BEHAVIOURAL INHIBITION UNDER ALCOHOL:

EFFECTS OF REINFORCEMENT AND THE SETTING

by

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ABSTRACT

The present research measured the effect of alcohol on inhibitory control (using a go-stop task) when two environmental factors, setting and reinforcement, were manipulated. Administering alcohol in a novel setting, or providing immediate positive reinforcement of inhibitions was predicted to counteract the impairing effect of alcohol on inhibitory control. Seventy-two male social drinkers were randomly assigned to one of eight treatment groups (n=9). Four groups were tested during their first visit to the laboratory (novel setting) whereas the remainder were tested after one drug-free exposure to the laboratory (familiar setting). In Phase 1 of the research, two groups in each setting received alcohol (.62 g/kg) or a placebo and performed the go-stop task with no consequences for task performance. The results showed that the administration of alcohol in a familiar setting impaired inhibitions, whereas alcohol in the novel setting had no significant effect on inhibitions. Furthermore, the setting had no effect on the response inhibitions of groups that expected alcohol but received a placebo. In Phase 2 of the research, three pairs of groups received alcohol and performed the task with either immediate positive reinforcement, or a monetary incentive, or no consequence for inhibitions. In accordance with the hypothesis, the impairing effect of alcohol in the familiar setting was reduced only when immediate positive reinforcement was administered. In contrast, in the novel setting, all groups showed little change from drug-free levels of inhibitory control. No systematic changes in response reaction time (RT) in any of the groups could account for these findings. This research provides the first experimental evidence to show that the effects of alcohol and of reinforcement on inhibitory control of behaviour depend on the novelty-familiarity dimension of the setting. Moreover, the results indicate that a loss of inhibitory control is not an inevitable effect of a moderate dose of alcohol.

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DEDICATION

To Geoff, without your love, support and understanding, this endeavour would not have been possible.

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INTRODUCTION

...O thou invisible spirit of wine,
if thou hast no name to be known by,
let us call thee devil!...
O God, that men should put an enemy
in their mouths to steal away their brains!
that we should, with joy, pleasance, revel
and applause, transform ourselves into beasts!
-William Shakespeare, *Othello* (II, iii)

When in the company of a superior whom we respect, or of a female in whose presence it would be indelicate to get intoxicated, a much greater portion of liquor may be withstood than in societies where no such restraints operate. The mind exercises considerable effect upon drunkenness, and may control it powerfully.
- Robert MacNish (1832), p. 45

Public opinion about whether alcohol is to blame for behavioural transgressions, ranging from socially inappropriate behaviour to violent criminal acts, has fluctuated over time. Colonial Americans did not excuse deviant behaviour under alcohol because they believed that the individual would have committed the same act in a sober state (Critchlow, 1983, 1986). In contrast, the notion that alcohol was to blame for inappropriate behaviour was prominent during the Temperance Movement when alcohol was viewed as "the destroyer of self-control and thus a direct cause of crime and deviant behaviour" (Critchlow, 1983, p. 453). Burgeoning research on alcohol during this time had indicated that the drug could impair the performance of many tasks (Jellinek & MacFarland, 1940) and likely added credence to the public perception of alcohol as the cause of deviant behaviour. Later, MacAndrew & Edgerton (1969) suggested that behavioural responses to alcohol are learned and that alcohol itself does not cause inappropriate behaviour. At the present time, confusion about whether alcohol leads to a loss of self-control still exists and, at times, alcohol's effect on behaviour is hotly debated.

The possibility that alcohol unleashes antisocial behaviour implies that the individual should not be held fully accountable for actions under the drug. Evidence for this opinion has been

provided in studies showing that individuals attribute less blame or responsibility to men who battered their wives, or who committed sexual assault when they were drinking than when the same action was carried out in a sober state (Richardson & Campbell, 1980, 1982). Similarly, participants reviewing scenarios depicting an individual engaged in a socially undesirable behaviour (e.g., insulting someone) or a criminal act (e.g., robbery, assault) attributed less responsibility and blame to an intoxicated, than to a sober perpetrator (Critchlow, 1985). Although it would be logical to reduce the penalty for individuals who are deemed not fully responsible for their behaviour, people tend to assess equally severe punishments for antisocial behaviour, whether or not alcohol is involved (Critchlow, 1985). More recent research also demonstrated that people are unsure about the effects of alcohol on self-control (Wild, 1998).

