg selectively impair components of cognition such as planning, inhibition, and flexibility. As discussed in Chapter 1, these components are complex and can be further fractionated into numerous sub-components. Therefore, the focus of the remaining chapters of this thesis will be to

design novel experiments that assess the effects of alcohol on the underlying mechanisms of these sub-components.

Table 3.4. Summary of the alcohol effects found using tests from the CANTAB battery. * Indicates significant differences between the alcohol and placebo groups ($p < 0.05$).

CANTAB Test	**Measure**	**Placebo Group**	**Alcohol Group**	**p value**	
MOT	Reaction time (ms)	799.49	721.95		0.10
BLC	Reaction time (ms)	646.40	598.41		1
PRM	N^{o} correct	22.13	21.33		0.41
	Reaction time (ms)	1642.01	1724.50		0.64
AGN	Reaction time (ms)	243.95	287.40	Group	0.01*
				Valence	0.57
				Interaction	0.71
	Omission errors	8.85	9.30	Group	0.01*
				Valence	0.01*
				Interaction	0.01*
	Distracter errors	8.49	9.08	Group	0.02*
				Valence	0.02*
				Interaction	0.02*
IED	Trials to criterion	7.55	9.60	Group	0.03*
				Stage	0.01*
				Interaction	0.05*
	N^{o} errors	1.45	3.83	Group	0.05*
				Stage	0.01*
				Interaction	0.06*
SOC	Minimum moves	8.93	7.40		0.03*
	Mean moves	4.61	4.35	Group	0.01*
				Stage	0.01*
				Interaction	0.16
	Initial thinking time (ms)	2811.68	3022.95	Group	0.05*
				Stage	0.01*
				Interaction	0.74
	Subsequent thinking time (ms)	715.26	735.52	Group	0.03*
				Stage	0.02*
				Interaction	0.33

CHAPTER 4:

EFFECTS OF ALCOHOL ON ATTENTIONAL MECHANISMS INVOLVED IN FIGURE REVERSALS

4.1. INTRODUCTION

Attention has been thought to play a role in figure reversals for some time (e.g. Meng & Tong, 2004; Toppino, 2003; Gomez et al., 1995; Horlitz & O'Leary, 1993; Liebert & Burk, 1985; Helmholtz, 1962). Further speculation about the involvement of attention came from the finding that the frontal-parietal areas of the brain that are involved in allocating attentional resources are activated when viewing ambiguous figures (Leopold & Logothetis, 1999). However, whilst these studies indicate an association between attention and figure reversals, they are unable to offer an indication as to the underlying mechanisms involved in figure reversals.

No study has examined alcohol's effects on the hypothesised attentional mechanisms involved in figure reversals. However, there is existing research that indicates that alcohol may impair the attentional processes involved. For example, alcohol is known to impair the ability to direct attention towards relevant stimuli and away from irrelevant stimuli (Fillmore et al., 2000a, 2000b; Tzambazis & Stough, 2000). It is frequently reported that alcohol disrupts performance on divided attention tasks that require simultaneously attending to two or more activities (e.g. Curtin et al., 2001; Fillmore et al., 1998). Divided attention studies also show that performance on primary tasks is unaffected by alcohol. Rather, it is the less salient task where alcohol is shown to impair performance (e.g. Fisk & Scerbo, 1987). Furthermore, the ability to ignore

stimuli on less salient tasks may be somewhat better than placebo (e.g. Erblich & Earleywine, 1995; Patel, 1988), indicating an improvement in the ability to screen out irrelevant information. The ability to ignore stimuli has been argued to be dependent upon inhibitory processes (Fillmore, 2000a).

Similarly, alternation between two versions of an ambiguous figure depends upon inhibition of the alternate interpretation (Helmholtz, 1962). Inhibitory mechanisms have been suggested to underlie this ability and to stabilise one interpretation over another (Girgus et al., 1977; Fisher, 1967). The finding that children below the age of five are unlikely to reverse supports this as there is evidence that inhibitory mechanisms are still developing at this age (Carlson & Moses, 2001; Kochanska et al., 1996; Girgus et al., 1977). Therefore a prediction arises that if alcohol reduces inhibition, it should make each interpretation less stable, and so should increase reversal rate. Consequently, the aim of this chapter is to explore this basic prediction in the context of several other factors likely to affect reversal rate.

Another prediction is that the effect of alcohol on reversal rate should interact with the effect of manipulations that alter the stability of the figure. Biased competition theory states that the competition between stimulus features can be controlled by introducing biases that favour the processing of a particular stimulus at the expense of competing stimuli (Duncan, 1996). Previous work has also shown that the addition of bias reduces the ambiguity of these figures (Toppino, 2003; Georgiades & Harris, 1997) thereby stabilising reversal rate, presumably because the unbiased interpretation becomes easier to suppress. In the case of ambiguous figures, the competition arises when the two

interpretations of the ambiguous figure compete for perceptual dominance in awareness. Therefore, the addition of a bias that favours the processing of one interpretation should do so at the expense of the alternate interpretation, resulting in fewer reversals when viewing biased figures. To manipulate this stabilising effect, biased versions of ambiguous figures will be presented along with the traditional ambiguous version. In the absence of alcohol, fewer reversals of perspective should be reported for the biased versions of the figures by making one of the interpretations weaker, and thus more easily inhibited, than the other. Alcohol, by weakening inhibition, should thus reduce this suppressive effect.

In addition, the number of reversals reported for a semantically meaningful illusion (Face-vase illusion) and a less semantically meaningful figure (Necker Cube illusion) will be recorded, to see whether alcohol affects reversal rate depending upon the type of figure presented. The ability to control reversal rate has been shown to be more effective when presented with more semantically meaningful ambiguous figures (Strüber & Stadler, 1999). This is thought to be because attentional mechanisms underlying the effect are able to act more effectively on semantically meaningful ambiguous figures due to the enhanced accessibility of meaningful information. Therefore it was predicted that the effects of alcohol on figure reversal might be more pronounced for the Face-vase illusion than the Necker Cube illusion.

4.2. STUDY 2: EFFECTS OF ALCOHOL ON ATTENTIONAL MECHANISMS INVOLVED IN FIGURE REVERSALS

4.2.1. METHOD

4.2.1.1. Participants

38 young social drinkers from the University of Birmingham with normal or corrected-to-normal vision participated in the study. There were two experimental groups, an alcohol (9 male and 9 female; mean = 24.3 years, sd = 5.81, average self reported consumption of 14.86 units of alcohol per week) and a placebo group (8 male and 12 female; mean = 21.9 years, sd = 2.63, average self reported consumption of 12.1 units of alcohol per week). Participants were told they were taking part in a study looking at the effects of alcohol on the perception of ambiguous figures, but were not told about different types of ambiguous figures or the effects of the bias on perception and the number of reversals usually reported in each case. Participants were the first to volunteer who met the criteria outlined in section 2.2.1 and were recruited through the Psychology departments' research participation scheme (see section 2.2).

4.2.1.2. Design

Participants were allocated pseudo-randomly to groups such that the groups were matched for age and gender. The ambiguous figures were presented in two blocks, one block contained three Necker cube stimuli, and the second block contained three Face-vase stimuli. Within each block, the unbiased figures were always presented first followed by the two biased versions. Unbiased stimuli were presented first within each

block due to the priming effects described by Long and Toppino (2004). The order of block presentations was fully counter balanced.

4.2.1.3. Inclusion and Exclusion criteria

Participants were excluded based on the criteria given in section 2.2.1.

4.2.1.4. Screening Tools

Details of the Lifestyle Questionnaire, Alcohol Use Questionnaire (AUQ; Mehrabian & Russell, 1978), Michigan Alcohol Screening Test (MAST; Selzer, 1971) that participants completed for this study are described in detail in section 2.3.

4.2.1.5. Alcohol administration

A detailed description of the alcohol administration is given in section 2.4.

4.2.1.6. Materials and Tasks

The ambiguous figures used were the Necker cube (Necker, 1832) and the Face-vase illusion (Rubin, 1958), see Figures 4.1a and 4.1d. These figures were chosen as previous studies have used them with other biasing manipulations effectively (e.g. Einhäuser et al., 2004; Andrews et al., 2002; Bisiach et al., 1999). Three versions of each ambiguous figure were used, the original ambiguous version of the figure, as well as two biased versions.

Necker Cube stimuli: In addition to the ambiguous version of the figure, in which all lines were equally bright, two biased versions were also used in which the luminance of

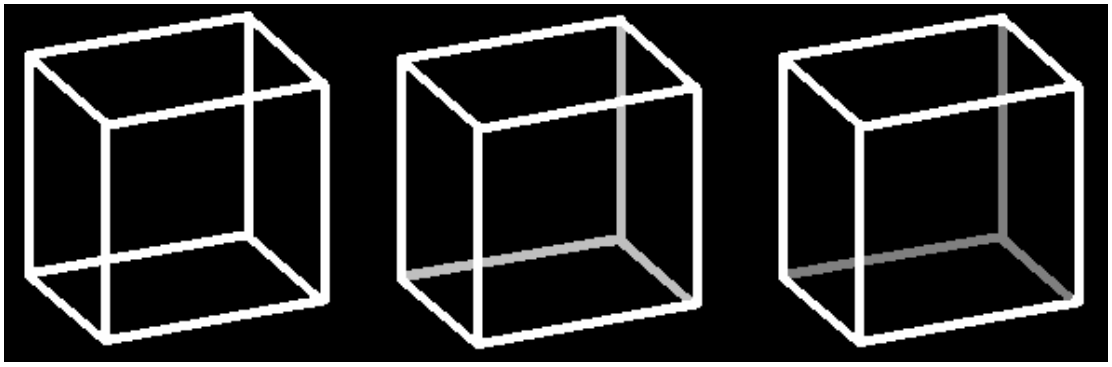
3 of the lines was reduced by 30% (Figure 4.1b) or 53% (Figure 4.1c) in order to bias interpretation towards the front face defined by the other lines. All Necker cube stimuli measured 8 x 8 cm.

Face-vase stimuli: Similarly, 2 biased versions of the face-vase illusion were used in which the salience of the vase interpretation was reduced by lowering its luminance by 30% (Fig 4.1e) or 50% (Fig 4.1f). Stimuli were presented on a white background with black contour lines distinguishing the components of the face and the vase (with the addition of grey to fill the vase component in the biased conditions) measuring 9 x 9 cm.

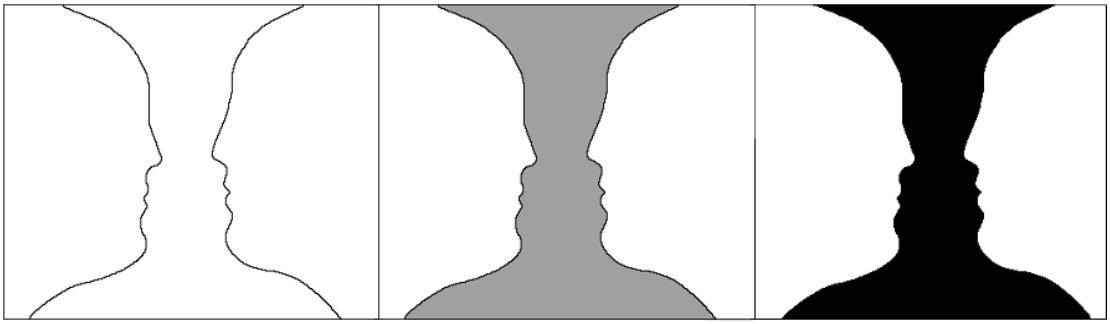
The experiment was run on a Packard Bell AMD Turion ™ 64 laptop (1.80 GHz, 44MB), with the stimuli presented on the computer screen with a resolution of 1024 x 768 pixels (0Hz) 32 bit at a viewing angle of 7.6° x 7.6° for the Necker cube stimuli and 8.5° x 8.5° for the face-vase stimuli.

Each ambiguous figure was presented for 1 minute. A 1 minute presentation time was chosen as previous studies have shown that the number of figure reversals increases up to 1 minute, after which the reversal rate reaches a steady rate (e.g. Cornwell, 1976; Virsu, 1975; Price, 1969a; Brown, 1955; Fisichelli, 1947; Philip & Fisichelli, 1945; Köhler, 1940). Reversal rate was under volitional control, participants were asked to increase reversal rate by making as many perceptual reversals as possible within the 1-minute presentation time, but only to report a reversal when they were certain of a change in perspective. Participants made their responses using the numeric keys

numbered 1 and 2 on the laptop's keyboard by holding down the appropriate key whilst experiencing the corresponding interpretation: key 1 representing the front face of the cube facing left and the key 2 representing the front face of the cube facing right for all Necker cube stimuli. Whereas the key 1 represented the two faces and key 2 represented the vase in the face-vase experiments. Participants were only shown an example of the ambiguous stimuli in the instructions sheet prior to testing. Previous research has shown that naïve observers are more likely to report one interpretation of an ambiguous figure more than the other interpretation (Botwinick, 1961; Leeper, 1935). Consequently, for ambiguous figures to be perceived as truly ambiguous, with each interpretation equiprobable, Georgiades and Harris (1997) have shown that by informing participants of the two interpretations prior to testing that equiprobablity can be achieved. For this reason, participants were informed of the two interpretations of the ambiguous stimuli prior to the experiment in the instruction sheet.



(a) Unbiased Necker cube *(b) Light-bias Necker cube* *(c) Dark bias Necker cube*



(d) Unbiased Face-vase *(e) Light-bias Face-vase* *(f) Dark bias Face-vase*

Figure 4.1. The ambiguous figures used for the experiment, including the two unbiased stimuli used for both the Necker cube and Face-vase illusions (Figures 4.1a and 4.1d), the two light biased stimuli (Figures 4.1b and 4.1e), and the two dark biased stimuli (Figures 4.1c and 4.1e).

4.2.1.7. Procedure

The general procedure is outlined in section 2.6. Following the 10-minute rest period described in section 2.6, the ambiguous figures task was completed, after which participants were breathalysed again.

4.2.1.8. Data Analysis

A mixed ANOVA was used to analyse group differences on the ambiguous figure tasks for the total number of reversals reported within one minute, with Group (Alcohol or Placebo group) as the between subjects factor, and two within subjects factors, Bias (unbiased, light bias, and dark bias), and the Illusion Type (Necker cube and Face-vase). To control for individual differences in alternation rate, the data for each participant was normalised. Normalised alternation rates were calculated by dividing the total number of reversals reported by the total viewing time after the first and last data values had been removed, with the resulting value being used for the ANOVA analysis. The first data value was removed from the analysis as this represented the initial planning time, and was not a measure of a reversal itself. The last data value was removed, as this was the time between the last reversal reported by the participant and the end of the test session and was not a measure of a reversal having occurred. A similar calculation has been used previously (see Zheng & Ukai, 2006), and is thought to be an appropriate measurement to evaluate the frequency of perceptual alternations.

Demographic information and questionnaire analysis followed the procedure outlined in section 2.7.

4.2.2. RESULTS

4.2.2.1. Demographics

Participant demographic and test results are summarised in Table 4.1. Independent *t*-tests and chi-squared tests revealed no age, gender, weight or education differences between the groups. There were no significant differences between the groups for units of alcohol per week. There were no group differences in caffeine use (ratio yes:no), and of those who reported caffeine consumption, there were no differences between the groups in caffeine consumption, or time since last use. There were no group differences in cigarettes use (ratio yes:no), and of those who reported smoking, there were no group differences in the number of cigarettes smoked per day, or the time since last use. No significant differences were found between the groups for the MAST, and AUQ score and Binge scores. Of the recreational drugs that participants reported using, there were no group differences in the numbers that reported use compared with those who did not, and of those who did report use, there were no group differences in the number of days used per month (data not shown).

4.2.2.2. Breath Alcohol Concentration

All breath alcohol levels were 0 at the beginning of the session. There was a significant difference between the groups after drink consumption (alcohol group = 0.59 mg/l BRAlc) [$t(35) = 13.11$, $p = 0.01$], and at the end of the session (alcohol group = 0.31 mg/l BRAlc) [$t(35) = 21.22$, $p = 0.01$]. Data from one participant was removed due to a false reading.

Table 4.1. Participant means, t-test and chi-squared results of between group comparisons (standard deviations in parentheses).

	Mean		Statistic	Value	Degrees of Freedom	*p* value
	Placebo Group (n = 20)	Alcohol Group (n = 18)				
Age (years)	22.15 (2.63)	24.33 (5.81)	t-test	1.52	36	0.14
Gender (male:female)	8:12	9:9	Chi-Squared	0.38	1	0.54
Weight (kg)	76.09 (18.02)	74.36 (12.88)	t-test	-0.34	36	0.74
Education (years)	16.60 (1.54)	17.22 (1.83)	t-test	1.14	36	0.26
Alcohol (units per week)	12.1 (7.04)	14.86 (11.42)	t-test	0.91	36	0.37
Caffeine (ratio yes:no)	14:6	13:5	Chi-Squared	0.02	1	0.88
(Cups per day)	3.64 (2.00)	2.77 (1.30)	t-test	-1.33	25	0.20
(Hours since use)	3.39 (1.85)	3.42 (1.96)	t-test	0.04	25	0.97
Cigarettes (ratio yes:no)	2:18	7:11	Fisher's Exact	n/a	n/a	0.06
(per day)	1.0 (1.41)	3.71 (2.63)	t-test	1.36	7	0.22
(hours since use)	7.00 (1.41)	5.14 (2.04)	t-test	-1.18	7	0.28
MAST	2.06 (1.47)	2.27 (1.33)	t-test	1.82	36	0.07
AUQ	27.63 (12.65)	28.52 (13.61)	t-test	0.21	36	0.83
BINGE	15.60 (10.23)	14.97 (11.76)	t-test	-0.18	36	0.86

4.2.2.3. Task Performance

A mixed ANOVA was conducted on the average reversal rate, with Group (Alcohol and Placebo) as the between-subjects factor, and two within-subjects factors, Bias (Ambiguous, Light Bias, and Dark Bias), Illusion Type (Face-vase and Necker Cube), see Table 4.2 for the mean number of figure reversals reported and total viewing times. This analysis revealed no significant main effects of Group [$F(1,36) = 2.86$, $p = 0.10$], or Bias [$F(2,72) = 1.98$, $p = 0.16$]. However, there was a significant Group by Bias interaction [$F(2,72) = 4.46$, $p = 0.03$]. Follow-up contrasts to explore the source of this interaction revealed no significant differences between the groups for the ambiguous stimuli [$t(36) = 0.82$, $p = 0.42$], or the Light Bias stimuli [$t(36) = 1.76$, $p = 0.09$], but there was a significant difference between the groups for the Dark Bias stimuli [$t(36) = 2.00$, $p = 0.05$], see Figure 4.2. Further follow-up contrasts showed the alcohol group reported a significant difference in figure reversals between the ambiguous and light bias [$t(17) = -2.09$, $p = 0.05$] and ambiguous and dark biased conditions [$t(17) = -2.17$, $p = 0.05$]. However, the alcohol group did not report a significant difference in the number of figure reversals between the light and dark biased conditions [$t(17) = -0.43$, $p = 0.67$]. Furthermore, a correlation analysis showed that reversal rate was not associated with changes in BrAlc on the ascending (e.g. BAC2, $r = 0.19$, $p = 0.46$) or descending (e.g. BAC2, $r = -0.29$, $p = 0.26$) alcohol curve. Whereas the Placebo group did not report a significant difference in reversals between the ambiguous and light biased condition [$t(19) = 0.46$, $p = 0.65$], ambiguous and dark biased condition [$t(19) = 0.84$, $p = 0.41$], or the light and dark bias condition [$t(19) = 1.00$, $p = 0.33$], see Figure 4.2. The main effect of Type was significant [$F(1,36) = 15.41$, $p = 0.01$], with both groups reporting more reversals for the Face-vase stimuli than the Necker cube stimuli. The

Type by Group interaction approached significance [F(1,36) = 3.89, p = 0.06]. Post hoc tests show that there were no group differences for the Necker Cube figures [t(36) = 0.72, p = 0.48], but the difference for the Face-vase figures was marginally significant once the bonferroni correction was applied [t(36) = 2.18, p = 0.03], see Figure 4.2. There were no significant interactions for Bias by Type [F(2,72) = 0.20, p = 0.76], or Bias by Type by Group [F(2,72) = 1.60, p = 0.21].

Table 4.2. Means and standard deviations (in parentheses) for the number of figure reversals and total viewing times for each group. * Indicates significant difference between alcohol and placebo groups ($p < 0.01$).

Experimental Task	**Placebo Group** (n = 20)		**Alcohol Group** (n = 18)	
	Number of reversals	Total viewing time	Number of reversals	Total viewing time
Face Vase Illusion				
(Ambiguous Stimulus)	46.70 (17.88)	57.33 (2.41)	56.56 (31.85)	56.72 (4.49)
(Light Bias Stimulus)	41.60 (24.24)	56.56 (1.74)	65.06 (35.94)	54.03 (7.24)
(Dark Bias Stimulus)	41.40 (24.53)	56.56 (1.93)	67.44 (41.57)	55.78 (5.61)
Necker Cube Illusion				
(Ambiguous Stimulus)	33.65 (23.52)	54.22 (9.06)	34.89 (30.83)	54.17 (4.82)
(Light Bias Stimulus)	35.80 (25.26)	56.79 (1.81)	42.17 (36.15)	56.86 (1.87)
(Dark Bias Stimulus)	32.65 (24.83)	56.85 (1.64)	43.33 (34.58)	55.08 (7.25
Mean	38.63 (23.38)	56.39 (3.10)	51.58 (35.15)*	55.44 (5.21)*

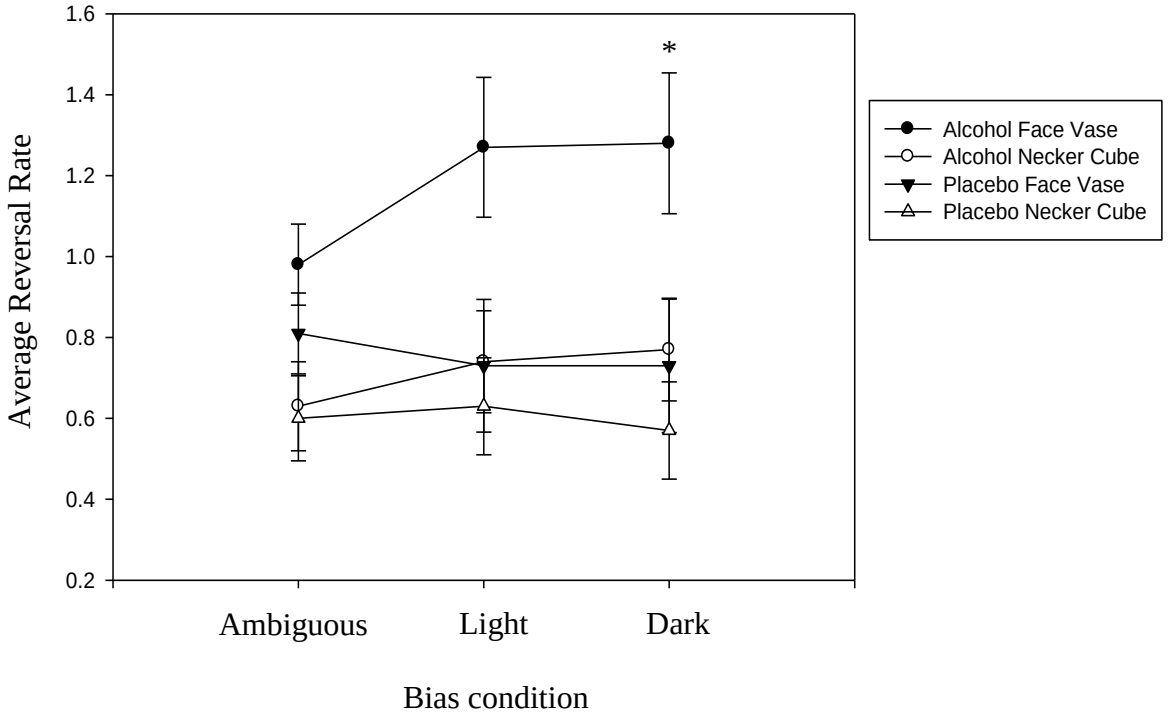


Figure 4.2. Group differences in average reversal rate for each condition depending upon the type of figure presented. * Indicates significant difference between alcohol and placebo groups ($p < 0.01$). Error bars represent Standard Errors of means.

4.2.3. DISCUSSION

Administration of a moderate dose of alcohol to social drinkers resulted in an increase in the ability to voluntarily reverse an ambiguous figure in line with prediction. Also, the results cannot be accounted for by differential effects of the two limbs of the blood alcohol concentration (BAC) curve on reversal rate as reversal rate was not associated with changes in BrAlc. However, the effect of alcohol was not as straightforward as predicted because there was no effect of alcohol when viewing the ambiguous version of the face vase illusion. Reversal rates were only significantly greater than placebo for the biased versions of the figure. In addition, the effect was seen predominantly for the face vase illusion and not for the Necker Cube illusion.

It is possible that the effect of alcohol on reversal rate is due to generalised effects of the drug on task performance. For example, alcohol might have had a general effect on arousal/mood or might have enhanced compliance with the experimental instructions or introduced a general positive response bias. However, this explanation cannot account for the fact that the facilitatory effect of alcohol is largely confined to the face-vase version of the illusion. Similarly, a general disinhibitory effect of alcohol seems unlikely for this reason.

One might argue that the group differences on this task might reflect the limited resources available for accurately processing information following alcohol. As capacity limitations on information processing are revealed when alternating between two tasks, the alternation between the two interpretations of the figure might also encounter similar capacity limitations. However, the capacity limitation argument suggests such

impairment is evident by a slowing of response time, which is assumed to reflect delay between completing the first task and beginning the second (Johnston & Heinz, 1978). In which case, the capacity limitation argument might predict that reversal rate would be lower than placebo following alcohol as the limited resources available for processing the information would result in a time delay, thus reducing reversal rate. Also, Reisberg (1983) and Reisberg and O'Shaughnessy (1984) have shown that cognitive load reduces reversal rate. Given that reversal rate increased following alcohol, a capacity limitation account cannot readily account for the findings.

Another possible explanation for the higher reversal rate reported by the alcohol group might be that alcohol impairs the ability to maintain a particular representation, rather than impairing underlying inhibitory mechanisms. It is possible that the inability to maintain a particular interpretation, resulting in increased reversals, is due to alcohol's known effects on impulsivity (Dougherty et al., 1999, 2000; Mulvihill et al., 1997; Marcinski et al., 2007). Increased impulsivity following alcohol might explain the increased reversal rate if alcohol increases impulsive responding. However, although this is one possible explanation, it seems unlikely given that alcohol increased reversal rate only under certain conditions when viewing the Face-vase illusion. Increased impulsivity following alcohol ought to produce a similar effect for both figure types. Given that there were no group differences when viewing the Necker cube figure, it seems unlikely that alcohol-induced increases in impulsivity can account for the results.

Effects of alcohol on attentional processes have been observed after similar doses using different methods. For example, studies have shown that the ability to divide attention is

greatly impaired following moderate doses of alcohol (e.g. Curtin et al., 2001; Schulte et al., 2001; Erblich & Earleywine, 1995; Moskowitz & Burns, 1990). In addition, it has long been claimed that selective attention and related aspects of visual information processing are impaired under similar alcohol doses (e.g. Fillmore et al., 2000a, 2000b; Tzambazis & Stough, 2000; Post et al., 1996). The finding that alcohol increased reversal rate is consistent with an effect to impair inhibitory attentional processes but is not consistent with the Alcohol Myopia model (e.g. Steele & Josephs, 1988, 1990; Steele & Southwick, 1985). The Alcohol Myopia model suggests that the allocation of attention is restricted to the most salient features in the environment. As a result, the processing of other information is greatly impaired (e.g. Steele & Josephs, 1988, 1990; Steele & Southwick, 1985). According to this model, it would be predicted that attention would be restricted to the biased features of the ambiguous figure and so fewer reversals would be reported following alcohol but this was not the case.

While the effect of alcohol to increase reversals is generally consistent with an interpretation in terms of alcohol-induced impairment of inhibitory attentional processes, this interpretation is complicated by the finding that the addition of bias to the ambiguous figures did not reduce reversals as predicted in the placebo group in the present study. The addition of bias produced only a small reduction in reversal rate in the absence of alcohol, and the effect of alcohol was generally facilitatory – increasing reversal rates above baseline, rather than restoring them to baseline. This unexpected result may be due to the type of bias used in the present study. The figures in this study were biased using different degrees of luminance to strengthen one interpretation over the other. The manipulation of luminance used for the Necker cube does seem a

plausible way to strengthen one interpretation over the other. For example, if the luminance of the manipulated lines is further reduced to zero, the figure becomes completely unambiguous. Indeed previous pilot studies had shown that the addition of bias was effective in reducing reversal rates. However, for the face-vase illusion, it is possible that the biasing altered the absolute strength of the interpretations rather than just the balance between the two possible interpretations as initially predicted. Manipulation of the luminance not only favours the "brighter" face interpretation of the figure, but it also increases the contrast between the two interpretations. Given this greater contrast, it is reasonable to suggest that stronger inhibition would be needed in order to suppress the alternative interpretation. Perhaps then, alcohol weakens the inhibition to the point where the suppression is no longer possible, and a faster reversal rate results.

Therefore, the effect of alcohol does not appear to be as simple as previously hypothesised. Alcohol does not result in more figure reversals being reported simply because inhibition is weakened. The effect of alcohol on reversals seems to depend upon the precise nature of the stimulus presented and may be dependent upon the specific experimental conditions of this first study. For example, participants were asked to make as many reversals as they could. The aim of the second study is to explore the extent to which this conscious control is important for the effects of alcohol on reversal rate.

4.3. STUDY 3: EFFECTS OF ALCOHOL ON AUTOMATIC CONTROL MECHANISMS INVOLVED IN FIGURE REVERSALS

4.3.1. INTRODUCTION

A distinction has been made between attentional mechanisms that are volitionally controlled and those that are automatic (Marzi, 1999; Shimojo et al., 1999). Ambiguous figures allow both these mechanisms to be assessed. Existing ambiguous figure research suggests an important role for automatic, low-level mechanisms in figure reversals (Blake, 1989; Köhler & Wallach, 1944) but that reversals can also be consciously controlled using different instructions (Strüber & Stadler, 1999). There is research to show that the instructions given to participants can determine whether controlled or automatic control mechanisms are activated. For instance, instructions to increase reversal rate is thought to activate intentional control mechanisms (e.g. Liebert & Burk, 1985; Peterson & Hochberg, 1983; Seth & Reddy, 1979; Pelton & Solley, 1968), whereas instructions not to bring reversals under volitional control is assumed to activate automatic control mechanisms (Strüber & Stadler, 1999).

Research on the effect of alcohol on conscious versus automatic attention has produced variable results, with some indicating that both mechanisms can be impaired under moderate doses of alcohol (Holloway, 1995), whereas others suggest that impairments are restricted to controlled mechanisms (Abroms et al., 2006; Fillmore & Vogel-Sprott, 2006; Holloway, 1995). In the first study in this chapter there were more reversals after alcohol than placebo when participants were asked to voluntarily reverse the ambiguous figures. In the present study, the volitional component was removed by asking

participants not to intentionally reverse their interpretation. Comparison of Study 3 with Study 2 should therefore allow us to investigate whether the effects of alcohol observed in Study 2 are dependent upon the volitional nature of the reversals.

In addition to varying the instructions, Study 3 will also include an additional no-drink control group. Participants in this group will know prior to testing that they have not received alcohol. Therefore, Study 3 will control for any expectancy effects that might have influenced the results of Study 2 in some way. There is some research to suggest that people can become hypervigilant following alcohol consumption in an attempt to compensate for their anticipated poorer performance (Marczinski & Fillmore, 2005). In which case, the addition of the no-drinks control group is hoped to provide reassurance that the placebo is an appropriate baseline and that expectations are not distorting the comparisons.

4.3.2. METHOD

4.3.2.1. Participants

40 young social drinkers from the University of Birmingham with normal or corrected-to-normal vision participated in the study. There were three experimental groups, an alcohol group (6 male and 9 female; mean = 21.4 years, sd = 2.41, average self reported consumption of 10.00 units of alcohol per week), a placebo group (7 male and 8 female; mean = 21.4 years, sd = 2.35, average self reported consumption of 15.13 units of alcohol per week), and a no-drinks control group (6 male and 4 female; mean = 22.2 years, sd = 3.29, average self reported consumption of 14.70 units of alcohol per week). Participants were told they were taking part in a study looking at the effects of alcohol on the perception of ambiguous figures, but were not told about different types of ambiguous figures or the effects of the bias on perception and the number of reversals usually reported in each case. Participants were the first to volunteer who met the criteria outlined in section 2.2.1 and were recruited through the Psychology departments' research participation scheme (see section 2.2).

4.3.2.2. Design

The design was identical to that used in Study 2, see section 4.2.1.2.

4.3.2.3. Inclusion and Exclusion criteria

Participants were excluded based on the criteria given in section 2.2.1.

4.3.2.4. Screening Tools

Details of the Lifestyle Questionnaire, Alcohol Use Questionnaire (AUQ; Mehrabian & Russell, 1978), Michigan Alcohol Screening Test (MAST; Selzer, 1971) that participants completed for this study are described in detail in section 2.3.

4.3.2.5. Alcohol administration

A detailed description of the alcohol administration is given in section 2.4.

4.3.2.6. Materials and Tasks

The stimuli used were identical to those in Study 2, see section 4.2.1.6.

The task is essentially identical to Study 2, which is described in detail in section 4.2.1.6, the only difference being that participants were asked to report spontaneous reversals during the 1-minute presentation time. Participants were asked not to intentionally reverse their interpretation, and only to report a reversal when they were certain of a change in perspective.

4.3.2.7. Procedure

The general procedure is outlined in section 2.6. Following the 10-minute rest period, participants completed the ambiguous figures task, after which participants were breathalysed again.

4.3.2.8. Data Analysis

Group differences in average reversal rate were analysed in the same way as Study 2, see section 4.2.1.8. The only difference here is that the between-subjects factor now included a no-drinks control group as well as the two groups used in Study 2.

One-way ANOVAs and chi-squared tests were used to analyse group differences based on demographic information and questionnaire responses provided by the Lifestyle, AUQ, and MAST questionnaires.

4.3.3. RESULTS

4.3.3.1. Demographics

Participant demographic and test results are summarised in Table 4.3. One-way ANOVAs and chi-squared tests revealed no age, gender, weight or education differences between the groups. There were no significant differences between the groups for units of alcohol per week. There were no group differences in caffeine use (ratio yes:no), and of those who reported caffeine consumption, there were no differences between the groups in caffeine consumption, or time since last use. There were no group differences in cigarettes use (ratio yes:no), and of those who reported smoking, there were no group differences in the number of cigarettes smoked per day, or the time since last use. No significant differences were found between the groups for the MAST, and AUQ score and Binge scores. Of the recreational drugs that participants reported using, there were no group differences in the numbers that reported use compared with those who did not, and of those who did report use, there were no group differences in the number of days used per month (data not shown).

4.3.3.2. Breath Alcohol Concentration

All breath alcohol levels were 0 at the beginning of the session. There was a significant difference between the groups after drink consumption (alcohol group = 0.42 mg/l BRAlc) [$F(2,37) = 170.57$, $p = 0.01$], and at the end of the session (alcohol group = 0.20 mg/l BRAlc) [$F(2,37) = 93.03$, $p = 0.01$].

Table 4.3. Participant means, ANOVA and chi-squared results of between group comparisons (standard deviations in parentheses).

	Group Mean			*Statistic*	Value	Degrees of Freedom	p value
	Control (n = 10)	**Placebo** (n = 15)	**Alcohol** (n = 15)				
Age (yrs)	22.2 (3.29)	21.4 (2.35)	21.4 (2.41)	ANOVA	0.35	2	0.71
Gender (Ratio male: female)	6:4	7:8	6:9	Chi-squared	1.75	2	0.42
Weight (kg)	73.03 (11.99)	67.72 (10.29)	65.08 (6.66)	ANOVA	2.08	2	0.14
Education (years)	16.5 (3.10)	16.07 (2.15)	15.73 (2.71)	ANOVA	0.26	2	0.78
Alcohol (units per week)	14.70 (10.26)	15.13 (11.06)	10.00 (5.49)	ANOVA	1.39	2	0.26
Caffeine (Ratio Yes:no)	6:4	9:6	14:1	Chi-squared	5.23	2	0.07
(cups per day)	3.33 (1.97)	2.44 (1.01)	1.93 (1.27)	ANOVA	2.24	2	0.13
(hours since use)	7.33 (9.11)	11.89 (11.91)	13.29 (16.69)	ANOVA	0.46	2	0.64
Cigarettes (Ratio yes:no)	1:9	2:13	2:13	Fisher's Exact	0.08	2	0.96
(per day)	5.00 (0)	3.00 (1.41)	2.50 (0.71)	ANOVA	1.72	2	0.37
(hours since use)	2.00 (0)	6.50 (7.78)	2.50 (2.12)	ANOVA	0.32	2	0.76
MAST Score	1.40 (1.17)	1.73 (1.28)	1.07 (0.59)	ANOVA	1.53	2	0.23
AUQ Score	38.68 (26.79)	42.43 (31.78)	35.39 (20.56)	ANOVA	0.26	2	0.77
Binge Score	26.08 (24.86)	24.49 (17.23)	24.72 (17.71)	ANOVA	0.02	2	0.98

4.3.3.3. Task Performance

A mixed ANOVA was conducted on the average reversal rate, with Group (Alcohol, Placebo and Control) as the between-subjects factor, and two within-subjects factors, Bias (Ambiguous, Light Bias, and Dark Bias), Illusion Type (Face-vase and Necker Cube), see Table 4.4 for the mean number of figure reversals reported and total viewing times. This analysis revealed no significant main effect of Group [$F(2,37) = 0.81$, $p = 0.45$]. The main effect of Bias was significant [$F(2,74) = 7.00$, $p = 0.01$], with more reversals reported for the ambiguous figures, followed by the light bias figures, with the least reversals reported for the dark bias figures Face-vase stimuli than for the Necker cube stimuli. However, there was no significant Group x Bias interaction [$F(4,74) = 0.43$, $p = 0.77$], see Figure 4.3. The main effect of Type was significant [$F(1,37) = 8.11$, $p = 0.01$], with more reversals reported for the Face-vase illusion, see Figure 4.3. There were no significant interactions for Type x Group [$F(2,37) = 0.34$, $p = 0.71$], Bias x Type [$F(2,74) = 1.80$, $p = 0.17$], or Bias x Type x Group [$F(4,74) = 2.11$, $p = 0.09$].

Table 4.4. Mean number of reversals and total viewing time (standard deviations in parentheses) for each group.

Experimental Task	**Control Group** (n = 10)		**Placebo Group** (n = 15)		**Alcohol Group** (n = 15)	
	Number of reversals	Total viewing time	Number of reversals	Total viewing time	Number of reversals	Total viewing time
Face Vase Illusion						
(Ambiguous Stimulus)	19.93 (12.87)	53.72 (4.49)	18.20 (8.98)	53.77 (7.23)	15.87 (7.80)	53.51 (8.27)
(Light Bias Stimulus)	17.80 (10.02)	50.17 (8.36)	15.90 (9.72)	55.36 (2.70)	13.93 (6.62)	54.49 (3.80)
(Dark Bias Stimulus)	14.70 (11.24)	49.87 (9.01)	14.70 (11.24)	53.69 (3.67)	9.87 (4.55)	51.36 (5.82)
Necker Cube Illusion						
(Ambiguous Stimulus)	14.13 (9.45)	54.33 (2.17)	15.70 (7.09)	51.10 (10.05)	11.87 (6.22)	54.34 (3.58)
(Light Bias Stimulus)	16.80 (13.40)	53.09 (4.93)	13.60 (6.24)	54.21 (3.98)	11.33 (4.69)	52.72 (7.47)
(Dark Bias Stimulus)	12.73 (9.22)	53.73 (4.34)	11.50 (3.27)	54.12 (3.93)	11.27 (7.11)	47.79 (14.49)

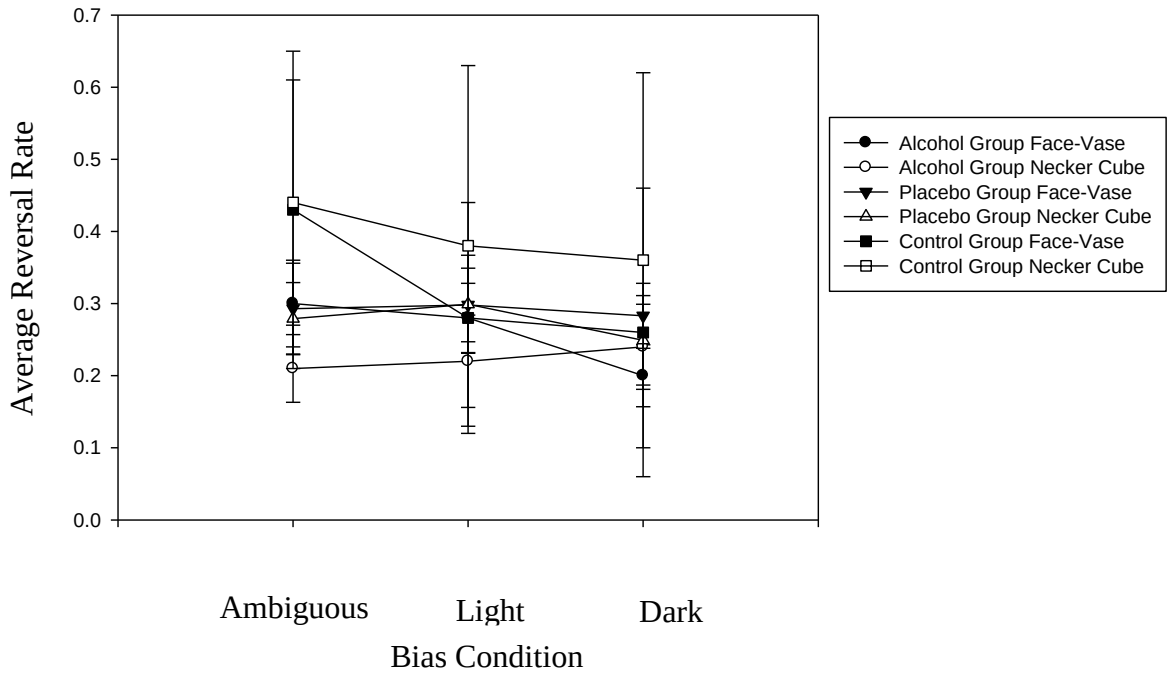


Figure 4.3. Group differences in average reversal rate for each condition depending upon the type of figure presented. Error bars represent Standard Errors of means.

4.3.4. DISCUSSION

The results of the present study show that, without the explicit instruction to reverse, alcohol has no effect on reversal rate and if anything tends to reduce it. This contrasts with the results of Study 2 in which alcohol increased reversal rate for biased versions of the face-vase figure. This suggests that a moderate dose of alcohol does not affect the number of figure reversals reported when the automatic control mechanisms are activated. Hence, an important factor influencing the effect of alcohol on ambiguous figure reversals is the conscious "set" of the observers. The results are consistent with previous reports that alcohol impairs controlled but not automatic attentional control mechanisms (Abroms et al., 2006; Fillmore & Vogel-Sprott, 2006; Holloway, 1995).

The results of the two studies presented so far show that the general effect of conscious control is as predicted. That is, reversal rates are higher when participants consciously attempt to make the figure reverse than under passive viewing conditions. However, the results are somewhat unexpected. It was predicted that under these conditions, alcohol would also increase reversal rate, but the effect would be to a lesser extent than in Study 2 as reversals were now reported under passive viewing conditions. However, the results of the present study show that, without explicit instruction to reverse, alcohol tends to reduce reversal rate below baseline.

The results of the present study provide reassurance that the placebo is an appropriate baseline and that expectations are not distorting the comparisons. This study included a second control group who did not receive any drinks at any stage of the study. Therefore, the participants in this group were aware from the outset that they had not

received an alcoholic beverage. This group was included to control for any expectancy effects that have sometimes been reported in other alcohol studies. It has been suggested that when participants think they are drinking alcohol, they are hypervigilant in an attempt to compensate for their anticipated poorer performance (Marczinski & Fillmore, 2005). However, the results presented here show that there were no group differences on any measure, suggesting that it is unlikely that the results of the placebo group are likely to be due to compensatory effects.

The studies so far have shown that the effects of alcohol depend upon instructions, upon the nature of the stimulus, and also upon the relative, and possibly the absolute, strengths of the two interpretations. The following study further investigates these complex interactions using a different type of bias that does not change the physical properties of the stimulus.

4.4. STUDY 4: IMAGE STABILISATION USING FIXATION CROSS: EFFECT OF ALCOHOL ON REVERSALS

4.4.1. INTRODUCTION

The results of Study 2 showed that the precise nature of the stimulus was important in studying effects of alcohol on the perception of ambiguous figures and used a biasing manipulation that was intended to favour one interpretation over the other. However, the results suggested that the absolute strength of the two interpretations might be important, as well as their relative strength due to changes in the luminance of the stimuli. The aim of the following study was similar to Study 2, but the reversal rates were manipulated in a different way that leaves the basic stimuli unchanged.

Studies have shown that reversal rate can be reduced when the locus of fixation is moved from the fovea to the periphery (Toppino, 2003; Georgiades & Harris, 1999). One explanation for this finding is that, as the fixation is moved towards the periphery, fewer features critical to both percepts fall within the same region of attention. The assumption here is that the fixation points and the centres of focal attention coincide (Gale & Findlay, 1983; Hoffman & Subramanian, 1995; Klein et al., 1992; Kowler, 1985; Posner, 1980). To examine the effect of locus of fixation in the effects of alcohol on perception of ambiguous figures, two figures were presented with one of three fixation point positions. Fixation could either be presented at the fovea, or moved increasingly toward the periphery. Because moving the point of fixation closer to the periphery has been shown to increasingly stabilise reversal rate, it is thought that the placebo group should report fewer reversals for more peripheral locations. Whereas, if

alcohol impairs the inhibitory mechanisms underlying the stabilisation of reversal rate then alcohol will undermine this effect.