The continuing debates about whether alcohol plays a causal role in antisocial and criminal acts has had a considerable impact on the justice system. In fact, different countries have different views about the role of alcohol in "causing" criminal behaviour to occur. In England and Germany intoxicated perpetrators are given lesser charges and therefore the penalties are less strict. Courts in Sweden, however, treat intoxicated and non-intoxicated perpetrators the same (Bergman, 1997).

In Canada's court system, the confusion over the effects of alcohol on inhibitory control is illustrated in a landmark case in Montreal, Quebec. In 1994, a man was tried in Quebec for sexually assaulting a 65-year-old woman who was confined to a wheelchair. At the trial the defendant testified that he had been drinking and could not remember anything. In the first ruling, he was acquitted because he had been under the influence of alcohol when he committed the offence and the judge had "a reasonable doubt about whether the accused...possessed the minimal intent necessary to commit the offence" (Henri Daviault v. Her Majesty the Queen, 1994, p.64). The

Crown appealed this decision stating that alcohol could not be used as a defence, and a verdict of guilty was subsequently obtained. However, an appeal to the Supreme Court of Canada resulted in an order for a new trial on the grounds that drunkenness can be used as a defence in sexual assault cases (Sheppard, 1994, Oct 4, 1994, p. B1). In response to this decision, the public rallied around the idea that alcohol consumption should not provide a defence for crimes and that it only provides an excuse for inappropriate behaviour (Kaufman, Oct. 4, 1994). Shortly thereafter, the Supreme Court ruling was overturned and alcohol was no longer a viable defence. Thus, it appears that the courts, like the public at large, have difficulty deciding whether alcohol causes a loss of inhibitory control or whether alcohol consumption simply provides an excuse for inappropriate behaviour. This controversy is likely to continue until more information is available concerning the extent and conditions under which alcohol reduces inhibitory control of behaviour. The provision of evidence on this topic is the major purpose of this thesis.

Pharmacology of alcohol

Early explanations of alcohol-induced loss of behavioural control proposed that alcohol disinhibited particular areas of the brain and caused a release of immoral behaviours (e.g., McCorkindale, 1926). However, no evidence to support this notion has been obtained. In fact, research to date indicates that the effects of alcohol on the brain and its functions are very complex and still not completely understood (Julien, 1998; McKim, 1996; National Institute on Alcohol Abuse and Alcoholism [NIAAA], 1993).

Alcohol is a psychoactive drug and is classified as a depressant, sedative/hypnotic drug (Brands, Sproule & Marshman, 1998; Julien, 1998). When ingested, it is distributed widely throughout brain tissue, but unlike most other drugs, alcohol does not exert its effect by interacting

with a particular receptor site (Leonard, 1997; McKim, 1996; Hunt, 1993). Instead alcohol alters the lipid layer of cell membranes and these changes in turn affect many aspects of neuronal functioning (Dildy-Mayfield & Harris, 1995; Hunt, 1993; Julien, 1998; McKim, 1996; NIAAA, 1993; Rall, 1991). Research in neuropharmacology has indicated that the functions of several neurotransmitters are affected by alcohol (Eckardt et al., 1998; Julien, 1998; NIAAA, 1993).

The major inhibitory neurotransmitter in the human brain is called GABA (Julien, 1998; NIAAA, 1993; Rall, 1991) and acute doses of alcohol have been found to increase the activity of GABA, possibly by increasing GABA mediated synaptic transmission at the $GABA_A$ receptor (Julien, 1998; Leonard, 1997; NIAAA, 1993). Benzodiazepines and barbiturates have sedative, anaesthetic and anxiolytic effects that also have been linked to the $GABA_A$ receptor (NIAAA, 1993). Because these drugs have some effects similar to those of alcohol, it has been suggested that alcohol's sedative, anaesthetic and anti-anxiety effects also might be due to the interference between GABA and the $GABA_A$ receptor (Dildy-Mayfield & Harris, 1995; NIAAA, 1993). Recently, it has been suggested that the $GABA_B$ receptor might play a role in the alcohol-GABA interaction (Eckardt et al., 1998). The action of alcohol on GABA also has an impact on other transmitter systems such as the cholinergic and dopaminergic systems (Julien, 1998).

In contrast to GABA, glutamate is the main excitatory neurotransmitter (NIAAA, 1993). Its excitatory actions are produced by interacting with three types of receptors. The action of glutamate at two of these receptor sites, NMDA (N-methyl-D-aspartate) and AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid) is reduced in the presence of alcohol (Dildy-Mayfield & Harris, 1995; Julien, 1998; McKim, 1996; NIAAA, 1993). As a result, the ability of cells to become excited is reduced. The NMDA receptors are thought to play a role in memory, and have

been implicated in cognitive impairment, amnesia and decreased ability to learn new information (NIAAA, 1993). Thus, it has been suggested that the interference of alcohol between glutamate and its NMDA receptor might play a role in amnesia and cognitive impairment.

Dopaminergic neurotransmitter systems are thought to play a role in the reinforcing (i.e., pleasurable, rewarding) effects of stimuli such as food, water and addictive drugs (Wise, 1988). Some evidence suggests that alcohol might be associated with a rise in brain level of dopamine (Eckardt et al., 1998; Julien, 1998; Leonard, 1997) and activation of the dopaminergic D2 receptors (Dildy-Mayfield & Harris, 1995; NIAAA, 1993). Other research suggests a role for serotonin and endogenous opioid systems along with dopamine in the reinforcing effects of alcohol (Eckardt et al., 1998).

Research on serotonin (5-HT) has suggested a possible link between alcohol and impulsive or aggressive behaviour (LeMarquand, Pihl & Benkelfat, 1994). The concentration of serotonin in the brain can be estimated by the concentration of its metabolite, 5-hydroxyindoleacetic acid (5-HIAA) (Pihl & Peterson, 1993). Research has revealed that abnormally low levels of this metabolite have been found in aggressive monkeys (Higley et al., 1992), and other research has suggested that low levels of 5-HIAA might be linked to aggressive and impulsive behaviour in humans (Linnoila et al., 1983). This has lead to speculation that reduced levels of 5-HT might play a causal role in the display of impulsive and aggressive behaviour (Pihl & Peterson, 1993). Some research with humans suggests that the administration of an acute dose of alcohol is associated with lower 5-HT levels in the brain (LeMarquand et al., 1994). Although these observations suggest that alcohol might cause impulsive and aggressive behaviour by lowering 5-HT levels, such a causal relationship has not yet been demonstrated. In fact, Pihl & Peterson (1993) argued that an interaction between

alcohol-induced changes in a number of neurotransmitters, such as dopamine, GABA and 5-HT, might cause impulsive and aggressive behaviour. Although investigators have attempted to find consistent relationships between the ingestion of alcohol and serotonin levels, the results are contradictory (Pihl & Peterson, 1993).

A few studies of the effect of an acute dose of alcohol on brain function in humans have used brain imaging techniques such as Positron Emission Tomography (PET) scans. Research that used PET scans has revealed that low and moderate doses of alcohol were associated with a general decrease in glucose metabolism (deWit, Metz, Wagner & Cooper, 1990). Others have investigated the effect of alcohol on regional cerebral blood flow and found an increase in cortical blood flow after the ingestion of a moderate dose of alcohol and inferred an increase in activity in the brain (e.g., Newlin, Golden, Quaife & Graber, 1982). It has been suggested, however, that the increase in cortical blood flow might be due to alcohol's vasodilatory effects rather than a reflection of actual cerebral activity (e.g., deWit et al., 1990). More research in this area is required to gain a better understanding of the effects of an acute dose of alcohol on neuronal activity.