4.4.2. METHOD

4.4.2.1. Participants

28 young social drinkers from the University of Birmingham with normal or corrected-to-normal vision participated in the study. There were two experimental groups, an alcohol group (6 male and 8 female; mean = 21.57 years, sd = 2.06, average self reported consumption of 10.00 units of alcohol per week), and a placebo (group 7 male and 7 female; mean = 22.71 years, sd = 2.37, average self reported consumption of 11.86 units of alcohol per week). Participants were told they were taking part in a study looking at the effects of alcohol on the perception of ambiguous figures, but were not told about different types of ambiguous figures or the effects of the bias on perception and the number of reversals usually reported in each case. Participants were the first to volunteer who met the criteria outlined in section 2.2.1 and were recruited through the Psychology departments' research participation scheme (see section 2.2).

4.4.2.2. Design

Participants were allocated pseudo-randomly to groups such that the groups were matched for age and gender. All Necker cube stimuli were presented together and Face-vase stimuli were presented together. The neutral figures were always presented first for each stimulus type, with the remaining stimuli presented randomly thereafter. The order of block presentation was fully counter balanced.

4.4.2.3. Inclusion and Exclusion criteria

Participants were excluded based on the criteria given in section 2.2.1.

4.4.2.4. Screening Tools

Details of the Lifestyle Questionnaire, Alcohol Use Questionnaire (AUQ; Mehrabian & Russell, 1978), Michigan Alcohol Screening Test (MAST; Selzer, 1971) that participants completed for this study are described in detail in section 2.3.

4.4.2.5. Alcohol administration

A detailed description of the alcohol administration is given in section 2.4.

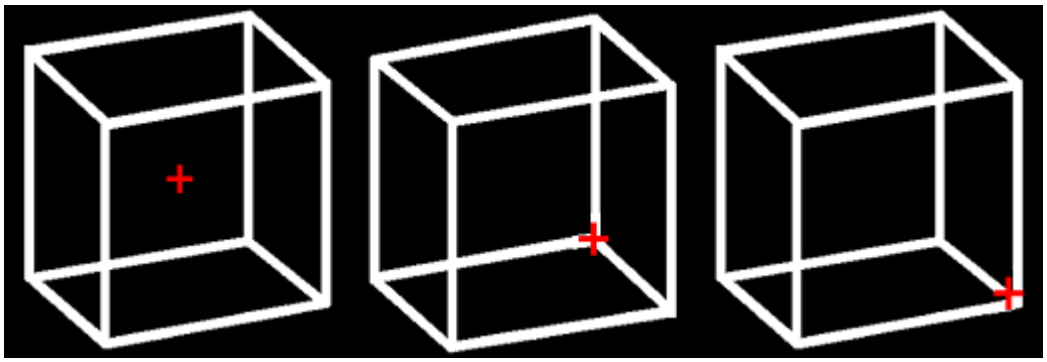
4.4.2.6. Materials and Tasks

The stimuli were the Necker cube (Necker, 1832) and the Face-vase illusion (Rubin, 1958). Three versions of each figure were used, which differed in the position of a red fixation cross. This was either positioned centrally, or moved increasingly towards the periphery.

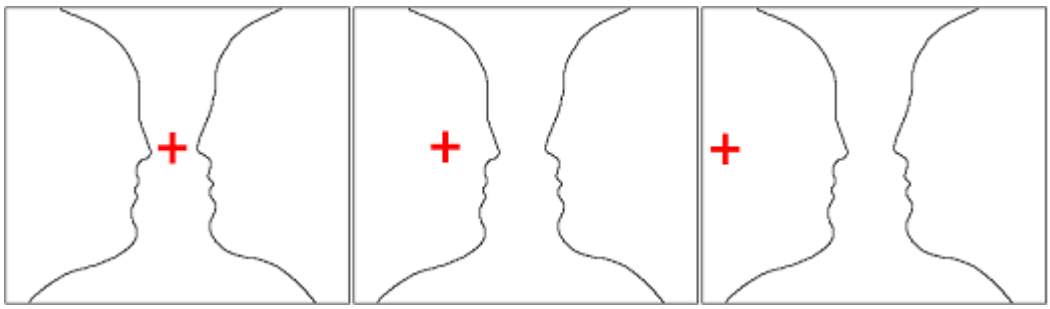
Necker Cube stimuli: For the Necker cube stimuli, the fixation cross in the neutral bias (e.g. 'Ambiguous' figure) was positioned centrally within the figure at a viewing angle of 0° (see Figure 4.4a). For the moderate bias, the fixation cross was located on the bottom right corner of the upper-most face of the cube, 1.9° (2 cm) from the centre of the figure (see Figure 4.4b). The fixation cross for the extreme bias was positioned on the bottom right corner of the lower-most face of the figure at a viewing angle of 4.3° (4.5 cm) from the centre of the figure (see Figure 4.4c). Stimuli were presented on a black background with the Necker cube presented as white lines measuring 8 x 8 cm.

Face-vase stimuli: For the Face-Vase stimuli, the fixation cross in the neutral bias was positioned centrally within the figure at a viewing angle of 0° (see Figure 4.4d). For the moderate bias, the fixation cross was located 1.9° (2 cm) to the left of the centre of the figure (see Figure 4.4e). The fixation cross in the extreme bias was located 3.8° (4 cm) to the left of the centre of the figure (see Figure 4.4f). The Face-vase stimuli were presented on a white background with black contour lines distinguishing the components of the face and the vase measuring approximately 9 x 9 cm.

The general procedure is outlined in section 2.6, the only difference being that participants were asked to keep their eyes fixated on the fixation cross at all times and to try not to move their eyes around the screen. Reversal rate was under volitional control, participants were asked to increase reversal rate by making as many perceptual reversals as possible within the 1-minute presentation time, but only to report a reversal when they were certain of a change in perspective.



(a) Neutral Bias *(b)* Moderate Bias *(c)* Extreme Bias



(d) Neutral Bias *(e)* Moderate Bias *(f)* Extreme Bias

Figure 4.4. The stimuli used for the experiment, including the two neutral stimuli used for both the Necker cube and Face-vase illusions (Figures 4.4a and 4.4d), the two moderate bias stimuli (Figures 4.4b and 4.4e), and the two extreme bias stimuli (Figures 4.4c and 4.4f).

4.4.2.7. Procedure

The procedure is outlined in section 2.6. Following the 10-minute rest period, participants completed the ambiguous figures tasks. After which, participants were breathalysed again.

4.4.2.8. Data Analysis

A mixed ANOVA was used to analyse group differences on the Ambiguous Figures task for the total number of reversals reported within one minute, with Group (Alcohol or Placebo group) as the between subjects factor, and Bias (neutral, moderate, and

extreme bias) as the within subjects factor. To control for individual differences in alternation rate, the data for each participant was normalised, see section 4.2.1.8.

Demographic information and questionnaire response analysis is described in section 2.7.

4.4.3. RESULTS

4.4.3.1. Demographics

Participant demographic and test results are summarised in Table 4.5. Independent t-tests and chi-squared tests revealed no age, gender, weight or education differences between the groups. There were no significant differences between the groups for units of alcohol per week. There were no group differences in caffeine use (ratio yes:no), and of those who reported caffeine consumption, there were no differences between the groups in caffeine consumption, or time since last use. There were no group differences in cigarettes use (ratio yes:no), and of those who reported smoking, there were no group differences in the number of cigarettes smoked per day, or the time since last use. No significant differences were found between the groups for the MAST, and AUQ score and Binge scores. Of the recreational drugs that participants reported using, there were no group differences in the numbers that reported use compared with those who did not, and of those who did report use, there were no group differences in the number of days used per month (data not shown).

4.4.3.2. Breath Alcohol Concentration

All breath alcohol levels were 0 at the beginning of the session. There was a significant difference between the groups after drink consumption (alcohol group = 0.33 mg/l BRAlc) [$t(26) = 8.21$, $p = 0.01$], and at the end of the session (alcohol group = 0.16 mg/l BRAlc) [$t(26) = 7.39$, $p = 0.01$].

Table 4.5. Participant means, t-test and chi-squared results of between group comparisons (standard deviations in parentheses).

	Mean		Statistic	Value	Degrees of Freedom	*p* value
	Placebo Group (n = 14)	Alcohol Group (n = 14)				
Age (years)	22.71 (2.37)	21.57 (2.06)	t-test	-1.36	26	0.19
Gender (male:female)	7:7	6:8	Chi-Squared	0.14	1	0.71
Weight (kg)	67.37 (8.59)	64.96 (8.64)	t-test	-0.74	26	0.47
Education (years)	16.14 (3.11)	16.21 (2.08)	t-test	0.07	26	0.94
Alcohol (units per week)	11.86 (3.61)	10.00 (2.48)	t-test	-1.59	26	0.12
Caffeine (ratio yes:no)	10:4	7:7	Fisher's Exact	n/a	n/a	0.44
(Cups per day)	2.70 (1.34)	2.00 (1.00)	t-test	-1.17	15	0.26
(Hours since use)	6.92 (6.51)	18.79 (16.44)	t-test	2.08	15	0.06
Cigarettes (ratio yes:no)	1:13	0:14	Fisher's Exact	n/a	n/a	1.00
(per day)	3.00 (0)	n/a	n/a	n/a	n/a	n/a
(hours since use)	2 (0)	n/a	n/a	n/a	n/a	n/a
MAST	2.29 (1.38)	1.64 (1.39)	t-test	-1.23	26	0.23
AUQ	41.80 (23.52)	29.25 (16.96)	t-test	-1.62	26	0.12
BINGE	29.16 (20.07)	18.39 (12.79)	t-test	-1.69	26	0.10

4.4.3.3. Task Performance

A mixed ANOVA was conducted on the average reversal rate, with Group (Alcohol and Placebo) as the between-subjects factor, and two within-subjects factors, Bias (Neutral, Moderate, and Extreme Bias), Type (Face-vase and Necker Cube), see Table 4.6 for the mean number of figure reversals reported and total viewing times. This analysis did not reveal a significant main effect of Group [$F(1,26) = 1.34$, $p = 0.26$]. There was a significant effect of Bias [$F(2,52) = 4.10$, $p = 0.05$], with the most figure reversals reported for the neutral figure, followed by the moderate biased figure, and the least figure reversals reported for the extreme biased stimulus. However, there was no significant Group by Bias interaction [$F(2,52) = 0.16$, $p = 0.74$], see Figure 4.5. The main effect of Type was not significant [$F(1,26) = 0.20$, $p = 0.66$], nor were the Type by Group [$F(1,26) = 0.22$, $p = 0.64$], and the Bias by Type by Group [$F(2,52) = 0.76$, $p = 0.40$] interactions. However, the Bias by Type interaction was significant [$F(2,52) = 4.21$, $p = 0.05$]. Post hoc tests were used to explore the source of this interaction. There was a significant difference in reversals between conditions for the Face-vase stimuli, e.g. neutral and moderate bias conditions [$t(27) = 2.84$, $p = 0.01$], moderate and extreme bias conditions [$t(27) = 3.11$, $p = 0.01$], and neutral and extreme bias conditions [$t(27) = 4.45$, $p = 0.01$]. A similar analysis for the Necker cube stimuli did not show significant differences in the number of reversals reported between the conditions, e.g. neutral and moderate bias conditions [$t(27) = -0.97$, $p = 0.34$], moderate and extreme bias conditions [$t(27) = 0.98$, $p = 0.34$], and neutral and extreme bias conditions [$t(27) = -0.54$, $p = 0.59$], see Figure 4.5.

Table 4.6. Means and standard deviations (in parentheses) of scores on all tasks for each group.

Experimental Task	**Placebo Group** (n = 14)		**Alcohol Group** (n = 14)	
	Number of reversals	Total viewing time	Number of reversals	Total viewing time
Face Vase Illusion				
(Neutral Bias)	43.21 (35.89)	54.75 (2.35)	27.50 (15.24)	52.47 (14.84)
(Moderate Bias)	30.71 (20.71)	55.07 (2.44)	23.36 (9.43)	55.15 (3.23)
(Extreme Bias)	26.14 (22.59)	52.75 (5.58)	19.00 (7.99)	54.40 (1.69)
Necker Cube Illusion				
(Neutral Bias)	28.86 (28.04)	52.61 (4.70)	19.57 (13.78)	54.78 (5.24)
(Moderate Bias)	31.29 (37.13)	52.84 (6.26)	20.21 (14.84)	53.64 (3.14)
(Extreme Bias)	26.79 (37.81)	50.85 (7.99)	20.43 (18.65)	54.10 (3.09)

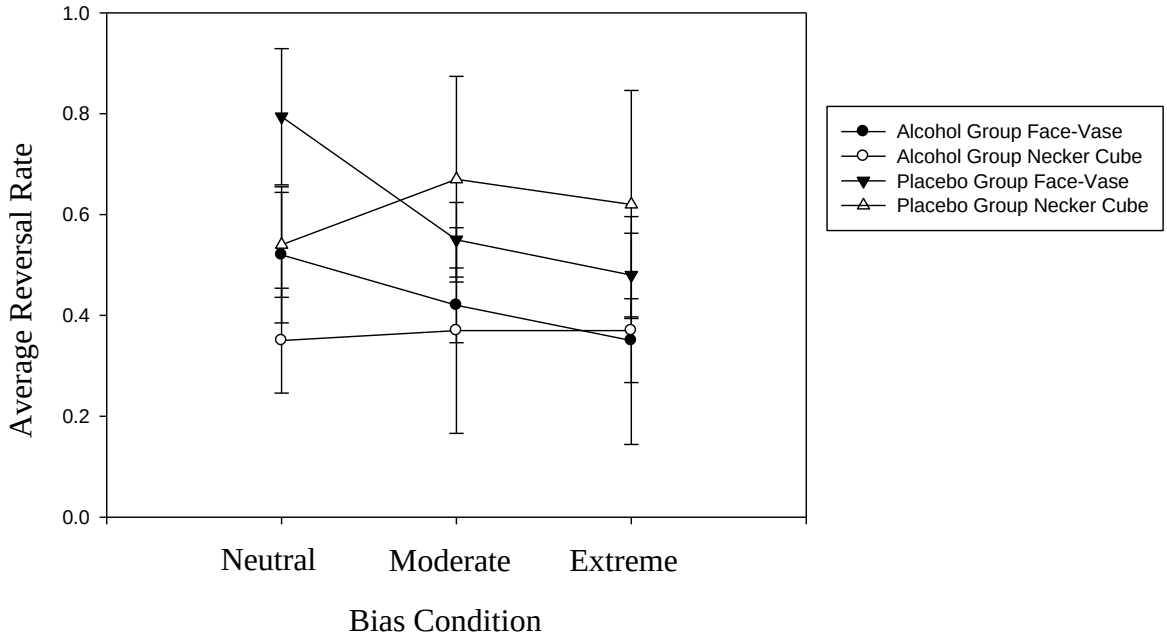


Figure 4.5. Group differences in average reversal rate for each condition depending upon the type of figure presented. Error bars represent Standard Errors.

4.4.4. DISCUSSION

The results of the present study are more similar to those found for Study 3 than those found in Study 2 and provide further support for the idea that the effects of alcohol on perception of ambiguous figures depends crucially on the specific configuration of the stimulus. Adding a fixation point had different effects on reversal rate for the face-vase and Necker cube illusions but in each case, the effect of alcohol is to tend to reduce reversal rate and, for both figure types, the pattern of reversals after alcohol is no different from the placebo group. This is somewhat unexpected result given that the removal of inhibition ought to increase reversals.

There is some support from the present results that peripheral fixation reduces reversal rate for the Face-vase illusion, but the reverse seems to be the case for the Necker cube. Once again, a detailed consideration of the stimuli may provide possible reasons as to why this may be. For the Necker cube illusion, the changes in fixation not only move the stimulus into the retinal periphery but also bring important features of the stimulus onto the fovea. Both the biased conditions direct fixation onto vertices that seem to be important for controlling the interpretation; reversal of the interpretation of either of these local vertices will lead to reversal of the global interpretation. This might account for the similar reversal rate across the conditions seen with the Necker cube. Similarly, Georgiades and Harris (1999) have shown that when features important for both percepts fall within the same region of attention, and so are both readily available to attention, reversal rate is not stabilised. Whereas, the fixation points used for the Face-vase illusion favour one particular interpretation as the fixation point is moved further into the periphery. Consequently, the bias used for the face-vase illusion has the effect of stabilising reversal rate. It has been shown that the ability to attend to certain features

of an ambiguous figure can affect the number of figure reversals that are reported. For instance, Toppino (2003) has shown that certain features within an ambiguous figure favour one interpretation more than the other. When stimulus manipulations were applied to these features, they helped to disambiguate the figure and increase the likelihood that attentional resources will be directed towards them (Toppino, 2003).

It is possible that the presence of a fixation point may simply reduce eye movements. Studies have shown that figure reversals are correlated with changes in fixation (Ellis & Stark, 1978), with reversals typically occurring following changes in eye position (Toppino, 2003). If fixation is maintained at one location, thus preventing eye movements, then it seems reasonable to assume that reversal rate would fall. It is possible then that one effect of alcohol might be to encourage exploratory eye movements, which, depending on the precise nature of the stimulus, tend to increase reversals (Study 2). The inclusion of a fixation point in the present study might be to prevent exploratory eye movements resulting in a stabilising effect on reversal rate.

The results of the studies presented so far have shown that alcohol produces an increase in reversal rate only for the Face-Vase figure. Furthermore, the effect is restricted to a specific stimulus configuration and is only evident when participants are encouraged consciously to increase reversal rate. Even under these specific conditions, the effect of alcohol is to increase the overall reversal rate above baseline, rather than simply reversing the reduction of reversals expected when bias is introduced. In all other cases, alcohol has no effect on reversal rate or tends to reduce the reversal rate, which is clearly contrary to a simple account based on reduced inhibition. The interactions

between the various factors are more complex than initially thought. However, the only condition where alcohol significantly affected reversal rate was when participants were free to move attention around the stimulus. The aim of the following study will be to further explore the role of exploratory eye movements in figure reversals and the effects of alcohol on the inhibitory mechanisms involved in this process.

4.5. STUDY 5: IMAGE STABILISATION USING INTERMITTENT PRESENTATION: EFFECT OF ALCOHOL ON REVERSALS.

4.5.1. INTRODUCTION

One possibility emerging from a comparison of Studies 2 and 4 is that observers may spontaneously control the rate of reversal by actively moving their eyes. Research has shown that reversals tend to occur following eye movements or blinks (Ellis & Stark, 1978; Kawabata et al., 1978). When these are under the conscious control of the observer, and if the stimulus is appropriate, the reversal rate increases. It is suggested that each new fixation resets the reversal process (Ellis & Stark, 1978; Kawabata et al., 1978). This raises the possibility that one effect of alcohol may be indirect, exerting its influence by increasing or decreasing the tendency to explore the stimulus under conditions of bias, and that the effects of alcohol on figure reversals observed in Study 4 were because eye movements were reduced by the addition of a fixation cross to the stimuli.

The aim of the present study was to use a different method of stabilising perception of ambiguous figures that does not involve directing fixation. Under normal viewing conditions, intermittent presentation of an ambiguous figure has been shown to reduce the number of reversals reported (Chen & He, 2004; Blake et al., 2003; Leopold et al., 2002). Under continuous viewing conditions, reversal rates increase over time, possibly due to changes in the balance of excitation and inhibition, with the inhibitory influence becoming relatively weaker as viewing continues, thus facilitating reversal. Intermittent stimulus presentation disrupts these processes and reduces reversal rates. Under conditions of intermittent presentation participants are free to make voluntary eye

movements but the outcome of the manipulation is to reduce reversals as in Study 4. Hence, comparison of Studies 3 and 4 will provide information on the importance of eye movements in the effects of alcohol on reversal.

4.5.2. METHOD

4.5.2.1. Participants

22 young social drinkers from the University of Birmingham with normal or corrected-to-normal vision participated in the study. There were two experimental groups, an alcohol group (5 male and 6 female; mean = 23.27 years, sd = 2.65, average self reported consumption of 12.00 units of alcohol per week), and a placebo group (6 male and 5 female; mean = 24.18 years, sd = 2.93, average self reported consumption of 10.27 units of alcohol per week). Participants were told they were taking part in a study looking at the effects of alcohol on the perception of ambiguous figures, but were not told about different types of ambiguous figures or the effects of intermittent viewing on perception and the number of reversals usually reported in each case. Participants were the first to volunteer who met the criteria outlined in section 2.2.1 and were recruited through the Psychology departments' research participation scheme (see section 2.2).

4.5.2.2. Design

Participants were allocated pseudo-randomly to groups such that the groups were matched for age and gender. All Necker cube stimuli were presented together and Face-vase stimuli were presented together. The control condition was always presented first for each stimulus type, with the remaining conditions presented randomly thereafter. The order of block presentation was fully counter balanced.

4.5.2.3. Inclusion and Exclusion criteria

Participants were excluded based on the criteria given in section 2.2.1.

4.5.2.4. Screening Tools

Details of the Lifestyle Questionnaire, Alcohol Use Questionnaire (AUQ; Mehrabian & Russell, 1978), Michigan Alcohol Screening Test (MAST; Selzer, 1971) that participants completed for this study are described in detail in section 2.3.

4.5.2.5. Alcohol administration

A detailed description of the alcohol administration is given in section 2.4.

4.5.2.6. Materials and Tasks

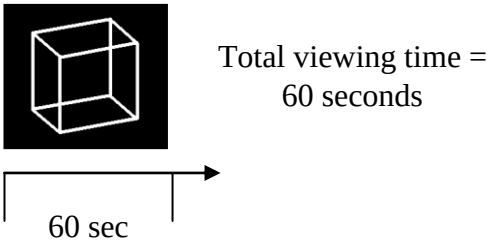
The stimuli were the Necker cube (Necker, 1832) and the Face-vase illusion (Rubin, 1958). Each ambiguous figure was presented three times, with each presentation differing in the interstimulus interval (ISI).

Necker Cube stimuli: The stimulus was the ambiguous figure used in Study 2, see Figure 4.1a.

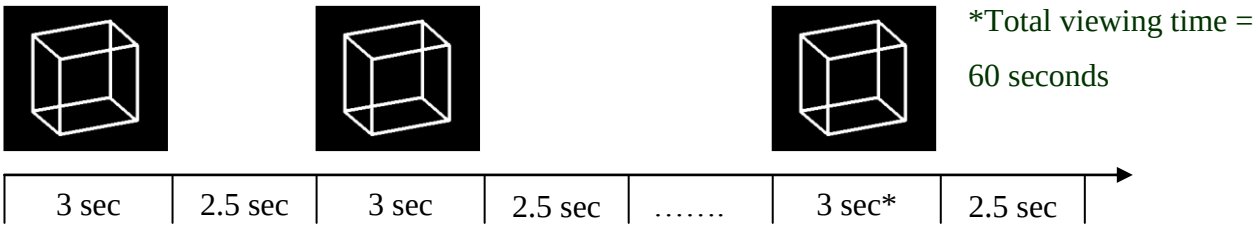
Face-vase stimuli: The stimulus was the ambiguous figure from Study 2, see Figure 4.1d.