In summary, progress has been made in identifying the effect of alcohol on neurotransmitters and neuronal functions. However, most of this research continues to highlight the complex and widespread effects of alcohol on brain functioning. Certainly, no research has yet demonstrated any causal relationship between specific physiological or neurological changes induced by a dose of alcohol and any corresponding changes in behaviour (Hunt, 1993; Ito, Miller & Pollock, 1996). On the other hand, much research shows that alcohol consumption is correlated with aggressive, violent and inappropriate behaviour (e.g., Forrest & Gordon, 1990; Murdoch, Pihl & Ross, 1990; Pernanen, 1976; Solnick and Heminway, 1994). The correlational nature of these

findings does not allow many definitive conclusions to be drawn about alcohol's causal role in aggressive behaviour (Murdoch, Pihl & Ross, 1990). Thus, in the absence of evidence for a causal pharmacological effect of alcohol on aggressive or other deviant behaviour, other investigators have conducted behavioural experiments examining alcohol-induced aggression.

Alcohol-induced aggression

One of the earliest explanations for antisocial aggressive behaviour under alcohol proposed that individuals are inherently aggressive, but anxiety about the punitive social and personal consequences of aggression normally inhibit such actions. Thus alcohol was assumed to impair or remove these inhibitions and unleash aggressive tendencies (Bennett, Buss, & Carpenter, 1969). Although this explanation was specific to aggressive behaviour, modern versions have expanded to include any behaviour that might normally be inhibited by fear or anxiety about adverse consequences. For example, it has been proposed that the expression of deviant, extreme actions under alcohol represents a loss of inhibitory control (Steele & Southwick, 1985). Thus an increase in aggression or other extreme, inappropriate behaviours under alcohol were all attributed directly to the effect of alcohol. Such changes in behaviour are loosely referred to as "disinhibition", and are assumed to reflect a reduced ability to inhibit an ongoing response (e.g., Bennett et al., 1969; Hull & Bond, 1986).

A considerable amount of laboratory research has been designed to test the hypothesis that alcohol causes disinhibition that results in aggressive behaviour (e.g., Bennett et al., 1969; Stuntich & Taylor, 1972; Gantner & Taylor, 1992). Much of this research has been conducted with social drinkers who received alcohol or placebo beverages and then engaged in various learning or game situations with a confederate who posed as another participant. To advance learning, or to win the

game, the participant was required to administer electric shocks to the fictitious participant. Aggression in this situation was measured by an increase in the intensity of electric shocks the participant administered (e.g., Bennett et al., 1969; Stuntich & Taylor, 1972). In this research, the increase in the intensity of aggressive behaviour under alcohol was a proxy measure of diminished inhibitory control.

Numerous experiments using this shock paradigm have measured aggressive responses under doses of alcohol that would yield peak blood alcohol levels ranging from about 40 mg/100 ml to more than 100 mg/100 ml. Research using a learning situation led to the conclusion that aggression was not a consequence of the pharmacological action of alcohol (Bennett et al., 1969). However, other investigators who administered similar doses of alcohol in a game situation concluded that more aggression was displayed under alcohol than under a placebo (Gantner & Taylor, 1992; Stuntich & Taylor, 1972; Taylor, Schmutte & Leonard, 1977).

These conflicting findings raised the possibility that the differences in the perceived threat in the learning and game situations might play a role in the different effects of alcohol (Stuntich & Taylor, 1972). In the learning situation, participants could shock the fictitious participant and knew that they would not themselves receive any shocks. In contrast, a participant in the game paradigm could give and receive shocks that increased in intensity as the game continued. Two studies have manipulated the level of perceived threat in the game situation by removing the participant's electrodes (Gantner & Taylor, 1992) or by allowing the participant to overhear the fictitious opponent stating that he was not comfortable shocking his partner (Taylor, Gammon & Capasso, 1976). However, the findings of these two studies were also inconsistent. Taylor et al. (1976) found that alcohol participants were more aggressive than placebo participants when they were threatened

with shock, but there was no difference in aggression between the alcohol and placebo groups when the threat of shock was removed. In contrast, Gantner & Taylor's (1992) investigation revealed that with or without the threat of shock, more aggression was displayed under alcohol.

In summary, research using a shock paradigm to investigate alcohol-induced aggression has shown that greater aggression under the drug is occasionally, but not reliably, displayed. Thus, rather than supporting the hypothesis that alcohol causes an increase in aggressive behaviour, the findings point to the possibility that the degree of aggression results from an interaction between the effects of the drug and the environmental conditions. However, the findings also fail to identify the crucial environmental events in the situation that interact with alcohol to reliably produce aggressive behaviour.