For the control condition of each ambiguous figure, the ISI was set to 0 seconds, therefore the illusion was continuously present on screen for 1 minute. For the first of the biased conditions, the illusion was presented and visible on screen for 3 seconds, followed by an ISI of 2.5 seconds where the illusion was not visible. In the second of the biased conditions, the illusion was also presented for 3 seconds, but followed by an

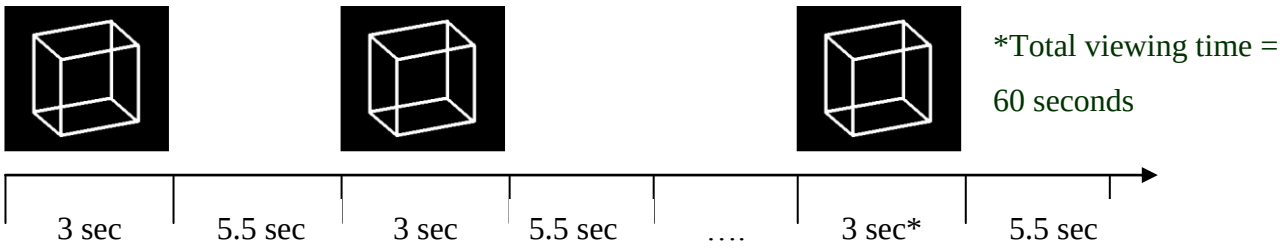
ISI of 5.5 seconds. Each ambiguous figure was visible on screen for a total of 1 minute and the total presentation time where the stimulus was visible on screen in the biased conditions equalled 1 minute, see Figure 4.6 for the task sequence timeline. Keeping the time the stimulus was present on screen equal across the three conditions enabled comparisons to be made with regards to the number of reversals reported in each condition. A 1 minute presentation time was chosen as previous studies have shown that the number of figure reversals increases up to 1 minute, after which the reversal rate reaches a steady rate (e.g. Brown, 1955; Cornwell, 1976; Fisichelli, 1947; Köhler, 1940; Philip & Fisichelli, 1945; Price, 1969a; Virsu, 1975). Reversal rate was under volitional control, participants were asked to increase reversal rate by making as many perceptual reversals as possible within the 1-minute presentation time, but only to report a reversal when the figure was visible on screen and when they were certain of a change in perspective. Participants made their responses following the same procedure outlined in section 4.2.1.6.



(a) 0 second ISI condition



(b) 2.5 second ISI condition



(c) 5.5 second ISI condition

Figure 4.6. Task sequence timeline for the intermittent presentation ambiguous figures task, including the 0 ISI condition (Figure 4.6a), the 2.5 ISI condition (Figure 4.6b), and the 5.5 ISI condition (Figure 4.6c).

4.5.2.7. Procedure

The general procedure is described in section 2.6. Following the 10-minute rest period, participants completed ambiguous figures tasks. After which, participants were breathalysed again.

4.5.2.8. Data Analysis

A mixed ANOVA was used to analyse group differences on the Ambiguous Figures task for the total number of reversals reported within one minute, with Group (Alcohol or Placebo group) as the between subjects factor, and two within subjects factors, ISI (O, 2.5, and 5.5 seconds) and Type (Necker cube or Face-vase). To control for individual differences in alternation rate and ISI, the data for each participant was normalised, see section 4.2.1.8.

Demographic information and questionnaire response analysis is described in section 2.7.

4.5.3. RESULTS

4.5.3.1. Demographics

Participant demographic and test results are summarised in Table 4.7. Independent t-tests and chi-squared tests revealed no age, gender, weight or education differences between the groups. There were no significant differences between the groups for units of alcohol per week. There were no group differences in caffeine use (ratio yes:no), and of those who reported caffeine consumption, there were no differences between the groups in caffeine consumption, or time since last use. There were no group differences in cigarettes use (ratio yes:no), and of those who reported smoking, there were no group differences in the number of cigarettes smoked per day, or the time since last use. No significant differences were found between the groups for the MAST, and AUQ score and Binge scores. Of the recreational drugs that participants reported using, there were no group differences in the numbers that reported use compared with those who did not, and of those who did report use, there were no group differences in the number of days used per month (data not shown).

4.5.3.2. Breath Alcohol Concentration

All breath alcohol levels were 0 at the beginning of the session. There was a significant difference between the groups after drink consumption (alcohol group = 0.38 mg/l BRAlc) [$t(22) = 13.66$, $p = 0.01$], and at the end of the session (alcohol group = 0.24 mg/l BRAlc) [$t(22) = 5,81$, $p = 0.01$].

Table 4.7. Participant means, t-test and chi-squared results of between group comparisons (standard deviations in parentheses).

	Mean		Statistic	Value	Degrees of Freedom	*p* value
	Placebo Group (n = 11)	Alcohol Group (n = 11)				
Age (years)	24.18 (2.93)	23.27 (2.65)	t-test	-0.76	20	0.45
Gender (male:female)	6:5	5:6	Chi-Squared	0.18	1	0.67
Weight (kg)	72.99 (17.48)	66.56 (11.68)	t-test	-1.01	20	0.32
Education (years)	17.36 (2.16)	17.36 (2.34)	t-test	0	20	1.00
Alcohol (units per week)	10.27 (3.47)	12.00 (5.31)	t-test	0.90	20	0.38
Caffeine (ratio yes:no)	8:3	6:5	Fisher's Exact	n/a	n/a	0.66
(Cups per day)	3.75 (1.91)	2.83 (0.98)	t-test	-1.07	12	0.31
(Hours since use)	3.50 (2.56)	2.00 (0.63)	t-test	-1.39	12	0.19
Cigarettes (ratio yes:no)	2:9	3:8	Fisher's Exact	n/a	n/a	1.00
(per day)	5.00 (0)	7.33 (6.81)	t-test	0.46	3	0.68
(hours since use)	25.00 (32.53)	16.00 (17.78)	t-test	-0.42	3	0.71
MAST	1.55 (1.29)	1.45 (1.37)	t-test	-0.16	20	0.87
AUQ	19.96 (8.81)	26.64 (21.88)	t-test	0.94	20	0.36
BINGE	12.96 (7.21)	16.36 (17.90)	t-test	0.58	20	0.57

4.5.3.3. Task Performance

A mixed ANOVA was conducted on the average reversal rate, with Group (Alcohol and Placebo) as the between-subjects factor, and two within-subjects factors, ISI (0, 2.5, and 5.5 seconds), and Type (Face-vase and Necker Cube), see Table 4.8 for the mean number of figure reversals reported and the total viewing times. This analysis revealed no significant main effect of Group [$F(1,20) = 0.10$, $p = 0.75$]. There was a significant effect of Bias [$F(2,40) = 25.36$, $p = 0.01$], with the most figure reversals reported for the 0 second ISI condition, followed by the 2.5 second ISI, and the least figure reversals reported for the 5.5 second ISI. However, there was no significant Group by Bias interaction [$F(2,40) = 0.21$, $p = 0.81$], see Figure 4.7. The main effect of Type was not significant [$F(1,20) = 1.11$, $p = 0.31$], nor were the Type by Group [$F(1,20) = 0.17$, $p = 0.69$] or the Bias by Type by Group [$F(2,40) = 1.29$, $p = 0.29$] interactions. However, the Bias by Type interaction was significant [$F(2,40) = 7.73$, $p = 0.02$]. Post hoc tests to explore the source of this interaction revealed that, for the Face-vase illusion, there was a significant difference in reversals between the 0 ISI and 2.5 ISI conditions [$t(21) = 8.38$, $p = 0.01$], 2.5 ISI and 5.5 ISI conditions $(21) = 2.63$, $p = 0.02$], and between the 0 ISI and 5.5 ISI conditions [$t(21) = 11.25$, $p = 0.01$]. For the Necker Cube illusion, there was a significant difference in reversals between the 0 ISI and 2.5 ISI conditions [$t(21) = 17.83$, $p = 0.01$], and 0 ISI and 5.5 ISI conditions [$t(21) = 11.63$, $p = 0.01$], but not for 2.5 ISI and 5.5 ISI conditions [$t(21) = 0.80$, $p = 0.43$].

Table 4.8. Means and standard deviations (in parentheses) of scores on all tasks for each group.

	Placebo Group (n = 11)		**Alcohol Group** (n = 11)	
	Number of reversals	Total viewing time	Number of reversals	Total viewing time
Face Vase Illusion				
(0 ISI)	22.36 (14.03)	51.85 (11.38)	21.00 (9.48)	56.63 (5.06)
(2.5 ISI)	14.45 (16.62)	32.77 (11.10)	14.64 (9.10)	35.39 (10.13)
(5.5 ISI)	7.64 (7.41)	28.08 (8.57)	9.18 (7.51)	32.19 (7.70)
Necker Cube Illusion				
(0 ISI)	17.55 (12.75)	55.69 (6.12)	15.64 (6.31)	56.65 (3.89)
(2.5 ISI)	9.55 (10.63)	26.28 (8.95)	9.55 (6.07)	26.66 (5.14)
(5.5 ISI)	12.18 (12.89)	26.76 (13.29)	7.91 (8.07)	23.15 (10.70)

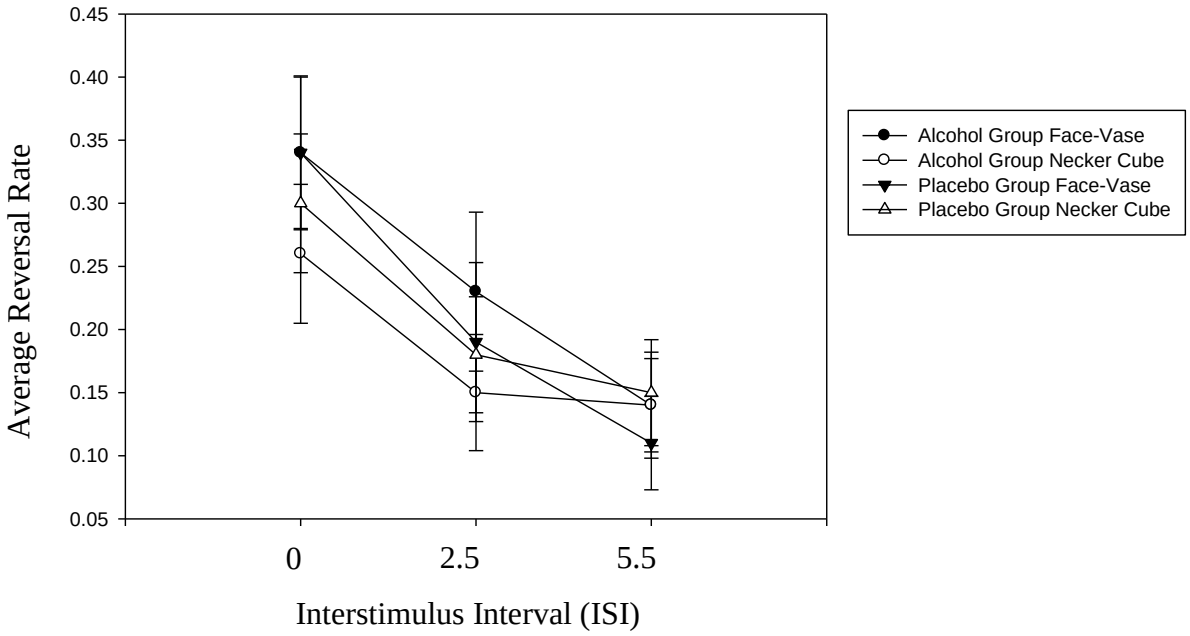


Figure 4.7. Group differences in average reversal rate for each condition depending upon the type of figure presented. Error bars represent Standard Errors.

4.5.4. DISCUSSION

The results from the present study confirm that intermittent presentation reduces reversal rate (Chen & He, 2004; Blake et al., 2003; Leopold et al., 2002) and in the present study this was true for both the alcohol and placebo groups. The implication is that reversal rate tends to increase during continuous presentation suggesting that the balance of excitation and inhibition changes over time, with the inhibitory influence becoming relatively weaker. The results show that alcohol has no systematic effect on reversal rates and, consequently, that it presumably does not change the balance of excitation and inhibition to further weaken inhibition, at least under the stimulus conditions investigated here. Furthermore, it seems that the lack of effect of alcohol on reversal rate in Study 4 is unlikely to be explained by the effect of the fixation cross on eye movements because under conditions in which eye movements could be freely made, no effect of alcohol on reversal rate was observed. It remains possible, of course, that different presentation regimes might have produced different effects. For example, they may more closely mimic the regime achieved by allowing subjects to control their eye movements to achieve a particular balance (Study 2). However, a more systematic study of different presentation regimes does not look particularly promising, given the absence of any alcohol effect in the current study.

4.6. GENERAL DISCUSSION

The results of the studies presented in this chapter show that alcohol does have an effect on figure reversals, but the effect is not as simple as might be expected. Instead, the effect of alcohol appears to depend on conscious “set” of the observer (Study 2), and upon the precise nature of the stimulus. A possibility that is emerging is that alcohol has

an effect on perception of ambiguous figure under conditions where absolute saliency of the representations is altered. The results of Study 2 show that alcohol resulted in an increase in the ability to voluntarily reverse an ambiguous figure. However, the effect of alcohol was not straightforward because there was no effect of alcohol when viewing the ambiguous version of the face vase illusion. In addition, the effect was seen predominantly for the face vase illusion and not for the Necker Cube illusion, suggesting that the Necker cube illusion may be less susceptible to reversals overall. Similar differences in reversal rate between figure types was reported by Strüber and Stadler (1999) and was taken to suggest that cognitive resources are able to act more effectively on ambiguous figures with semantically meaningful content rather than those requiring a simple realignment of perspective. Study 3 looked at the extent to which conscious control is important for the effects of alcohol on reversal rate. The results showed that, without the explicit instruction to reverse, alcohol had no effect on reversal rate and if anything tended to reduce it. Study 4 used a different type of bias that did not change the physical properties of the stimulus, and the results were more similar to those found for Study 3 than those found in Study 2 and provided further support for the idea that the effects of alcohol on perception of ambiguous figures depends crucially on the specific configuration of the stimulus. The fixation bias had different effects on reversal rate for the face-vase and Necker cube illusions but in each case, the effect of alcohol is the same. For both figure types, the pattern of reversals after alcohol is no different from the placebo group. Study 5 explored the role of exploratory eye movements in figure reversals and the effects of alcohol on the inhibitory mechanisms involved in this process. The results show that alcohol had no systematic effect on reversal rates and, consequently, that it presumably does not change

the balance of excitation and inhibition to further weaken inhibition, at least under the stimulus conditions investigated. Furthermore, it suggests that the lack of effect of alcohol on reversal rate in Study 4 is unlikely to be explained by the effect of the fixation cross on eye movements because under conditions in which eye movements could be freely made no effect of alcohol on reversal rate was observed.

These studies show that the effects of alcohol are observed only under certain conditions. This includes both the absolute as well as the relative strengths of the two interpretations of the figure. The effects of alcohol upon figure reversals therefore seems to depend on whether the stimulus contains obvious local regions that directly determine the absolute interpretation. Specifically, whether these local regions affect the overall meaning in an either/or situation, rather than simply changing the relative meaning incrementally also seems to be crucial. Similarly, the effect also seems dependent on whether the observer directs attention to these local regions, either internally by attentional focus (Study 2), or externally by appropriate fixation (Study 4).

The following chapter will try an alternative approach in an attempt to tease apart all of the complex interacting factors that have emerged from the studies presented so far in this thesis.

CHAPTER 5:

IMAGE STABILISATION USING PRIMING: EFFECT OF ALCOHOL ON REVERSALS

5.1. INTRODUCTION

The results of the studies presented in the previous chapter show that the mechanisms underlying figure reversals are affected by a moderate dose of alcohol when reversals are under conscious control. Furthermore, when biasing manipulations change local feature information (Study 4) or the image is presented intermittently (Study 5) alcohol does not affect reversals. Whereas biasing that increases the contrast between the two interpretations reveals an alcohol-induced impairment in performance (Study 2). The aim of this chapter will be to explore the possibility that alcohol impairs reversals when the absolute salience of the figure is altered. To do this, priming stimuli will be presented before the ambiguous figure to examine its effect on the absolute interpretation of the figure, revealed through the initial interpretation of the figure as well as changes in reversal rate.

The effects of priming and cognitive set have previously been explored using ambiguous figures and Helmholtz (1962) suggested that expectation plays an important role in the perception of ambiguous figures. Furthermore, numerous studies have shown that brief presentations of an unambiguous version of an ambiguous figure can prime observers into perceiving the subsequently presented ambiguous figure in the same configuration as the unambiguous previewed figure (Botwinick, 1961; Bugelski & Alampay, 1961; Fisher, 1967; Leeper, 1935; Long et al., 1992; Long & Moran, 2007).

This positive-bias effect is consistent with ideas of cognitive set effects whereby participants perceive the ambiguous figure to be in the interpretation for which they were primed by the prior exposure. For example, Bruner and Minturn (1955) demonstrated that perception of an ambiguous figure could be altered by the context in which it was presented; the figure *13* could be seen either as the letter *B* or the numbers *1* and *3*, depending on whether it was embedded within a sequence of numbers or letters. Taken together, these studies provide evidence that prior presentation of primes can influence the absolute salience of ambiguous figures.

Collectively, these studies demonstrate that the presentation of visually, or categorically, or contextually relevant information can prime the perception of one or the other interpretation of an ambiguous figure. Such factors reflect the influence of higher order cognitive processes on figure reversals. Although there is existing research to show that cognitive processes are affected by alcohol, no study has yet looked at the effect of priming on reversals after alcohol consumption. However, there is some existing literature from visual attention and negative priming studies that suggest that alcohol may impair similar processes responsible for the priming effects observed using ambiguous figures.

Support for the suggestion that the increased reversal rate reported by the alcohol group in Study 2 being due to impairment in the processing of absolute saliency comes from the observation in visual attention research that different types of biasing are processed in different areas of the brain (Treisman & Sato, 1990; Wolfe, 1994; Itti & Koch, 2000; Beck & Kastner, 2009). Changes in the absolute salience of a stimulus are computed in

the parietal and pre-frontal areas (Balan & Gottlieb, 2006; Goldberg et al., 2006; Buschman & Miller, 2007; Moore et al., 2003; Thompson & Bichot, 2005), whereas changes to local feature information are computed in the primary visual cortex (Treisman & Sato, 1990; Wolfe, 1994; Itti & Koch, 2000). Assuming that absolute salience refers to information affecting the overall meaning, then it might be that the shading bias added to the Face-vase figure in Study 2 changes the absolute salience of the figure and is therefore computed within the parietal and pre-frontal areas of the brain. Whereas, the addition of fixation (Study 4) and prevention of satiation (Study 5) might provide changes in local features of the ambiguous figure only and therefore be computed within the primary visual cortex. As the parietal and prefrontal areas of the brain are known to be affected by moderate doses of alcohol (Kähkönen et al., 2001; Volkowa et al., 2008), this might be why there are a different pattern of results depending upon the type of biasing manipulation used in the previous chapter.

Studies have shown that a moderate dose of alcohol can suppress negative priming (Fillmore et al., 2000a). Under normal circumstances, residual inhibition from previous trials affects the time taken to respond to subsequent trials. However, because alcohol impairs this inhibitory mechanism, preventing the distracter attribute from being inhibited, there is no residual inhibition to overcome, and so the reaction time cost is not found. It is possible that the inhibitory mechanism responsible for the time cost shown in negative priming might also underlie the effects revealed when an unambiguous version of an ambiguous figure is presented before the ambiguous figure. Supposing that the same inhibitory mechanism is responsible for suppressing the alternate interpretation of an ambiguous figure, it is possible that the residual inhibition will

affect subsequent figure reversals, not just the initial interpretation of the figure. As the residual inhibition responsible for suppressing the alternate interpretation takes time to overcome, reversal rate might show a similar time cost similar to that observed in negative priming. However, as alcohol is known to suppress negative priming, then it is possible that there will be no residual inhibition to overcome, and so reversal rate will not be affected by the prior presentation of a prime. Additionally, because alcohol may impair the processing of absolute saliency information, it is likely that the presentation of the prime will not bias observers into perceiving the subsequently presented ambiguous figure in the same configuration.

In order to test this, the following study will present semantic and neutral primes before the presentation of an ambiguous figure. The ambiguous figure used will be the Face-vase illusion (Rubin, 1958), and so semantically meaningful primes associated with this figure will be the prior presentation of either an image of a face or the image of a vase. Neutral primes will be presented that bear no relation to the Face-vase figure, and their prior presentation should have no effect on figure reversals. It is predicted that, when presented with semantically meaningful primes, the placebo group will be more likely to report their initial interpretation to be in the same configuration as the prime compared with the alcohol group. In terms of the number of reversals reported, it is predicted that the prior presentation of semantic primes will reduce the number of reversals reported by the placebo group during the early stages of the test phase, but not for the alcohol group. Whereas, the prior presentation of neutral primes will not affect the number of figure reversals for either group.

5.2. METHOD

5.2.1. Participants

30 young social drinkers from the University of Birmingham with normal or corrected-to-normal vision participated in the study. There were two experimental groups, an alcohol group (7 male and 8 female; mean = 18.87 years, SD = 0.64, average self reported consumption of 19.00 units of alcohol per week), and a placebo group (9 male and 6 female; mean = 19.60 years, SD = 3.00, average self reported consumption of 16.47 units of alcohol per week). Participants were told they were taking part in a study to look at the effects of alcohol on the perception of ambiguous figures. They were not told about the effects of priming on perception, or the number of reversals usually reported. Participants were the first to volunteer who met the criteria outlined in section 2.2.1 and were recruited through the Psychology departments' research participation scheme (see section 2.2).

5.2.2. Design

Participants were allocated pseudo-randomly to groups such that the groups were matched for age and gender. The study consisted of 20 trials, with the presentation of the different priming stimuli pre-randomised and fully counter balanced.

5.2.3. Inclusion and Exclusion criteria

Participants were excluded based on the criteria given in section 2.2.1.

5.2.4. Screening Tools

Details of the Lifestyle Questionnaire, Alcohol Use Questionnaire (AUQ; Mehrabian & Russell, 1978), Michigan Alcohol Screening Test (MAST; Selzer, 1971) that participants completed for this study are described in detail in section 2.3.

5.2.5. Alcohol administration

A detailed description of the alcohol administration is given in section 2.4.

5.2.6. Materials and Tasks

The ambiguous figure was the Face-vase illusion (Rubin, 1958). In addition, two types of priming stimuli were used which either had semantic meaning for the Face-vase illusion, or had a neutral meaning for the Face-vase illusion.

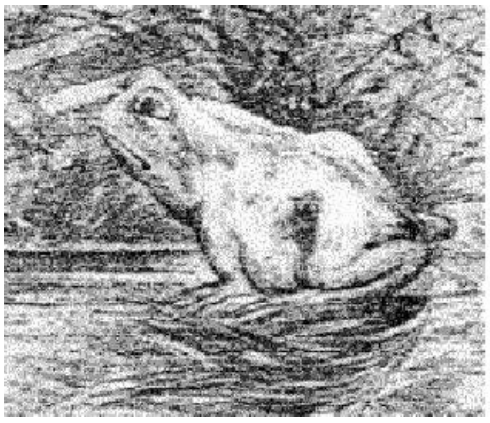
Face-vase stimuli: The Face-vase stimuli used in the current study was identical to the ambiguous version used in Study 2, see section 4.2.1.6 for details.

Semantic priming stimuli. A profile image of two faces looking at each other (http://www.flickr.com/photos/85519425@N00/2562292069/) served as the semantic prime for the two faces interpretation of the ambiguous figure, see Figure 5.1a. The image was presented centrally at a viewing angle of 9.5 x 9.5 ° (10 x 10 cm). An image of a vase (http://turnyourhead.com) served as the semantic prime for the vase interpretation of the ambiguous figure, see Figure 5.1b. The image was presented centrally at a viewing angle of 7.6 x 9.5 ° (8 x 10cm). Each semantic prime was viewed at a distance of 60cm.

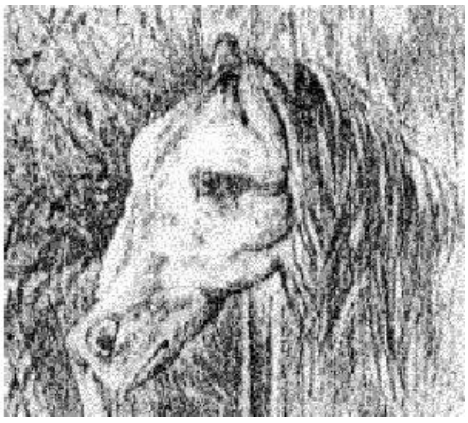
Neutral priming stimuli. The neutral priming stimuli used was the Horse/Frog illusion (www.planetperplex.com), see Figures 5.1c and 5.1d. The images were presented centrally at viewing angles of 8.5° x 8.5° (9 x 9 cm) at a distance of 60cm.

(a) Faces semantic prime

(b) Vase semantic prime



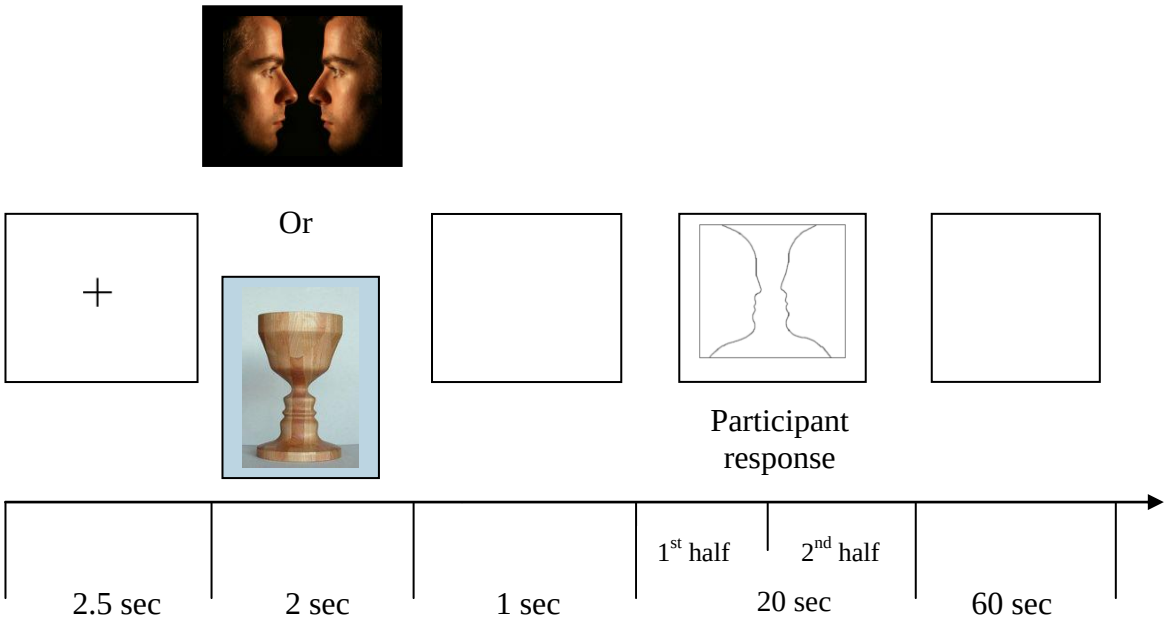
(c) Frog neutral prime



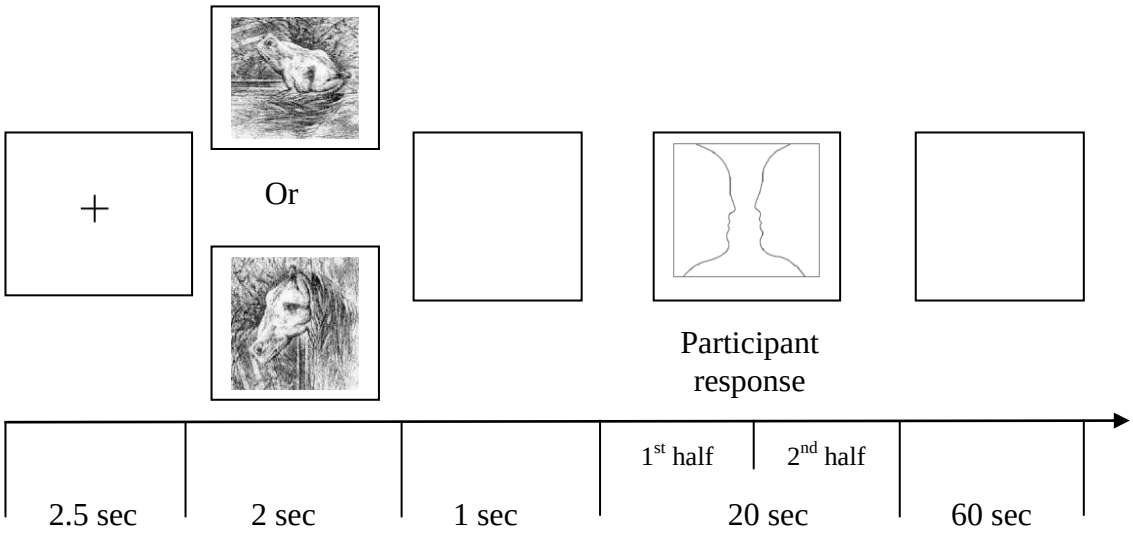
(d) Horse neutral prime

Figure 5.1. The stimuli used for the experiment, including the two semantic priming stimuli (Figures 5.1a and 5.1b), the two neutral priming stimuli (Figures 5.1c and 5.1d).

The experiment began with a blank screen for 2000ms as a warning that the first trial was about to begin. Each trial began with a fixation cross presented on screen for 2500 ms. The fixation cross was replaced with one of the priming stimuli, which was presented on screen for 2000ms before being replaced with another blank screen for 1000ms. The ambiguous Face-vase stimulus was then presented on screen for 20 seconds, after which participants were given a 1-minute rest period before the next trial, during which a blank screen was presented, see Figure 5.2 for the task sequence timeline. The priming stimuli were presented for 2000ms as previous studies have shown that this duration produces biasing effects, with the initial interpretation being in the same configuration as the prime (e.g. Long et al., 1992; Long & Moran, 2007; Long & Toppino, 2002). A 20 second presentation time was chosen as previous studies have shown that differences in reversal rate over time typically occurs between the first 10-second and the second 10-second presentation times (e.g. Long & Moran, 2007).



(a) Semantic prime trials



(b) Neutral prime trial

Figure 5.2. Task sequence timeline for the priming task, including the semantic prime condition (Figure 5.2a), and the neutral prime condition (Figure 5.2b).

Participants were asked to report figure reversals only during the presentation of the ambiguous Face-vase figure, but were required to view the screen at all times, even during blank screen presentations. Also, participants were asked to make as many reversals as possible, but only when they were certain of a change in perspective. When participants saw the two faces interpretation, they were required to press and hold down the left mouse button for as long as they saw that particular interpretation. Alternatively, when participants saw the figure as a vase they were required to press and hold down the right mouse button for as long as they saw that interpretation. If at any time during the presentation of the ambiguous figure participants could not make out either of the interpretations, they were required to release the mouse and not press either mouse button until they could clearly see one of the interpretations.

Participants were shown an example of the Face-vase illusion in the instructions sheet prior to testing, but were not shown any of the priming stimuli. They were informed that they would see an image prior to the presentation of the ambiguous figure, but no further details were given. Previous research has shown that naïve observers are more likely to report one interpretation of an ambiguous figure more than the other interpretation (Botwinick, 1961; Leeper, 1935). Consequently, for ambiguous figures to be perceived as truly ambiguous, with each interpretation equiprobable, Georgiades and Harris (1997) have shown that by informing participants of the two interpretations prior to testing that equiprobablity can be achieved. For this reason, participants were informed of the two interpretations of the ambiguous stimuli prior to the experiment in the instruction sheet.

The programme DMDX (Forster & Forster, 2003) was used to run the experiment, including presenting the stimuli, recording the duration each mouse key was held down for and the duration where no key responses were recorded, the number of reversals reported, and recording the total viewing time (ms). This data was recorded to determine how many reversals were made during the presentation time and, according to which mouse button had been pressed first, whether the first interpretation of the ambiguous figure had been influenced by the prime.

5.2.7. Procedure

The general procedure is outlined in section 2.6. Following the 10-minute rest period, the ambiguous figures tasks were completed. After which, participants were breathalysed again.

5.2.8. Data analysis

Group differences based on demographic information and questionnaire responses were analysed using independent *t*-tests.

Priming effect on first reversal. To analyse group differences in the initial interpretation of the ambiguous figure, the first key press was coded in terms of whether the ambiguous figure was seen in the same configuration as the preceding priming figure. If the first key press indicated that participant's initial interpretation of the ambiguous figure was in the same configuration as the preceding priming stimulus, a score of 1 was allocated to that trial. If the first key press indicated that participants' initial

interpretation of the ambiguous figure was in the alternate configuration to the priming stimulus a score of 0 was allocated to that trial. The first interpretation reported by the participants was recorded for each of the ten semantic trials. Data from the ten neutral trials were not included in this analysis as the neutral primes were unlikely to act as priming stimuli for the ambiguous Face-vase illusion. As each participant viewed the ambiguous Face-vase illusion ten times during the semantic trials, a participant's total first percept score could range from 0 to 10. The resulting value (0-10) was entered into an independent *t*-test to analyse group differences on the priming effect. A similar analysis has previously been conducted by Long and Moran (2007) (see also Long et al., 1992 from which Long & Moran, 2007 based their analysis).

Number of figure reversals. A mixed ANOVA was used to analyse group differences for the total number of reversals reported during the first and second half of the presentation of the ambiguous figure, with Group (Alcohol or Placebo group) as the between subjects factor, and two within subjects factors, Presentation half (first or second half), and Prime Type (semantic and Neutral). To control for individual differences in alternation rate, the data for each participant was normalised. Normalised alternation rates were calculated by dividing the total number of reversals reported by the total viewing time after the first and last data values had been removed, with the resulting value being used for the ANOVA analysis. The first data value was removed from the analysis as this represented the initial planning time, and was not a measure of a reversal itself. The last data value was removed, as this was the time between the last reversal reported by the participant and the end of the test session and was not a measure of a reversal having occurred. A similar calculation has been used previously

(see Zheng & Ukai, 2006), and is thought to be an appropriate measurement to evaluate the frequency of perceptual alternations.

5.3. RESULTS

5.3.1. Demographics

Participant demographic and test results are summarised in Table 5.1. Independent *t*-tests and chi-squared tests revealed no age, gender, weight or education differences between the groups. There were no significant differences between the groups for units of alcohol per week. There were no group differences in caffeine use (ratio yes:no), and of those who reported caffeine consumption, there were no differences between the groups in caffeine consumption, or time since last use. There were no group differences in cigarettes use (ratio yes:no), and of those who reported smoking, there were no group differences in the number of cigarettes smoked per day, or the time since last use. No significant differences were found between the groups for the MAST, and AUQ score and Binge scores. Of the recreational drugs that participants reported using, there were no group differences in the numbers that reported use compared with those who did not, and of those who did report use, there were no group differences in the number of days used per month (data not shown).

5.3.2. Breath Alcohol Concentration

All breath alcohol levels were 0 at the beginning of the session. There was a significant difference between the groups after drink consumption (alcohol group = 0.51 mg/l BRAlc) [$t(28) = 16.76$, $p = 0.01$], and at the end of the session (alcohol group = 0.25 mg/l BRAlc) [$t(28) = 9.23$, $p = 0.01$].

Table 5.1. Participant means, t-test and chi-squared results of between group comparisons (standard deviations in parentheses).

	Mean		Statistic	Value	Degrees of Freedom	*p* value
	Placebo Group (n = 15)	Alcohol Group (n = 15)				
Age (years)	19.60 (3.00)	18.87 (0.64)	t-test	-0.93	28	0.36
Gender (male:female)	9:6	7:8	Chi-Squared	0.54	1	0.46
Weight (kg)	68.61 (13.09)	65.96 (11.34)	t-test	-0.59	28	0.56
Education (years)	13.47 (0.74)	13.47 (0.99)	t-test	0.00	28	1.00
Alcohol (units per week)	16.47 (11.84)	19.00 (10.25)	t-test	0.63	28	0.54
Caffeine (ratio yes:no)	10:5	10:5	Chi-Squared	0	1	1.00
(Cups per day)	2.40 (1.51)	3.60 (1.51)	t-test	-1.78	18	0.09
(Hours since use)	5.20 (6.18)	15.22 (16.24)	t-test	1.82	18	0.09
Cigarettes (ratio yes:no)	1:14	0:15	Fisher's Exact	n/a	n/a	1.00
(per day)	5.00 (n/a)	n/a	n/a	n/a	n/a	n/a
(hours since use)	20.00 (n/a)	n/a	n/a	n/a	n/a	n/a
MAST	1.40 (1.24)	2.00 (1.31)	t-test	1.29	28	0.21
AUQ	39.88 (36.33)	49.57 (36.49)	t-test	0.73	28	0.47
BINGE	28.61 (29.27)	37.04 (27.17)	t-test	0.82	28	0.42

5.3.3. Task Performance

Priming effect on first reversal: An independent *t*-test was conducted on the mean number of "same" responses (ranging from 0-10), whereby the initial interpretation reported was in the same configuration as the preceding priming stimulus. This analysis showed a significant difference between the alcohol [mean = 3.40, sd = 1.72] and placebo [mean = 7.93, sd = 1.39] group [$t(28) = -7.94$, $p = 0.01$], with the placebo group more likely to report the first interpretation of the ambiguous Face-vase figure to be in the same configuration as the preceding priming stimulus.

Number of figure reversals: A mixed ANOVA was conducted on the average reversal rate, with Group (Alcohol and Placebo) as the between-subjects factor, and two within-subjects factors, presentation half (first and second half), and prime type (Semantic and Neutral). This analysis revealed no significant main effect of Group [$F(1,28) = 1.60$, $p = 0.22$], and a marginally significant main effect of presentation half [$F(1,28) = 3.77$, $p = 0.06$], with more reversals reported during the second half, see Figure 5.4. The main effect of prime type was significant [$F(1,28) = 11.03$, $p = 0.01$], with more reversals reported for the neutral stimuli. The Group x prime type interaction was marginally significant [$F(1,28) = 3.50$, $p = 0.07$]. There were significant Group x presentation half [$F(1,28) = 4.25$, $p = 0.05$], prime type x presentation half interaction [$F(1,28) = 5.03$, $p = 0.03$], and significant 3 way interactions between group x presentation half x prime type [$F(1,28) = 4.83$, $p = 0.04$]. To explore this interaction, 2 way ANOVAs were conducted for each presentation half. This revealed a significant group x prime type interaction for the first half presentation [$F(1,28) = 8.02$, $p = 0.01$] but not the second [$F(1,28) = 0.91$, $p = 0.35$]. Post hoc tests revealed that the placebo group reported fewer

figure reversals than the alcohol group for semantic prime conditions [t(28) = 2.39, p = 0.02], but there were no group differences in reversals for neutral prime conditions [t(28) = 0.56, p = 0.51], see Figure 5.3.

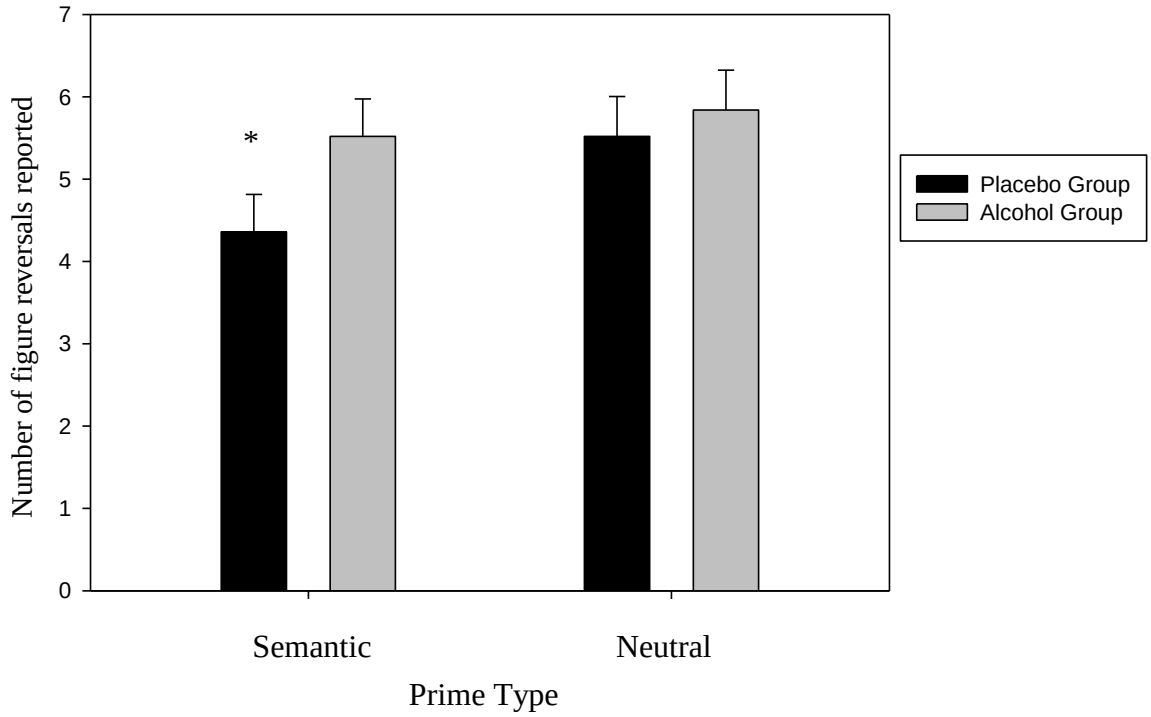


Figure 5.3. Number of figure reversals reported by the Placebo and Alcohol groups for each Prime Type during the first presentation half. Error bars represent Standard Error mean. * Indicates significant difference between Placebo and Alcohol groups (p = 0.02).

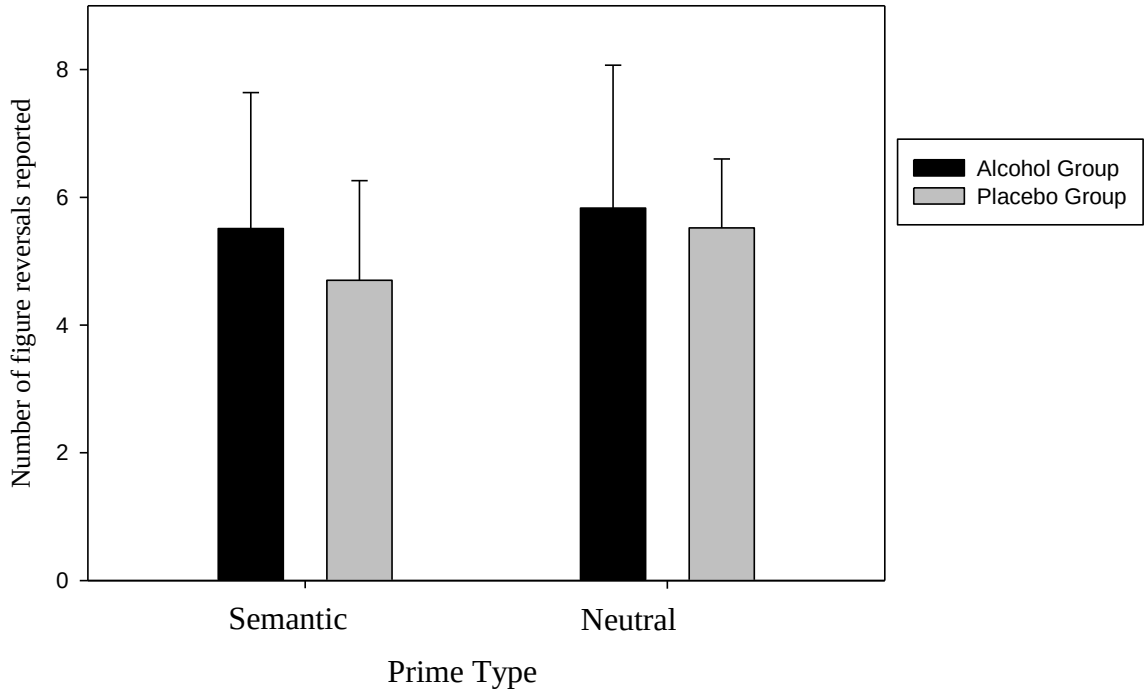


Figure 5.4. Number of figure reversals reported by the Placebo and Alcohol groups for each Prime Type during the second presentation half. Error bars represent Standard Error mean.

5.4. DISCUSSION

The aim the present study was to determine whether an effect on absolute processing of stimulus information was responsible for the alcohol effect found in Study 2. The results of the present study show that the placebo group were influenced by the presentation of the semantic prime. The placebo group were more likely to report their initial interpretation of the ambiguous figure to be in the same configuration as the prime. Furthermore, the reversal rate of the placebo group was also affected by the semantic prime. Fewer reversals were reported during the first presentation half, suggesting that the residual inhibition responsible for suppressing the alternate interpretation resulted in stabilising reversal rate. As the inhibitory mechanisms recovered during the second presentation half, reversal rate rose. For the alcohol group, the initial interpretation of the ambiguous figure was not influenced by the prior presentation of a semantically meaningful prime. Further alcohol-related inhibitory impairment was evident by the lack of a reduction in reversal rate during the early stages of the test phase, where the reversal rate of the alcohol group remained at a stable level throughout testing.