Even if alcohol were found to consistently cause an increase in aggression, this evidence alone would not reveal whether alcohol directly increases aggressivity, or whether the drug directly disrupts inhibitory control of behaviour. This latter possibility implies that alcohol's "disinhibiting" effect should presumably be evident in a range of behaviours in addition to aggression.

Disinhibition of social behaviour under alcohol

Studies of disinhibiting effects of alcohol on a wide variety of behaviours have continued to use an increase in the intensity of a response to infer a lack of inhibitory control. In large part these studies have been influenced by the notion that disinhibited extreme or deviant social behaviour under alcohol results from impaired information processing of cues in the environment (Pernanen, 1976). Others have argued that impaired inhibitory control of behaviour is specific to cues concerning the punitive consequences of behaviour and that alcohol impairs the ability to foresee the adverse consequences of an action (Steele & Southwick, 1985). These investigators

advanced this argument on the basis of a meta-analysis of studies of alcohol effects on various social behaviours (e.g., aggression, self-disclosure, risk-taking). The situations in the studies were classified in terms of high or low conflict on the basis of the presence or absence of "salient" cues for strong reward and punishment of the same response. The results indicated that more extreme behaviour was observed in situations in which both cues were present and of equal strength (i.e., high conflict situations).

Although Steele & Southwick (1985) attributed these effects to the impairing effect of alcohol on the cognitive ability to foresee the negative consequences of behaviour, the findings are open to another interpretation. Most experiments testing the effect of alcohol on social behaviours have deliberately or inadvertently included signals for, or actual rewards or punishments for the behaviour. The motivational properties of such events might greatly determine the behaviour that will be displayed (e.g., Conger, 1951). Thus, the results might be contaminated by the consequences of behaviour under alcohol.

In summary, studies of the effect of alcohol on social behaviours have commonly attributed the display of more extreme behaviour to a disinhibiting effect of the drug, but this interpretation is questionable. The results might be due to a "disinhibiting" effect of alcohol, or to the interaction of alcohol and environmental consequences for behaviour. Moreover, the findings rest on inferring impaired inhibitory control from an increase in the intensity of a response under alcohol rather than a direct observation of its inhibition. An adequate experimental paradigm testing inhibitory control under alcohol would require a direct measure of the ability to inhibit an ongoing response, when environmental consequences of behaviour are manipulated. Possibilities for an appropriate experimental paradigm that meets these requirements are discussed in the next section.

Paradigms testing models of inhibitory control

An influential model of inhibitory control has been proposed by Gray (1982). Gray postulated that a Behavioural Inhibition System (BIS) is responsible for inhibiting behaviour. In contrast, the Behavioural Activation System (BAS) is considered to be responsible for initiating or activating behaviour (Fowles, 1987). According to this theory, behavioural control depends on two processes: the BAS and the BIS. In theory, certain stimuli in the environment are believed to activate each system. For example, the BAS is activated by stimuli associated with reward, whereas the BIS is activated by stimuli associated with punishment or frustration. Punishing environmental stimuli are hypothesized to increase anxiety, which activates the BIS and stops a response. In contrast, stimuli that reduce anxiety should reduce the action of the BIS, allowing the BAS to dominate and responding to occur.

Gray's theory implied that anxiolytic drugs would reduce anxiety which would in turn decrease BIS action and increase responding in spite of punishment. In this experimental design, the continuation of responding (i.e., lever pressing by animals) was used to infer the reduced operation of the BIS. In other words, increased responding was used as a proxy measure of reduced inhibitory control. Thus, the research used to explain and understand this model provides no direct measure of actual response inhibition.