On the other hand, the results might be explained in terms of alcohol-related memory impairments. It is possible that the alcohol group were unable to accurately process and hold the prime in memory. In support, studies have shown that alcohol impairs the ability to encode information (Soderlund et al., 2005; Parker et al., 1974; Mueller et al., 1983), and is particularly evident when mnemonic strategies are required to retain the information (Saults et al., 2007). Given that verbal labels could be given to the prime, and research has shown that alcohol impairs the mnemonic strategies to achieve this is,

it might seem plausible to attribute the results to alcohol-related deficits in the ability to encode the prime. However, this is complicated by the fact that the experimental instructions did not ask participants to remember the prime and did not make the importance of the prime explicit. In which case, it seems unlikely that participants actively tried to process and encode the prime and groups differences in both reversal rate and initial interpretation can be explained in terms of alcohol-induced impairment of encoding.

Rather than attribute the reversal rate difference between the presentation halves shown by the placebo group to transient neural adaptation and recovery (Long et al., 1992; Long & Olszweski, 1999), an alternative possibility is that the reduction in reversal rate reflects the additional time required to overcome the residual inhibition responsible for suppressing the unprimed interpretation. As these inhibitory mechanisms recover, and residual inhibition is overcome, reversal rate rises. Whereas, the constant level of reversals reported by the alcohol group suggests that the underlying inhibitory mechanisms responsible for the reduction in reversal rate of the placebo group are impaired by alcohol. As the alcohol group have no residual inhibition to overcome, there is no reduction in reversal rate.

These results suggest that moderate doses of alcohol affect the ability to processes absolute saliency information. When presented with a semantically meaningful prime, the alcohol group were not biased by this information and so were less likely to report the subsequently presented ambiguous figure to be in the same configuration. In contrast the initial interpretation reported by the placebo group was more likely to be

influenced by the semantic prime, resulting in the initial interpretation of the figure to be in the same configuration. This result is similar to the effect observed in negative priming studies, whereby similar doses of alcohol suppress the priming effect (Fillmore et al., 2000a). Under normal circumstances, residual inhibition from previous trials affects the time taken to respond to subsequent trials. However, because alcohol impairs this inhibitory mechanism, preventing the distracter attribute from being inhibited, there is no residual inhibition to overcome, and so the reaction time cost is not found.

One conclusion that can be drawn so far is that alcohol affects inhibitory processes important for determining saliency under conditions of cognitive control. However, the specific role of inhibition remains uncertain. Therefore, the aim of the following chapter will be to further explore the effects of alcohol on inhibitory attentional processes using a different methodology.

CHAPTER 6:

THE ACUTE EFFECTS OF ALCOHOL ON THE INHIBITORY MECHANISMS INVOLVED IN ATTENTIONAL ORIENTING.

6.1. INTRODUCTION

Visual search represents another attention-based operation that might be enhanced by inhibitory mechanisms (Klein, 1988, 2000; Klein & MacInnes, 1999), and much of the research in this area has used modified versions of the allocation of visual attention model suggested by Posner and Peterson (1990) and Posner and Raichle (1994). In these tasks, a display is presented with a central fixation point and two peripheral boxes. The task is to detect a target in either of the peripheral boxes. Prior to target onset, a warning cue appears either peripherally or centrally. If the peripheral cue is not predictive of target location, it is believed to activate automatic (i.e. exogenous) attentional response mechanisms and attract attention in a reflexive manner (Danckert & Maruff, 1997; Rafal & Henik, 1994; Yantis & Jonides, 1990). A predictive central cue is referred to as a consciously controlled cue (i.e. endogenous), and attracts attention in a more controlled manner (Yantis & Jonides, 1990). This cue may either be valid, enhancing reaction time to the target, or invalid, interfering with reaction time to the target. Through varying the type of cue, the probability that the cue is correct, and the timing of the cue-target interval (stimulus onset asynchrony, or SOA), distinct attentional and corresponding inhibitory systems can be activated.

The cue-response paradigm described above can shed light on inhibitory functions because facilitatory and inhibitory components are required in the process of attentional

orienting (Posner & Peterson, 1990; Rafal & Henik, 1994). Consequently, automatic cues activate the automatic orienting system. From an inhibitory viewpoint, the automatic system is difficult to voluntarily suppress (Jonides, 1981; Lambert et al., 1987; Remington et al., 1992; Yantis & Jonides, 1990). Alternatively, controlled cues activate the intentional orienting system and are relatively easy to suppress intentionally (Jonides, 1981).

Furthermore, when SOAs are more than 450 ms, orienting to the location of the target is delayed if a non-predictive automatic cue appeared in that location beforehand (Klein, 2000), a phenomenon known as inhibition of return (IOR; Posner et al., 1985). IOR operates to enhance the information-gathering efficiency of searches by biasing attention toward new information in unexplored locations and away from redundant, old information contained in previously searched locations (Klein, 1988). Thus, the paradigm also offers a look at an automatic inhibitory process that occurs in response to reflexive orienting.

At brief cue-target SOAs, RTs are shorter on same-cue-target trials than control trials. However, the RT advantage for targets appearing at the cued location in tasks using central cues depends on the participant's expectancy that the target will appear at the cued location (Jonides, 1981; Rafal & Henik, 1994). With a high cue-target validity probability of around 80%, the RT advantage for detecting targets appearing at the cued location is greater than the same advantage observed using smaller cue-target validity probabilities, suggesting that participants are able to utilise the predictive information contained in the cues to increase the RT advantage for targets appearing at the cued

location (Maruff & Currie, 1995). Interestingly, when the cue-target validity probability ratio is reversed (i.e. 80% probability that targets will appear contralateral to the cued location), the RT advantage is also reversed (Danckert & Maruff, 1997), and participants show an RT advantage for targets appearing contralateral to the cued location (Danckert & Maruff, 1997; Maruff & Currie, 1995). This result suggests that participants are able to use the expectancy information contained in the spatial cues to facilitate effective search strategies (Danckert & Maruff, 1997; Maruff & Currie, 1995).

Schulte et al. (2001) examined the possibility that alcohol might impair the controlled mechanism involved in visual search. The attentional shift in the Schulte et al. (2001) study was manipulated by a central arrow, which on 80% of trials correctly indicated the location of the target. A short SOA was used to separate the cue and the onset of the target, which was intended to produce facilitatory effects on target detection. The results of the Schulte et al. (2001) study also showed a facilitatory effect, with RTs being faster on valid-cue trials than invalid-cue trials. However, the results showed that this effect was not impaired following alcohol consumption. From this study, it might be concluded that alcohol has no overall effect on controlled attention; however some caution should be applied before such claims are accepted. The Schulte et al. (2001) study shows that when the cue-target validity is high, the ability to use the expectancy information to facilitate an effective search strategy is not compromised following alcohol. However, as Maruff and Currie (1995) have shown, when the cue-target validity is lowered the RTs are longer than those using a higher cue-target validity. This suggests that when the cue-target validity is lowered, using the cue to facilitate detection of the target becomes a less effective search strategy. In effect, the information provided

by the cue needs to be inhibited in order to facilitate effective search. It is not known how alcohol would affect the ability to ignore controlled attention when the cue-target validity is lowered. Presumably, if using the cue to predict the location of the target is not an effective search strategy to use to detect the target, then one must inhibit the information given by the cue in order to locate the target more effectively.

Abroms and Fillmore (2004) examined the possibility that alcohol might selectively impair specific inhibitory mechanisms involved in automatic visual search rather than facilitatory mechanisms. On the basis of previous research that showed alcohol reduces inhibitory influences on selective attention (Fillmore et al., 2000a, 2000b), it was hypothesised that alcohol would also impair inhibitory influences involved in visual search and thereby diminish the magnitude of the IOR effect observed. Alternatively, as existing research has consistently shown that alcohol does not impair performance on tasks measuring other kinds of automatic attention (Fisk & Schneider, 1981), it seems reasonable to assume that this would also be the case when measuring automatic selective attention. The attentional shift was manipulated in the Abroms and Fillmore (2004) study by a peripheral cue presented prior to target onset. A range of SOAs separated the cue and the onset of the target, which was intended to produce both facilitatory and inhibitory effects on target detection. In terms of the inhibitory effect following longer SOAs, Abroms and Fillmore (2004) found that alcohol reduced the IOR effect by shortening its duration of influence, whereas the strength of the IOR effect was consistent under placebo. The manner in which the duration of IOR was diminished under alcohol suggests that the drug reduced inhibitory influences on target detection. The results showed that alcohol diminished the magnitude of the IOR effect

by reducing the time delay associated with detecting targets in previously attended locations. It is argued that the time delay normally observed in this condition is due to the operation of an inhibitory influence on the visual search process that delays the return of attention to previously attended locations (Klein, 2000). The reduced detection delay under alcohol could indicate some impairment of this inhibitory process so that its normal influence on target detection is diminished under the drug.

In terms of the facilitation effect, Abroms and Fillmore (2004) found no difference in RTs between cue-valid and cue-invalid trails, and no effect of alcohol. This is inconsistent with some other studies that reported facilitation of detection times at brief SOAs in this condition in controls (e.g. Lupianez et al., 2001). However, unlike those studies, the Abroms and Fillmore (2004) study included a procedure to disengage attention from the cue before the onset of the target. In this study, following the peripheral cue, the same brightening of the peripheral box was added to the central box so that attention was directed back to centre before the onset of the target. This procedure was adopted in light of previous research that suggested that the facilitatory effect reported elsewhere was not the result of a true facilitatory mechanism of attention, but the result of a procedural artefact where the brief SOA does not allow time for attention to leave the cued location before the target is presented (Lupianez et al., 2001). The procedure adopted by Abroms and Fillmore (2004) was thought to disengage attention from the cue and redirect it back to the centre of the display (Briand et al., 2000). However, by using this procedure to redirect attention back to centre before the onset of the target, Abroms and Fillmore (2004) may actually have been measuring the simple reflexive orienting to the onset of the target in the periphery. In

which case, the similarity in RTs between cue-valid and cue-invalid trials is not unexpected.

One conclusion that can be drawn from the studies presented in this thesis so far is that alcohol affects inhibitory processes important for determining saliency under conditions of cognitive control. However, the specific role of inhibition in this process remains unclear. Therefore, the aim of present study will be to further explore the effects of alcohol on inhibitory attentional processes using a different methodology. Although previous studies have suggested that alcohol does not impair controlled attentional orienting (Schulte et al., 2001), it is not known how alcohol affects the ability to inhibit this information when it is not conducive to facilitate effective search strategies. In order to test this, the present study will use a modified version of the allocation of attention model designed by Posner (1980) to investigate the ability to use predictive information contained in central cues to control consciously controlled orienting. The cue-target validity will be set at 50% so that on only half of trials will the location of the target be correctly indicated by the cue. Therefore, using the cue to detect the target will not be an effective search strategy and so the information provided by the cue should be inhibited in order to facilitate target detection. However, if alcohol impairs conscious control, then it is predicted that the information provided by the cue will not be inhibited. It was hypothesised that the alcohol group would be unable to inhibit controlled orienting strategies, resulting in faster RTs on trials where the cue and the target were the same side, and longer RTs on trials where the cue and target where on opposite sides. Alternatively, the placebo group should be able to inhibit controlled orienting strategies when the cue is unlikely to predict the target location, instead

adopting automatic orienting strategies shown by similar RTs for trials where the cue and target are on the same side and trials where the cue and target are on opposing sides.

6.2. METHOD

6.2.1. Participants

24 young social drinkers from the University of Birmingham with normal or corrected-to-normal vision participated in the study. There were two experimental groups, an alcohol (7 male and 5 female; mean = 20.25 years, sd = 2.67, average self reported consumption of 13.17 units of alcohol per week) and a placebo group (5 male and 7 female; mean = 19.33 years, sd = 1.44, average self reported consumption of 12.50 units of alcohol per week). Participants were told they were taking part in a study looking at the effects of alcohol on attention, but were not told about its effects for different cue-target probabilities. Participants were the first to volunteer who met the criteria outlined in section 2.2.1 and were recruited through the Psychology departments' research participation scheme (see section 2.2).

6.2.2. Design

Participants were allocated pseudo-randomly to groups such that the groups were matched for age and gender. The visual attention task was presented in two blocks, one block containing the predictive-cue trials, and the second block containing the non-predictive-cue trials. Within each block, the order of stimuli was pre-randomised such that same-side cues, same-side targets and catch trials were not presented together. The first 12 stimuli in each block were used as practice trials, and so RT data from these were not used in the analysis. The order of presentation was fully counter balanced.

6.2. 3. Inclusion and Exclusion criteria

Participants were excluded based on the criteria given in section 2.2.1.

6.2. 4. Screening Tools

Details of the Lifestyle Questionnaire, Alcohol Use Questionnaire (AUQ; Mehrabian & Russell, 1978), Michigan Alcohol Screening Test (MAST; Selzer, 1971) that participants completed for this study are described in detail in section 2.3.

6.2.5. Alcohol administration

A detailed description of the alcohol administration is given in section 2.4.

6.2.6. Materials and Tasks

The attention task required participants to shift their attentional focus according to a cue that appeared before each stimulus presentation. The attentional shift was manipulated by a central arrow cue which, in the predictive cue block, indicated the correct location of the target on all trials. In the non-predictive cue block, the cue correctly predicted the location of the target on 50% of the trials and incorrectly on 50% of trials.

Each trial began with a 1000 ms presentation of a central fixation cross (5 x 5 mm) and two rectangular boxes (36 x 26 mm) presented on either side of the screen, 85 mm from fixation. Participants were instructed to maintain their gaze on the central fixation cross. After 1000 msec, the fixation cross was removed and replaced with either a central leftward or rightward facing arrow cue (11 x 9 mm) for 100 msec.

Predictive cue block: the central arrow cue was removed after 100 msec and replaced with a target (an asterisk, 5 x 5 mm), either 103mm to the left or to the right of fixation

(presented centrally within one of the two peripheral rectangular boxes) according to the direction of the central arrow cue. In all cases, the location of the target was the same as the location predicted by the central arrow cue, see Figure 6.1.

Non-predictive cue block: after the 100msec presentation of the central arrow cue, it was removed and replaced by the target. In 50% of trials, the target appeared in the location predicted by the central arrow cue 103mm to the left or right of fixation. In 50% of trials, the target appeared in the opposite location to that predicted by the central arrow cue 103mm to the left or right of fixation, see Figure 6.1.

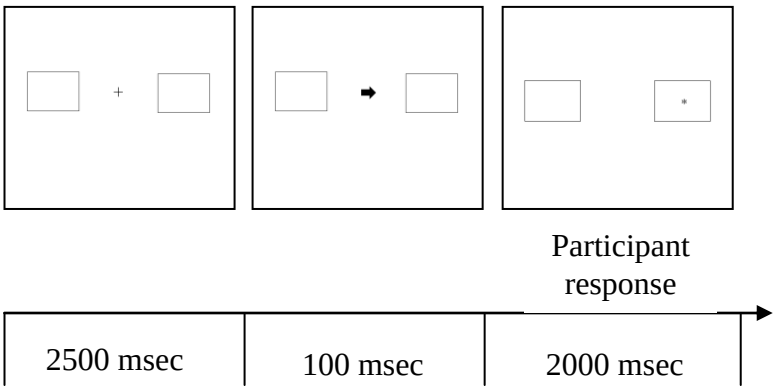
(a) Symbolic cue predicting the location of the target

(b) For all valid trials, the target was in the location predicted by the cue

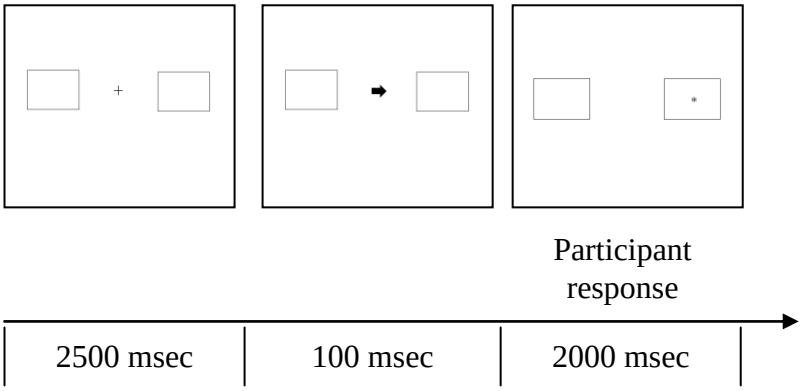
(c) For all invalid trials, the target was in the opposite location to that predicted by the cue

Figure 6.1. An example of the cueing task with a symbolic cue predicting the location of the target (Figure 6.1a), which could either be a valid cue (Figure 6.1b), or an invalid (Figure 6.1c).

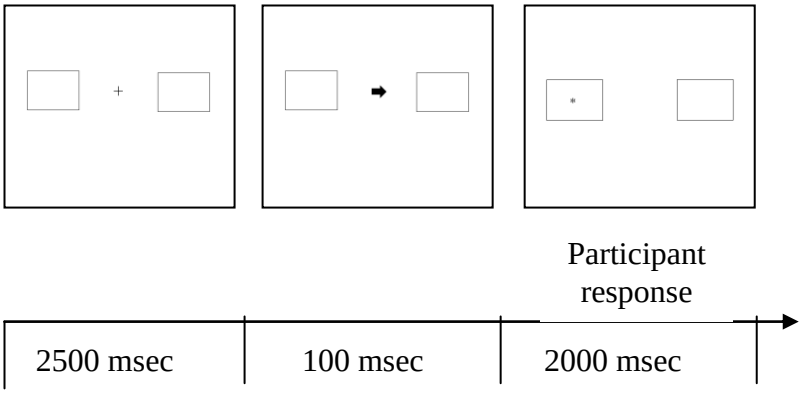
For both the predictive and non-predictive cue blocks, once the target appeared in one of the peripheral boxes, participants were asked to make a response as quickly as possible once by pressing the spacebar key on the laptop's keyboard. The target appeared on the screen for 2000ms or until it was terminated by pressing the spacebar. The next trial started either once the display of the target had been terminated, either by a button press by the participant or following the 2000ms presentation time, see Figure 6.2 for the task sequence timeline. The stimuli were presented in black on a white background.



(6.2a) Predictive cue condition



(6.2b) Non-predictive cue condition: valid trial



(6.2c) Non-predictive cue condition: invalid trial

Figure 6.2. Task sequence timeline for the cueing task, including the predictive cue condition (Figure 6.2a), the Non-predictive cue: valid trial condition (Figure 6.2b), and the Non-predictive cue: invalid trial condition (Figure 6.2c).

Each block consisted of 132 trials in total, and of the 132 trials, 20% were catch trials where no target was presented after the cue. Participants were required not to respond on catch trials and to wait until the next trial started. Catch trials are commonly used in target-detection tasks to prevent anticipatory responding (Lupianez et al., 2001). The first 12 trials within a block served as practice trials (10 trials with a target, plus 2 catch trials), these were not used for analysis. The remaining 120 trials were used for analysis (100 trials with a target, plus 20 catch trials). Participants were shown an example of stimuli in the instructions sheet prior to testing.

The programme DMDX (Forster & Forster, 2003) was used to run the experiment, including the presentation of the stimuli, recording the reaction times, missed responses and incorrect responses to catch trials.

Reaction time to detect a target was the performance measure and was recorded in milliseconds to respond after the onset of the target. For each participant, a mean RT score was calculated for the predictive cue block and the non-predictive cue block and entered into the analysis. Responses with RTs less than 100msec were interpreted as anticipatory responses and were excluded from analysis (0.2% of trials). Trials on which no response was made (misses, 0.2% of trials) and false alarms (erroneous responses on catch trials, 0.3% of trials) were removed from the analysis. Finally, RTs above or below 2 standard deviations from the mean RT were removed as outliers (0.1% of trials).

6.2.7. Procedure

The general procedure is outlined in section 2.6. Following the 10-minute rest period, participants completed the visual attention task, after which participants were breathalysed again.

6.2.8. Data Analysis

Reaction times below 100 ms were considered anticipatory and removed, and reaction times above 2 standard deviations above an individual's mean were deemed outliers and removed (0.2% and 0.1%).

For the predictive-cue condition, a mixed ANOVA was used to analyse group differences in RTs. The between-subjects factor was Group (Alcohol and Placebo), and the within-subjects factor was Target Side (Left or Right). For the non-predictive-cue condition, a mixed ANOVA was used to analyse group differences in RTs, with one between-subjects factor (Alcohol and Placebo), and two within-subjects factors, Cue Type (Valid and Invalid), and Target Side (Left or Right).

Demographic information and questionnaire response analysis is described in section 2.7.

6.3. RESULTS

6.3.1. Demographics

Participant demographic and test results are summarised in Table 6.1. Independent t-tests and chi-squared tests revealed no age, gender, weight or education differences between the groups. There were no significant differences between the groups for units of alcohol per week. There were no group differences in caffeine use (ratio yes: no), and of those who reported caffeine consumption, there were no differences between the groups in caffeine consumption, or time since last use. There were no group differences in cigarettes use (ratio yes: no), and of those who reported smoking, there were no group differences in the number of cigarettes smoked per day, or the time since last use. No significant differences were found between the groups for the MAST, and AUQ score and Binge scores. Of the recreational drugs that participants reported using, there were no group differences in the numbers that reported use compared with those who did not, and of those who did report use, there were no group differences in the number of days used per month (data not shown).

6.3.2. Breath Alcohol Concentration

All breath alcohol levels were 0 at the beginning of the session. There was a significant difference between the groups after drink consumption (alcohol group = 0.43 mg/l BRAlc) [$t(22) = 0.43$, $p = 0.01$], and at the end of the session (alcohol group = 0.22 mg/l BRAlc) [$t(22) = 0.22$, $p = 0.01$].

Table 6.1. Participant means, t-test and chi-squared (Fisher's Exact test where applicable) results of between group comparisons (standard deviations in parentheses

	Mean		Statistic	Value	Degrees of Freedom	*p* value
	Placebo Group (n = 12)	Alcohol Group (n = 12)				
Age (years)	19.33 (1.44)	20.25 (2.67)	t-test	1.05	22	0.31
Gender (male:female)	5:7	7:5	Chi-Squared	0.67	1	0.41
Weight (kg)	68.82 (12.98)	68.60 (13.49)	t-test	-0.04	22	0.97
Education (years)	14.25 (1.66)	14.58 (1.98)	t-test	0.45	22	0.66
Alcohol (units per week)	12.50 (5.40)	13.17 (5.51)	t-test	0.30	22	0.77
Caffeine (ratio yes:no)	9:3	10:2	Fisher's Exact	n/a	n/a	1
(Cups per day)	3.45 (2.62)	3.60 (1.71)	t-test	0.15	19	0.88
(Hours since use)	11.73 (12.99)	8.10 (7.05)	t-test	-0.78	19	0.44
Cigarettes (ratio yes:no)	4:8	3:9	Fisher's Exact	n/a	n/a	1
(per day)	6.25 (2.99)	8.33 (4.73)	t-test	0.72	5	0.50
(hours since use)	1.75 (0.50)	2.33 (0.58)	t-test	1.44	5	0.21
MAST	2.50 (1.31)	2.00 (1.21)	t-test	-0.97	22	0.34
AUQ	49.95 (31.24)	55.58 (25.99)	t-test	0.48	22	0.64
BINGE	37.78 (26.24)	43.92 (22.97)	t-test	0.61	22	0.55

6.3.3. Task Performance

Predictive-cue Condition: A mixed ANOVA was conducted on RT, with Group (Alcohol and Placebo) as the between-subjects factor, and Target Side (Left or Right) as the within-subjects factor. This analysis revealed no significant main effect of Group [$F(1,22) = 1.34$, $p = 0.26$]. The main effect of Target Side was also not significant [$F(1,22) = 0.03$, $p = 0.87$], nor was the Group x Target Side interaction [$F(1,22) = 0.26$, $p = 0.62$], see Table 6.2 for mean scores and standard deviations.

Non-predictive-cue Condition: A mixed ANOVA was conducted on RT, with one between-subjects factor (Alcohol and Placebo), and two within-subjects factors, Cue Type (Valid and Invalid), and Target Side (Left or Right). This analysis revealed no significant main effect of Group [$F(1,22) = 0.01$, $p = 0.98$]. The main effect of Cue Type was significant [$F(1,22) = 10.14$, $p = 0.01$] as was the Group x Cue Type interaction [$F(1,22) = 7.56$, $p = 0.01$]. Post hoc tests revealed that the alcohol group were significantly faster to detect the target following a valid cue [$t(11) = -5.02$, $p = 0.01$]. Whereas, the RTs of the placebo group were unaffected by cue type [$t(11) = -0.27$, $p = 0.79$], see Table 6.2 for mean scores and standard deviations. The main effect of Target Side was not significant [$F(1,22) = 0.10$, $p = 0.76$], nor were the Group x Target Side [$F(1,22) = 0.03$, $p = 0.87$], Cue Type x Target Side [$F(1,22) = 0.01$, $p = 0.98$], and Group x Cue Type x Target Side [$F(1,22) = 0.01$, $p = 0.93$] interactions, see Table 6.2 for mean scores and standard deviations.

Table 6.2. Means and standard deviations (in parentheses) of scores on all tasks for each group. * Indicates significant differences between the alcohol and placebo groups ($p = 0.01$)

Visual Attention Task	Placebo Group (n = 12)	Alcohol Group (n = 12)
Predictive-Cue Condition		
Left-side Target	355.64 (41.20)	335.35 (37.93)
Right-side Target	354.37 (38.88)	337.87 (42.03)
Mean Reaction Time	355.00 (39.21)	336.61 (38.72)
Non-Predictive-Cue Condition		
Valid Cue		
Left-side Target	350.29 (40.84)	341.90 (34.76)
Right-side Target	353.47 (39.85)	342.46 (43.93)
Mean RT	351.88 (40.35)	342.18 (39.35)*
Invalid Cue		
Left-side Target	352.31 (47.27)	361.80 (29.39)
Right-side Target	354.40 (30.43)	362.88 (57.21)
Mean RT	353.36 (38.85)	362.34 (43.30)

6.4. DISCUSSION

The present study examined the effects of a moderate dose of alcohol on performance on a selective attention task designed to measure the inhibitory mechanisms involved in controlled attentional orienting. The task was designed to initially show that alcohol does not affect the ability to use information provided by a central cue when using this information can facilitate search, but does affect the ability to inhibit the information provided by the cue when using the information would be detrimental to locate the target.

In the task used to determine whether alcohol would impair the ability to use controlled information when the information was likely to facilitate effective searching behaviour, the alcohol group performed on a par with the placebo group. In this task, the cue-target validity was set at 100%, and so the information provided by the cue would always correctly indicate the location of the target. Moderate doses of alcohol are well known to produce heightened impulsivity and erroneous responding in tasks such as go/no-go and stop-signal tasks (e.g. Easdon et al., 2005), and so it is a possibility that the apparent similarity in performance on this task between the alcohol and placebo group happens to reflect this well documented impairment. However, the task also included catch trials whereby the cue was presented as expected, but no target was presented. On such trials, participants were instructed to refrain from responding. If the apparent similarity in the performance of the two groups on this task simply happened to reflect the alcohol groups impulsive responding, then it would also follow that the alcohol group would also make an increased number of erroneous responding on catch trials. The results of this task show that both groups made relatively few responses on catch trials, and so the

similarity of performances on this task is unlikely to be a reflection of alcohol resulting in increased impulsivity.

The lack of an alcohol effect on this task was not unexpected and is similar to studies whereby the cue-target validity was set at 80% (Schulte et al., 2001). In the Schulte et al (2001) study, the lack of an effect of alcohol on the task was unexpected and the overall conclusion from the study was that alcohol did not seem to impair performance on this task. This was an unexpected result given that previous research in other areas of the attention literature has shown deficits on similar tasks.

The task used to determine whether alcohol would impair the ability to inhibit controlled information when the information was unlikely to facilitate effective searching behaviour revealed differences in search behaviour between the alcohol and placebo group. In this task, the cue-target validity was set at 50%, and so the information provided by the cue would only correctly indicate the location of the target on half of trials. As a result, using the information provided by the cue would not facilitate search behaviours and so in order to detect the target more efficiently the information provided by the cue ought to be inhibited. The results of this task show that the placebo group were able to inhibit the information provided by the controlled cue and automatically orient towards the cue upon its appearance in the peripheral location. In contrast, the alcohol group were unable to inhibit the information provided by the cue. They produced an apparent facilitation effect on those trials where the cue happened to indicate the correct location of the target. On the other hand, on those trials where the cue and the target were on opposing sides, the RTs were significantly greater

reflecting the additional time needed to re-orientate from the location predicted by the cue to the location of the target.

The manner in which the RTs of the alcohol group were increased on those trials where the cue and the target were in opposing locations suggests that inhibitory influences on target detection were reduced by the drug. Although trials where the cue and target were in the same location show a facilitation effect, the increase in RT on trials where they were on opposing sides reveals the detrimental effects of alcohol on attention.

The lack of a facilitation effect shown by the placebo group in this study was also found in a similar study by Abroms and Fillmore (2004); RTs were similar for same cue-target location and different cue-target locations. Initially, these results appear to be inconsistent with other studies that have revealed facilitation effects on same cue-target location trials (e.g. Lupianez et al., 2001). However, unlike those studies, Abroms and Fillmore (2004) used a procedure designed to disengage attention from the cue and redirect it to centre before the onset of the target. This ensured that attention was refocused on the centre of the display prior to each target presentation. The disengagement–recentering effect was thought to ensure greater control over attention and reduce the likelihood of facilitation by preventing attention from remaining at the cue area throughout the trial (Briand et al., 2000; MacPherson et al., 2003). However, redirecting attention to the centre before the onset of the target may have resulted in reflexive orienting towards the target upon its presentation in the periphery. In which case, the similarity in RTs between same cue-target location and different cue-target location trials is not unexpected, regardless of whether the prior cue summoned

attention automatically (as in the Abroms & Fillmore, 2004 study) or consciously controlled as in the present study.

The reduced inhibitory effect suggested here might reflect a mechanism by which alcohol can impair attention-based behaviour. Evidence that alcohol reduces inhibition that normally biases attention away from explored locations provides new insights into how the drug might disrupt attention-based behaviour. The reduced duration of IOR under alcohol suggests that redundant searching of previously explored locations might be more likely to occur under the drug. Unnecessary reacquisition of visual information would slow the rate at which new information could be obtained, and subsequently processed. This redundancy could have the effect of diminishing visual search efficiency under the drug. Such a propensity to acquire redundant visual information could contribute to alcohol-related slowing effects on information processing (e.g. Carpenter, 1962).

In summary, the results of the present study give a more detailed explanation of the effects of alcohol on selective attention. Rather than impairing performance in all tasks that place demands on visual spatial attention (Post et al., 1996), the present study shows that alcohol impairs controlled spatial attention when the information provided by the cue does not facilitate target detection. Here, alcohol seems to impair the ability to inhibit information provided by a spatial cue, leading to a facilitation effect on trials where the cue and target happen to be in the same location, and increased RTs when they are in opposing locations.

CHAPTER 7:

GENERAL DISCUSSION

7.1. INTRODUCTION

This chapter will integrate the findings of the studies presented within this thesis, discussing their implications, and identifying areas for future research. The first section evaluates the thesis by discussing the strengths and limitations of the studies. In the second section, the effects of alcohol on visual perception are considered and discussed. The third section will then discuss the implications of these findings in relation to existing knowledge of this research area. Finally, areas for future research that will enhance understanding of the underlying mechanisms involved in visual perception are explored.

7.2. EVALUATION OF THE RESEARCH

7.2.1. Strengths of the research

One main aim of this thesis was to assess the effects of a moderate dose of alcohol on the attentional processes involved in figure reversals. This approach offers a number of important advantages:

First, the use of ambiguous figures provides a novel way to assess several mechanisms that have been shown to be impaired by alcohol using a single stimulus. Previously, studies comparing the effects of alcohol on these mechanisms have used different experimental tasks to assess performance on automatic and controlled inhibition. Although the response required in the two tasks is similar in these studies, the

implications of using different experimental tasks was not fully addressed, although they assumed that the underlying mechanisms would be the same between them. This has obviously complicated any direct comparison between the effects of alcohol on automatic and controlled mechanisms in previous studies. Rather than having to make comparisons across different modalities and experimental tasks, the studies of ambiguous figures presented throughout this thesis allow a direct comparison between automatic and controlled processes using a single stimulus type.

Second, the use of ambiguous figures allows various influences on reversal rate to be explored in varying degrees whilst keeping the stimulus itself constant throughout. The studies presented in this thesis assessed the effects of alcohol on image stabilisation using shading (Study 2 and Study 3), fixation location (Study 4), intermittent presentation (Study 5), and priming (Study 6).

Third, the performance measure of reversal rate is simple and consistent. Previous studies have shown that the dose of alcohol used in this thesis does not impair basic psychomotor performance such as simple keyboard or mouse presses. Consequently, any study in this thesis where an effect of alcohol was evident was unlikely to be clouded by alcohols effect on the motor response.

Fourth, the placebo drink used throughout was shown to be effective. The determination of the effects of a drug often requires the use of placebo, especially for drugs acting on the central nervous system. This is particularly problematic for alcohol because of its distinctive taste and smell when it is administered at doses intended to be intoxicating.

Typically, orange juice or some other citrus flavoring is used to dilute and mask the alcohol, but when the alcohol concentrations are at the levels used in this thesis, the alcohol is readily detected. The alternative is to make the placebo taste as though it contains alcohol. Pilot studies showed that few participants in a placebo group could identify whether they were in the alcohol or placebo group. When asked after participants had consumed all three drinks, few participants could correctly identify which group they had been assigned to, see Table 7.1 for a summary of participants who thought they had received placebo or alcohol for each study. In addition, even upon completion of the experiment, a considerable number of participants remained uncertain of which group there were in.

Table 7.1. Perceived consumption of alcohol and placebo for each study.

	Placebo group	(ratio yes:no)	**Alcohol group**	(ratio yes:no)
Study 1	N = 15	7:8	N = 15	11:4
Study 2	N = 20	8:12	N = 18	13:5
Study 3	N = 15	9:6	N = 15	11:4
Study 4	N = 11	6:5	N = 11	9:2
Study 5	N = 15	8:7	N = 15	11:4
Study 6	N = 12	5:7	N = 12	9:3

Fifth, a number of variables known to impact on cognitive processes were controlled in the studies presented within this thesis. For instance, a detailed lifestyle questionnaire was used to collect information on caffeine use, tobacco use, recreational drug use, and the AUQ and MAST questionnaires gave an indication of participants past and present drinking histories. This ensured that differences between the groups were a function of acute alcohol consumption on task performance, and not confounded by other factors relating to drinking history. Although such measurement is consistent with some of the

previous research into the cognitive effects of alcohol (e.g. Weissenborn & Duka, 2003; Townshend & Duka, 2001, 2002; Nicol & ford, 1986; Conley, 2001; Skinner & Sheu, 1982), measurement of these variables is not routinely performed.

7.2.2. Limitations of the research

This research has a number of potential limitations that should also be considered:

First, the population of social drinkers investigated in this thesis was relatively young, and so their previous experiences with alcohol might be considered to be limited. However, not only was self-reported usage on the AUQ and MAST questionnaires in the samples used throughout the thesis comparable to other studies of alcohol effects (e.g. Weissenborn & Duka, 2003; Townshend & Duka, 2001, 2002; Nicol & ford, 1986; Conley, 2001; Skinner & Sheu, 1982), but so was the age range of the samples (e.g. Townshend & Duka, 2002; Fillmore et al., 2000a, 2000b). Furthermore, there is a relatively small window of opportunity in alcohol studies as participants must be above the legal age limit, but also under the age of 35 when cognitive decline begins (Nilsson et al., 2009).

Second, the patterns of drinking could have been explored in more detail. Although groups were matched in terms of alcohol use and binge drinking, the possible effects of binge drinking on performance in these tasks was not measured. For example, previous studies have shown that binge drinkers perform worse on cognitive tasks than non-bingers (Weissenborn & Duka, 2003).

Third, participants were recruited only from a university population, which is likely to have relatively higher levels of IQ than the general population. There is some research to link levels of intelligence with cognitive performance and the ability to alternate between interpretations of ambiguous figures (e.g. Rock et al., 1994; Fulgosi & Guilford, 1966). However, it is important to note that previous research with social drinkers has recruited participants from similar populations (e.g. Weissenborn & Duka, 2003; Townshend & Duka, 2001, 2002). Consequently, the results obtained in this thesis can be seen in relation to other studies that have examined alcohol-related deficits.

Fourth, the sample used within this thesis tended to consist of participants whose first language was English. There is a small body of research from the ambiguous figures literature that suggests that bi- or multi-lingual participants are likely to report more figure reversals than monolinguals (Bialystok & Shapero, 2005). This is thought to be due to the ability of bi- and multilingual participants to reassign numerous meanings to the same object. Again, it is important to note that the samples used in the present thesis are similar to those used in other studies (e.g. Weissenborn & Duka, 2003; Townshend & Duka, 2001, 2002).

Fifth, although, the sample sizes chosen for the experiments within this thesis are comparable to the majority of the studies in the alcohol (Abroms et al., 2006; Abroms & Fillmore, 2004; Fillmore et al., 2000a, 2000b) and ambiguous figure (Nakatani & van Leeuwen, 2006; Kornmeier & Bach, 2004; Georgiades & Harris, 1997) literature, it remains a possibility that the sample size may not have been large enough to detect

some of the impairing effects of alcohol. However, the significant effects of alcohol obtained in Chapter 3 using tests taken from the CANTAB test battery showed that the use of these sample sizes was suitable to detect previously identified alcohol-related deficits in performance. As such, it seems unlikely that the sample sizes used here would not be large enough to detect alcohol-related deficits on the tasks used in this thesis, and significant effects were observed on some tests.

Last, only one dose of alcohol was used throughout this thesis. However, the dose used is comparable to that typically consumed by social drinkers on a single occasion (Kerr et al., 1991). Importantly, studies have shown that low alcohol doses do not impair performance on many cognitive tasks (e.g. Heishman et al., 1997; Mangold et al., 1996; Hindmarch et al., 1991, 1992; Lukas et al., 1989). Impairment is evident only at higher alcohol doses (e.g. Pickworth et al., 1997; Millar et al., 1995; Wilkinson, 1995; Kennedy et al., 1993), with consistent effects being reported at doses of around 0.8 g/kg used in the present thesis (Hindmarch et al., 1991). In addition, few studies assess the effects of alcohol at doses much beyond the dose used here because the sensitivity to detect subtle effects at these doses is considerably reduced (Hindmarch et al., 1991). And so, testing the effects of alcohol on the ability to perform the tasks used in this thesis at doses above 0.8 g/kg would have been unlikely to detect subtle impairments in cognitive functions. Therefore, any resulting deficit found in the studies presented in this thesis is likely to be similar to those deficits experienced in real life situations where similar doses of alcohol are consumed in a single occasion. The primary aim of this thesis was not to measure dose-related effects of alcohol, but more to establish reliable alcohol-induced performance effects on visual perception tasks.

7.3. MAIN FINDINGS

The findings of this thesis not only add further to previous work on the effects of alcohol on automatic and controlled behaviours, but also provide novel information about how alcohol affects visual perception revealed through reporting figure reversals. The following section begins with a brief overview of the findings of the studies presented in this thesis (see Table 7.2 for a summary of the interpretations of the ambiguous figures studies) before discussing the results in greater detail.

7.3.1. Overview of findings

The results of Study 2 showed no group differences in reversal rate when viewing the ambiguous figure voluntarily. However, group differences were evident when viewing the biased versions of the figure, with the alcohol group reporting more reversals than the placebo group when presented with the most biased version of the figure. Additionally, the reversal rate of the alcohol group increased as a function of bias, whereas the placebo group had a similar reversal rate across all conditions. However, this effect was evident only when presented with the Face-vase illusion, no group differences were found for the Necker cube.

The results of Study 3 showed no group differences in reversal rate in any of the conditions under spontaneous reversal. Fewer figure reversals were reported once the biasing manipulation was introduced. The results again show that more reversals were reported for the Face-vase figure than the Necker cube figure, although this was not affected by alcohol.

The aim of Study 4 was to further investigate the complex interactions found in Studies 2 and 3 using a different type of bias that did not change the physical properties of the stimulus, by manipulating the reversal rates in a different way that leaves the basic stimuli unchanged. The results did not show group differences in the number of figure reversals reported for any of the conditions. Once again, the reduction in reversal rate was evident only when viewing the Face-vase illusion.

The aim of Study 5 was to further explore the possible role of exploratory eye movements in figure reversals. The results showed a reduction in reversal rate during intermittent presentation, but no group differences in reversal rate. Again, the difference in reversal rate between conditions was found only for the Face-vase illusion, but this was not affected by alcohol.

The results of Study 6 show that the initial interpretation of the figure reported by the alcohol group, unlike the placebo group, was not influenced by the prior presentation of a prime. In addition, unlike the placebo group, the reversal rate of the alcohol group was not affected by the prior presentation of a semantically meaningful prime.

Study 7 moved away from ambiguous figures and used a simple cuing task to show that automatic attentional orienting was not impaired following alcohol per se, but the ability to inhibit consciously controlled information when the information was unlikely to facilitate searching behaviour did reveal alcohol-induced impairment. In contrast, the

placebo group was able to inhibit the consciously controlled information in the latter condition, and automatically orient towards the cue, facilitating search behaviour.

Table 7.2. Summary of the interpretations from each of the ambiguous figures studies

	Ambiguous figure	**Bias manipulation**	**Reversal instruction**	**Interpretation of results**
Study 2	Face-vase	Shading	Voluntary	Alcohol increases reversal rate (dark-biased figures only)
	Necker cube	Shading	Voluntary	No effect of alcohol on reversal rate
Study 3	Face-vase	Shading	Automatic	No effect of alcohol on reversal rate
	Necker cube	Shading	Automatic	No effect of alcohol on reversal rate
Study 4	Face-vase	Fixation	Voluntary	No effect of alcohol on reversal rate
	Necker cube	Fixation	Voluntary	No effect of alcohol on reversal rate
Study 5	Face-vase	Intermittent	Voluntary	No effect of alcohol on reversal rate
	Necker cube	Intermittent	Voluntary	No effect of alcohol on reversal rate
Study 6	Face-vase	Priming	Voluntary	Alcohol is less susceptible to priming stimuli; first interpretation and first half reversal rate unaffected

.

7.3.2. Automatic versus consciously controlled inhibitory mechanisms

The effect of alcohol on the intentional control mechanism was explored in Studies 2 and 3 where reversal rate was under volitional and automatic control respectively. The results showed that that both the alcohol and placebo group reported a similar number of figure reversals during passive viewing conditions but group differences were observed when figure reversals were under volitional control.

The involvement of inhibitory mechanisms in figure reversals has previously been identified (Wimmer, 2007; Doherty & Wimmer, 2005). However, Studies 2 and 3 suggests that alcohol has a different effect on the inhibitory mechanisms depending on whether reversals are under volitional or automatic control. When reversals were under volitional control (Study 2), alcohol tended to increase reversal rate, at least for the face-vase stimulus, and this is compatible with a simple account in which alcohol tends to disrupt an inhibitory process involved in stabilizing the perceived interpretation. However, when no volitional control was exerted (Study 3), alcohol tended to reduce reversal rate, and this is clearly at odds with a simple inhibitory account. Therefore, it appears that a moderate dose of alcohol does not reduce inhibition when automatic control mechanisms are activated.

Initially, the conclusion from Study 2 and Study 3 might be that the results replicate existing research showing that alcohol impairs performance only when the intentional mechanism of control is activated (Abroms et al., 2006; Fillmore & Vogel-Sprott, 2006; Holloway, 1995). However, the finding that alcohol tends not to affect performance when presented with the unbiassed versions of the figure complicates this conclusion.

Previous studies have shown that alcohol does not affect performance in all cases when the intentional control mechanism is activated (Fillmore, 2004) and the reason why alcohol would act only on certain aspects of conscious control is not fully understood. A detailed comparison of the stimuli used in these studies may provide some reasons for this selective effect when the intentional control mechanism is activated. The following section offers some possible explanations for the different effects of alcohol on reversals when viewing ambiguous and biased figures.

7.3.3. Alcohol effects on intentional inhibitory mechanisms

The studies presented in this thesis suggest that alcohol selectively affects the intentional mechanism of control as no effect of alcohol was evident on those tasks that measured only the automatic control mechanism. It seems unlikely that alcohol has a general effect on intentional control since it acts differently in different conditions. Moreover, the results do not support the Alcohol Myopia model because alcohol was shown to have a facilitatory effect on reversals in Study 2 and Study 6. According to this model, alcohol should restrict the focus of attention to the most salient features (e.g. Steele & Josephs, 1988, 1990; Steele & Southwick, 1985). In this case, alcohol should produce fewer reversals in the biassed conditions, and the results do not support this.

The finding that alcohol increased reversal rate in Study 2 is more consistent with an alcohol-related deficit on inhibitory attentional processes. The ability to alternate between two versions of an ambiguous figure is thought to depend upon inhibitory processes as the alternate interpretation of the figure needs to be suppressed (Helmholtz, 1962). Consequently, inhibitory mechanisms are responsible for stabilising one

interpretation over another (Girgus et al., 1977; Fisher, 1967). Therefore the increased reversal rate shown in Study 2 fits the prediction of impaired inhibitory mechanisms as alcohol reduced the ability to stabilise reversals.

Although, the results of Study 2 support other studies showing an alcohol induced impairment of inhibitory processes, the effect of alcohol does not seem to be quite so straightforward. In particular, the alcohol effect is not seen for both figure types, but only with the Face-vase illusion. Further complications arise from the finding that the effect of alcohol on the Face-vase figure was seen only for the biased versions; the effect was much smaller for the unbiassed versions. Similarly, different biasing manipulations previously shown to stabilise reversal rate revealed different effects in response to alcohol, which is interesting given that the removal of inhibition ought consistently to increase reversals.