An experimental paradigm based on the BIS has been devised to test humans thought to have deficits in inhibitory control (e.g., psychopaths) (Newman, Widom & Nathan, 1985; Patterson & Newman, 1993). In this experimental paradigm, individuals performed a "go/no-go" task that presented "go" and "no-go" signals on separate trials. When responses to go-signal trials were rewarded, and responses to no-go signal trials were punished, continued responding to no-go trials

in spite of punishment was considered a loss of inhibitory control (i.e., a reduction in the operation of the BIS was inferred). Once again, increased responding was used was a proxy measure of response inhibition. Furthermore, the measures of separate responses on go (reward) and no-go (punishment) trials provide measures of behavioural sensitivity to reward and punishment, respectively, but these measures do not provide information about the ability to inhibit an ongoing response. Thus, it appears that experimental paradigms designed to test the BIS model are not suitable to examine the effect of alcohol on the inhibition of an ongoing response.

A Race Model

An alternative model of inhibitory control has been advanced by Logan and his colleagues (Logan & Cowan, 1984; Logan, Cowan & Davis, 1984). This model assumes that go- and stop-signals activate two separate processes, a go process and a stop process. In the race model, inhibitory control is construed as a "horse-race" between these two processes. Thus when go- and stop-signals are presented for a given response, the two processes are activated and "race" to completion. If the go process finishes before the stop process, then the response will be displayed, but if the stop process finishes first, the response will be inhibited.

An experimental stop-signal paradigm has been developed to test this model of cognitive inhibitory control. In this paradigm, individuals are engaged in responding to go-signals that are occasionally interrupted by stop-signals that tell them to inhibit the response. Inhibitory control is measured by the number of successful inhibitions to go-signals when stop-signals are presented. The most commonly used paradigm, based on the race model, is a computerized task in which go-signals are letters presented on the monitor, and a tone serves as the stop-signal. However, the stop-signal paradigm has also been applied to a variety of tasks such as typewriting; eye, hand and

arm movements; speech; and simple and choice reaction-time tasks (Logan & Cowan, 1984). Reviews of the findings have provided support for the race model (Logan, 1994).

The paradigm has also been used in clinical assessments of inhibitory control of behaviour among children who display impulsivity and hyperactivity that characterize attention deficit hyperactivity disorders (ADHD). This research has found that children diagnosed with ADHD display fewer inhibitions on a stop-signal task than do normal controls, and the drug methylphenidate (Ritalin) that is used to treat ADHD increases the inhibitions that ADHD children display on the task (Schachar & Logan, 1990; Schachar, Tannock, Marriot & Logan, 1995; Tannock, Schachar, Carr, Chajczyk & Logan, 1989; Tannock, Schachar & Logan, 1995).

The stop-signal paradigm appears to provide the tool that meets the requirements needed to test the effect of alcohol on the inhibition of an ongoing response[1]. The go-stop task directly measures the inhibition of an ongoing response (i.e., the number of times a response is withheld to go-signals when stop-signals are presented). A computerized go-stop task allows individuals to perform the task alone in a room so that no environmental consequences or feedback are provided. This ensures that any environmental consequences (e.g., reward/punishment) or interpersonal variables that might affect task performance can be excluded.

Alcohol and behavioural inhibition

The ability of the go-stop task paradigm to test the effect of a moderate dose of alcohol on response inhibition has been demonstrated (Mulvihill, 1995; Mulvihill et al., 1997). In this research, groups of male and female social drinkers performed a computerized go-stop task that

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[1] The task developed from the stop-signal paradigm has been referred to as a "stopping task" and a "go-stop task" (Tannock et al., 1989; Mulvihill, Skilling & Vogel-Sprott, 1997). It will be referred to as a go-stop task in this thesis.

engaged them in responding to repeated go-signals, and occasionally presented stop-signals that meant they were to withhold their response to the go-signal. Inhibitory control was directly measured by the number of times the ongoing response was withheld when the stop-signal was presented and the response to go-signals was measured by reaction time (RT). The results showed that alcohol reduced response inhibitions (to a similar degree in men and women), and did not affect their response RT to go-signals. In contrast, groups that expected alcohol but received a placebo, or received no beverage at all showed no significant change in inhibitions or response RT. The finding that a moderate dose of alcohol (mean peak BAC of 73 mg/100ml) did not affect response RT on the go-stop task is not unusual as RT is typically not affected until BACs are over 80 mg/100 ml (e.g., Mitchell, 1985). The fact that this dose of alcohol reduced response inhibitions indicated that alcohol can selectively target the stop process, and is consistent with the race model assumption of separate go and stop processes.