It seems that the types of biasing used in these figures is responsible for the different effects of alcohol between the figure types. Whereas the biasing used for the Necker cube effectively strengthens one interpretation over the other, the biasing used for the Face-vase illusion seems to alter the absolute strength of the interpretations, rather than just the balance between the two possible interpretations. When the biasing manipulations used in Study 4 used a different type of manipulation that did not change the physical properties of the stimulus but only the relative strength of the two interpretations, no effect of alcohol was found. Therefore, it seems that alcohol is most effective when the two interpretations are each very salient.

One might argue that the group differences found in Study 2 might reflect the limited resources available for accurately processing information following alcohol. As capacity limitations on information processing are revealed when alternating between two tasks, the alternation between the two interpretations of the figure might also encounter similar capacity limitations. However, the capacity limitation argument suggests such impairment is evident by a slowing of response time, which is assumed to reflect delay between completing the first task and beginning the second (Johnston & Heinz, 1978). In which case, the capacity limitation argument might predict that reversal rate would be lower than placebo following alcohol as the limited resources available for processing the information would result in a time delay, thus reducing reversal rate. Also, Reisberg (1983) and Reisberg and O'Shaughnessy (1984) have shown that cognitive load reduces reversal rate. Given that reversal rate increased following alcohol, a capacity limitation account cannot readily account for the findings.

It is also possible that the increased reversal rate shown by the alcohol group in Study 2 is due to drug-related increases in exploratory eye movements, and that the addition of the fixation point in Study 4 simply reduced eye movements. This raises the possibility that one effect of alcohol may be indirect, acting to increase or decrease the tendency to explore the stimulus under conditions of bias, so that alcohol has no effect in Study 4 because eye movements were reduced by the addition of a fixation cross to the stimulus. But reversal rate also decreased in Study 5, where the stimuli were presented intermittently but participants could still make voluntary eye movements, so it seems unlikely that the results can be explained solely by eye movements.

Alcohol is known to have different effects depending upon the dose of alcohol that is administered. Typically, studies that have used low doses of alcohol fail to produce consistent effects of alcohol on similar measures of attention. Whereas those studies that have used higher doses of alcohol typically report alcohol-related impairment in performance. It therefore seems possible that some of the differences between these studies can be accounted for by different individual responsiveness to the alcohol doses. However, comparison of the breath alcohol levels across the studies presented within this thesis suggests that possible differences in response to the doses cannot account for the differing results. In all studies the breath alcohol levels reported are comparable.

These studies show that the effect of alcohol does not appear to be as simple as previous studies might imply. Alcohol does not result in more figure reversals simply because inhibition is weakened, because no effect of alcohol was seen in Studies 4 and 5. Instead, the effect of alcohol on reversals seems to depend upon the precise nature of the stimulus. One difference between Study 2 and Studies 4 and 5 is that the type of biasing manipulation used in Study 2 seems to alter the absolute salience of the figure, whereas in Studies 4 and 5 the biasing alters only the relative strength of the interpretation. This suggests that one effect of alcohol is selectively to impair the processing of absolute saliency information. The results of Study 6 also suggest that alcohol affects the ability to processes absolute saliency information. The prior presentation of a semantically meaningful prime failed to bias the initial interpretation and reversal rate following alcohol. Similarly, Study 7 shows that the alcohol group were unable to inhibit absolute saliency information when it was detrimental to performing an effective search strategy. This further suggests that the effect of alcohol is to impair the ability to accurately

process absolute saliency information. Such factors reflect the influence of higher order cognitive processes on figure reversals.

Another possible explanation for the alcohol-related impairments found in Study 6 and Study 7 is that alcohol impaired the memory processes responsible for holding information about the prime (Study 6) and the cue (Study 7). However, the effect found in Study 7 was restricted to the intentional control condition, no global deficit was evident. Furthermore, it is hard to see how an alcohol-induced impairment of memory process can account for the results of Study 2. As a result, it seems unlikely that the results of Study 6 and Study 7 are caused by alcohol-related memory impairments.

Support for the suggestion that alcohol impairs the processing of absolute saliency comes from the observation in visual attention research that different types of biasing are processed in different areas of the brain (Treisman & Sato, 1990; Wolfe, 1994; Itti & Koch, 2000; Beck & Kastner, 2009). As alcohol has a selective effect on certain areas of the brain (Kähkönen et al., 2001; Volkowa et al., 2008), and these areas are thought to be responsible for processing absolute saliency information (Balan & Gottlieb, 2006; Goldberg et al., 2006; Buschman & Miller, 2007; Moore et al., 2003; Thompson & Bichot, 2005), then the present set of findings seem to encourage this conclusion. In which case, the lack of an alcohol effect when the figures were biased using fixation points (Study 4) and presented intermittently (Study 5) is not surprising, given that the local saliency information contained within these figures is computed in areas of the brain shown to be unaffected by alcohol.

The results of the present thesis suggest that alcohol selectively impairs higher order cognitive processes involved in figure reversal. In support, attention has been thought to play a role in figure reversals for some time (e.g. Meng & Tong, 2004; Toppino, 2003; Gomez et al., 1995; Horlitz & O'Leary, 1993; Liebert & Burk, 1985; Helmholtz, 1962), and frontal-parietal areas of the brain involved in allocating attentional resources have been shown to become activated when viewing ambiguous figures (Leopold & Logothetis, 1999). Furthermore, some studies provide evidence that the mediation of GABA and its influence on the brain structures involved may account for some of the findings in this thesis. These effects are more pronounced in the parietal and frontal cortex (Pittaluga & Raitieri, 1988), the main structures involved in attentional processing. Similarly, alcohol stimulates the release of ACh in the hippocampus (Henn, 1998), and is thought to influence attention (Warburton & Rusted, 1993). Alcohol is known to have a biphasic effect on ACh release in the prefrontal cortex (Stancampiano et al., 2004; Henn, 1998), low-moderate doses increase ACh release, while higher doses decrease cortical ACh release (Stancampiano et al., 2004). Although lower doses of alcohol seem to produce a facilitatory effect on attentional processes, high alcohol intake inhibits ACh production and impairs performance on attentional tasks (Rossetti et al., 2002; Givens, 1995).

7.3.4. Alcohol effects on ambiguous figure types

The differential activation of cognitive mechanisms has also previously been shown to be dependent upon the type of ambiguous figure that is presented during these studies. A distinction has been drawn in the literature between ambiguous figures that are semantically meaningful, and those that are less semantically meaningful, requiring

only a realignment of perspective rather than a change in meaningful content (Strüber & Stadler, 1999). These studies have shown that when reversals are under volitional control, more figure reversals are reported for the more semantically meaningful figures. However, under passive viewing conditions, a similar number of reversals is reported for both figure types. The difference in reversal rate between the figure types when reversals are under volitional control was taken to suggest that cognitive mechanisms were activated (Peterson et al., 1992) and that attentional resources are able to act more effectively on semantically meaningful ambiguous figures under these circumstances. As a result, it was predicted that the results of Study 2 would show that the placebo group would report more reversals for the Face-vase figure than the Necker cube. Whereas, because alcohol is known to impair attentional mechanisms, a similar number of reversals for both figure types was expected. Alternatively, a similar number of reversals have been reported for both figure types under passive viewing conditions (Peterson et al., 1992). This was taken to suggest that attentional mechanisms are not activated to the same extent under these viewing conditions. The results of Study 2 and Study 3 are broadly compatible with this account. In Study 2 (where intentional processes are activated) alcohol affects only the more semantically meaningful face-vase stimulus, whereas in Study 3 (under passive viewing) it has a similar effect on both stimulus types.

The results of Study 2 also show that more figure reversals were reported when viewing the face-vase figure than the Necker cube figure. This supports the findings of Strüber and Stadler (1999) who found more figure reversals were reported for figures with semantically meaningful content then a change of perspective. This difference in

reversal rate according to the type of figure presented is thought to show that attentional resources are able to act more effectively on semantically meaningful ambiguous figures when the intentional mechanism of control is activated. It was predicted that moderate doses of alcohol would impair the attentional mechanism responsible for the difference in reversal rate depending upon the type of figure presented, and so no difference in reversal rate would be found. However, both the alcohol and control group reported more figure reversals when viewing the Face-vase figure than the Necker cube. This suggests that attentional control mechanisms are able to act more effectively on semantically meaningful figures. In addition, the fact that this difference in reversal rate between the figure types was not affected following alcohol suggests that the mechanism responsible for this is not affected by alcohol when the intentional mechanism of control is activated.

The results of Study 3 show that both the alcohol and the placebo group reported more figure reversals when presented with the more semantically meaningful Face-vase illusion than for the Necker cube illusion. Furthermore, this difference in reversal rate between the figure types was not affected by alcohol. Unlike the findings of Peterson et al. (1992), it appears that attentional resources are able to act more effectively on semantically meaningful ambiguous figures even when the automatic mechanism of control is activated. And so this is the first study to show that cognitive mechanisms may be activated under passive viewing conditions. Interestingly, the results of Study 3 show that the difference in reversal rate between the two figure types was not affected by alcohol. This result is somewhat unexpected given that the difference in reversal rate between the two figure types is thought to reflect differences in the activation of

cognitive mechanisms. It would seem reasonable to assume that alcohol would impair these cognitive mechanisms and so reversal rate would be similar between the two types of figure. Therefore, the current results may indicate that the cognitive mechanism responsible for the difference in reversal rate between figure types is independent to that responsible for the increased reversal rate shown by the alcohol group in Study 2.

The results also suggest that the current distinction in the ambiguous figure literature between bottom-up and top-down processes may not be the best distinction. The different reversal rates found between studies in the present thesis, suggest that different types of sensory level information can have different effects on reversal rate. One possible outcome from the studies presented in this thesis is that the current distinction in the ambiguous figures literature between semantic and geometric figure types may be too simplistic. Similarly, these studies further support the notion that the existing trend do categorise reversals into either "bottom-up" or "top-down" processes may not be the best way of explaining the patterns of results. It seems that the contribution of both processes may be involved in varying degrees depending upon the precise nature of the tasks (e.g. Long & Toppino, 2003). For example, the studies presented within this thesis have shown that some of these factors may be the instructions given to participants, or the nature of the stimuli used, to name a few.

It should be noted that eye movements were not measured in any study presented in this thesis. There has been a long discussion about the role of eye movements as a causal factor for figure reversals (e.g. Toppino, 2003; Ellis & Stark, 1978). And so it is possible that the results obtained in this thesis may be partially caused by differences in

eye-movements. However, studies suggest that eye movements are not always necessary for a reversal to occur (Kornmeier et al., 2009; Kornmeier et al., 2007; Gale & Findlay, 1983), and it is not known whether it is the change of eye movement that results in a reversal or whether it is the reversal itself that causes a change in gaze. The finding that reversals still occur even though eye movements are restricted strongly suggests that eye movements may not be a causal factor for reversals to occur. Thus, studies in this area conclude that any differences in reversal rate can be better explained by differences in the observer's ability to control reversal rate rather than by differences in eye-movements. However, the lack of consensus on this issue may suggest that the role of eye movements in figure reversals warrants closer inspection and measurement in these studies.

7.3.5. Conclusions

These studies suggest that moderate doses of alcohol affect intentional control mechanisms more than automatic control mechanisms but that they do not affect intentional mechanisms in all circumstances. The facilitatory effect of alcohol was evident only for biased versions of the figures and only for the Face-vase stimulus. It also appears that the type of biasing manipulation used is crucial in determining any alcohol-related effect. The conclusion from these studies is that the effects of moderate doses of alcohol are more complex than previously thought. There is some justification for the suggestion that alcohol selectively impairs the intentional mechanism of control due to the impairment of the inhibitory mechanisms involved in selective attention. However, this effect is dependant upon the conscious "set" of the observer, and upon the precise nature of the stimulus. The effects also depend on whether the stimulus

contains obvious local regions that directly determine the absolute interpretation, as well as whether attentional resources are directed towards these local regions.

7.3.1.4. Implications

It is acknowledged that the alcohol effects on inhibition in these basic attention models are in the order of millisecond changes. However, it is also recognised that subtle disturbances at basic levels of attention could have a considerable impact on higher order cognitive and behavioural functions. Many fundamental cognitive and perceptual processes, such as inhibitory influences, are considered to operate in a bottom-up fashion to exert increasing influence at each stage of higher order attentional and cognitive functions (e.g. Barkley, 1997; McClelland & Rumelhart, 1981). For instance, basic inhibitory mechanisms in visual search assessed in these studies could facilitate the operation of working memory by ensuring its contents are updated with new, rather than old, visual information. Thus, although alcohol might produce only a slight disruption in attention-based mechanisms, the disturbance might exert considerable influence on the higher order behavioural functions (i.e. working memory) that rely on those mechanisms. The reduced inhibitory effect represents a mechanism by which alcohol might impair attention-based behaviour. Evidence that alcohol reduces inhibition that normally biases attention away from explored locations provides new insights into how the drug might disrupt attention-based behaviour. The reduced duration of IOR under alcohol suggests that redundant searching of previously explored locations might be more likely to occur under the drug. Unnecessary reacquisition of visual information would slow the rate at which new information could be obtained, and subsequently processed. This redundancy could have the effect of diminishing visual

search efficiency under the drug. Such a propensity to acquire redundant visual information could contribute to alcohol-related slowing effects on information processing (e.g. Carpenter, 1962).

The studies presented within this thesis indicate that both sensory (e.g. shading, fixation, intermittent presentation biases) and cognitive (e.g. volitional control, priming) factors can influence figure reversals. The current argument is that evidence exists for both types of processes, and so a single process is unlikely to account for reversals. This research has shown that the influence of variables that favour either sensory or cognitive level effects in figure reversal can be affected by a moderate dose of alcohol depending on the particular viewing conditions. By establishing differences in the pattern of results, this thesis provides strong evidence for the involvement of multiple processes in the perception of ambiguous figures by revealing how sensory or cognitive processes may be more or less evident depending on viewing conditions. Although not the primary aim of this thesis, it is hoped that the research contained within it can add further to the conjoined role of both processes revealed through ambiguous figures. The neural process underlying the perception of an ambiguous figure can be modulated by several controlled and automatic factors independently and at several processing stages. It is therefore not surprising that, though the notion that alcohol impairs inhibitory processes can explain some of the results reported here, we need a much better understanding of the role of inhibition in mediating the interactions between these many processes, before such a general idea can explain all of them.

7.4. AREAS FOR FUTURE RESEARCH

The distinction between behaviours dependent upon automatic and controlled processes also might be valuable in understanding the effects of other drugs. Other CNS depressant drugs and CNS stimulants, which also affect attention, might also have differential effects on attention-based activities depending on the involvement of automatic and controlled mechanisms in the selection of attention. Of particular interest are the well-documented facilitating effects of psychostimulants on selective attention and the possibility that such facilitation depends on the degree to which ignoring distraction depends on automatic or intentional inhibitory mechanisms.

The suggestion that different areas of the brain might be responsible for processing absolute and local saliency information warrants further investigation. Although research has shown that alcohol has an effect on the parietal and pre-frontal brain areas and less effect on the occipital lobes, the suggestion that this distinction is responsible for the different set of results found in this thesis is merely speculative at this time. Studies looking at brain activation whilst performing similar studies presented within this thesis may offer further support for these claims.

The results from Study 6 suggest that the inhibitory mechanisms underlying figure reversals may be more likely to reveal group differences during initial viewing periods, rather than over extended viewing periods. It is a possibility that the extended viewing conditions used in this thesis mask some of the effects of alcohol. Consequently, future studies may benefit from using brief viewing periods to see whether the time course of reversals is also affected.

Finally, there is research to suggest that binge drinkers may be more susceptible to cognitive impairment, and that binge drinking may result in greater damage to the brain. It has been found that binge drinkers perform worse than non-bingers on some cognitive tasks (Weissenborn & Duka, 2003). The studies presented within this thesis matched the groups on the AUQ outcome measures, although differences between patterns of drinking in the alcohol group were not looked at. It is possible, that interpreting the results reported here may be complicated by the different drinking patterns of the participants used in different studies. Future studies may wish to separate binge drinkers from non-binge drinkers and measure both groups' performance on the ambiguous figures tests, as well as using the separate placebo condition employed throughout this thesis.

7.5. SUMMARY

Although the results further highlight conditions where moderate doses of alcohol impair performance on some tasks and not others, at this stage, the results obtained may only be considered to be suggestive rather than conclusive. The results obtained are not exhaustive, and so it is possible that slight methodological changes might produce different results. Consequently, the studies presented within this thesis warrant further investigation and replication. The findings nonetheless add to the collective understanding of the acute effects of alcohol consumption on cognitive processing and attention. For example, they lend further support to effects of alcohol on inhibitory mechanisms. But alcohol does not result in more figure reversals simply because inhibition is weakened; its effects seem to depend upon the precise nature of the

stimulus, upon the relative and absolute strengths of the two interpretations, and upon the specific experimental conditions. These findings are clearly contrary to a simple account based on reduced inhibition.

APPENDIX 1

Lifestyle Questionnaire

INSTRUCTIONS

The following questions are optional. If you do not understand a question, please ask the experimenter. All answers will be treated as strictly confidential. Please circle the appropriate response or write in your answer where indicated:

1. How old are you? Age_____ yrs

2. Gender (circle): Male Female

3. Which of the following educational qualifications do you have? (Tick)

a. GCSEs/'O'-levels ☐

b. AS and/or A-levels ☐

c. Bachelor's degree ☐

d. Postgraduate degree (Masters or Ph.D.) ☐

e. Other (please specify) _____

4. Number of years in full-time education.

5. Do you drink alcohol? Yes No

If yes, please indicate how many units of alcohol you drink per week, on average *(1 unit = ½ pint beer, lager or cider; 1 small glass of wine; or 1 single measure of spirits)*:

_____

6. Do you smoke cigarettes? Yes No

If yes, how many cigarettes do you smoke per day, on average? _____

When did you last have a cigarette? _____ Hours/minutes ago

7. How many cups of tea do you have in an ordinary day? _____

8. How many cups of coffee do you have in an ordinary day? _____

When did you last have a cup of tea/coffee? _____ Hours/minutes ago

9. If you are currently taking any prescribed medication (excluding the contraceptive pill), then please list below (*optional)*:

_____

10. If you are currently taking any non-prescribed medication, then please list:

_____

11. Have you ever been diagnosed with a psychiatric illness? Yes No

If yes, please describe _____

12. Are you currently taking any of the following:

Anti-psychotic medication? Yes No

Anti-depressant medication? Yes No

Other drug(s) for psychiatric problems Yes No

13. Have you ever suffered a serious head injury (with loss of consciousness)?

Yes No

If yes, please describe _____

14. Do you currently suffer from a neurological disorder (e.g. Epilepsy)?

Yes No

If yes, please describe _____

15. Have you ever been diagnosed as drug or alcohol-dependent, or have you ever been treated for such conditions?

Yes No

If yes, please describe _____

16. Have you ever taken recreational drugs? Yes No

If you answered yes, please fill in the table below. If you have never taken the drug DO NOT tick any box. *If you have taken a drug not listed, please list them at the bottom of the table.*

Drug	Age at first use	Average frequency of current use: days per month	Duration at this frequency (months)	Typical amount consumed per occasion	Time since last used (days)
Amphetamine					
Amyl/Butyl Nitrate					
Cannabis					
Cocaine					
Ecstasy					
GHB					
Heroin					
LSD					
Magic mushrooms					
Solvents					

APPENDIX 2

Alcohol Use Questionnaire (AUQ; Townshend & Duka, 2002)

The following questions ask about your habitual use of various types of alcoholic drinks. Please consider you drinking for the last 6 months in answering the questions and take your time to give an accurate answer to each question.

1. On how many days per week do you drink wine, or any wine type product e.g. sherry, port, martini (at least one small glass)? _____ Please state your usual brand(s) _____
2. On those days you do drink wine (or similar), about how many glasses (pub measures) do you drink? _____ If you are unsure, please estimate the number of bottles or parts of a bottle _____
3. How many glasses (pub measure) of wine do you have in a week, in total? _____
4. On how many days per week do you drink beer or cider (at least half a pint)? _____Please state usual brand (e.g. Carlsberg special, White Lightning etc) _____
5. On those days you do drink beer/cider, about how many pints do you typically have? _____
6. How many points of beer/cider do you drink in a week, in total? _____
7. On how many days per week do you drink spirits (Whisky, vodka, gin, rum etc – but not beer or wine)? _____ Please state usual brand (e.g. Smirnoff Blue Label) _____

8. On those days you do drink spirits, about how many shorts (pub measure) do you typically have? _____ If unsure, please estimate number of bottles or parts of a bottle _____
9. How many drinks of spirits do you have in a week, in total? _____
10. When you drink, how fast do you drink? (Here, a drink is a glass of wine, a pint of beer, or a shot of spirits, straight or mixed). Please circle the correct response.

    Drinks per hour: 7+ 6 5 4 3 2 1

    1 drink in 2 hours

    1 drink in 3 or more hours
11. How many times have you been drunk in the last 6 months? By 'drunk' we men loss of co-ordination, nausea, and/or inability to speak clearly.

    _____
12. What percentage of the times that you do drink do you get drunk? _____

APPENDIX 3

Michigan Alcohol Screening Test (MAST; Selzer, 1971)

Please answer 'Yes' or 'No' to each of the following questions

1. Do you feel you are a normal drinker? ('Normal' – drink as much or less than most other people)

2. Have you ever awakened the morning after some drinking the night before and found that you could not remember a part of the evening?

3. Does any near relative or close friend ever worry or complain about your drinking?

4. Can you stop drinking without difficulty after one or two drinks?

5. Do you ever feel guilty about your drinking?

6. Have you ever attended a meeting of Alcoholics Anonymous (AA)?

7. Have you ever gotten into physical fights when drinking?

8. Has drinking ever created problems between you and a near relative or close friend?

9. Has any family member or close friend gone to anyone for help about your drinking?

10. Have you ever lost friends because of your drinking?

11. Have you ever gotten into trouble at work because of drinking?

12. Have you ever lost a job because of drinking?

13. Have you ever neglected your obligations, your family, or your work/studies for two or more days in a row because you were drinking?

14. Do you drink before noon fairly often?

15. Have you ever been told you have liver trouble such as cirrhosis?

16. After heavy drinking have you ever had delirium tremens (D.T's), severe shaking, visual or auditory (hearing) hallucinations?

17. Have you ever gone to anyone for help about your drinking?

18. Have you ever been hospitalised because of drinking?

19. Has your drinking ever resulted in your being hospitalised in a psychiatric ward?

20. Have you ever gone to any doctor, social worker, clergyman or mental health clinic for help with any emotional problem in which drinking was part of the problem?

21. Have you been arrested more than once for driving under the influence of alcohol?

22. Have you ever been arrested, even for a few hours because of other behaviour while drinking? (If yes, how many times)

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