The results of this research provided the first demonstration that alcohol can reduce the ability to inhibit an ongoing response. Although this evidence is in line with the notion that alcohol impairs inhibitory control and disinhibits behaviour, it does not necessarily mean that alcohol will inevitably disinhibit behaviour in all circumstances. Indeed, considerable research has pointed to the involvement of environmental factors (i.e., the consequences for behaviour under alcohol) as a possible determinant of the display and extent of inappropriate behaviour under alcohol (e.g., Ito et al., 1996; Steele & Southwick, 1985). A test of this possibility requires an experimental procedure that measures drug-free baseline inhibitory control and compares it to inhibitory control under alcohol when environmental consequences for inhibition are present or absent. The stop-signal paradigm is well suited for this purpose because the consequences for inhibitory control can

be withheld to provide a measure of the effect of alcohol. In addition, environmental consequences can be introduced and systematically manipulated to evaluate their causal influence on response inhibition under alcohol. Much research in behavioural pharmacology has investigated the influence of environmental consequences on behaviour under drugs, and findings in this area provide a basis for predicting the effect of environmental consequences on response inhibition under alcohol.

Alcohol, environmental consequences and behaviour

Early experiments calling attention to the importance of environmental consequences of behaviour under a drug showed that the pecking response of pigeons could be increased or decreased under a dose of pentobarbital depending upon the schedule of reinforcement (i.e., the temporal occurrence and frequency of food) (Dews, 1955). Reviews of ensuing research over the next three decades confirmed that the behavioural effect of a drug can be altered by positive and negative reinforcement, as well as by punishment (e.g., Branch, 1984). Unfortunately, the findings from the operant conditioning research have not led to any theoretical interpretation to account for these effects.

In contrast, associative learning theory has contributed importantly to understanding how environmental events in a situation affect behaviour (e.g., Bolles, 1972). In theory, a reliable relationship between two events, A and B, provides an opportunity to learn that A predicts B. The learning of this relationship provides the bases for a cognitive expectancy: when A is presented, B is expected to follow. These learned expectations are assumed to guide behaviour. Basic research using classical conditioning and instrumental training procedures have provided considerable support for this theory (e.g., Rescorla, 1990).

The application of associative learning principles to behaviour under a drug have proven

similarly fruitful. Research using a classical conditioning paradigm has shown that environmental events preceding repeated administrations of many types of drugs can affect the intensity of the behavioural effect of a drug. For example, research in which repeated doses of a drug were administered until animals displayed behavioural tolerance has shown that greater tolerance is subsequently displayed when the drug is expected (i.e., the cues for the drug are present) than when the drug is not expected (i.e., the cues for the drug are absent) (Siegel, 1983, 1989).

Associative learning theory also assumes that any environmental outcome reliably associated with a response can result in the expectation of this outcome whenever the response is displayed (e.g., Bolles, 1972; Rescorla, 1990). A great deal of research guided by this theory has investigated the behavioural effect of a moderate dose of alcohol in humans when the expected outcome of the response is manipulated. This research has shown that when drinkers are trained to expect some reward for resisting psychomotor impairment under alcohol (e.g., by positively reinforcing a sober level of performance) they show a reduction in impairment (i.e., tolerance) (e.g., Beirness & Vogel-Sprott, 1984; Mann & Vogel-Sprott, 1981; Myrsten, Lamble, Frankenhaeuser, & Lundberg, 1979; Sdao-Jarvie & Vogel-Sprott, 1991; Zack & Vogel-Sprott, 1993). The reinforcers used in this research have included money and approving verbal feedback. Research has indicated that these reinforcers are equally effective in promoting tolerance to alcohol-induced impairment (e.g., Sdao-Jarvie, 1991). The associative learning explanation of such effects proposes that associating a rewarding consequence with a drug-tolerant response provides an opportunity to learn this relationship. This information in turn forms a basis for expecting a reward for a sober (i.e., drug-free) level of performance under the drug. Support for this interpretation has been provided by showing that tolerance is not displayed in situations that make it difficult to acquire information

about the relationship between drug-tolerant performance and positive reinforcement. For example, when a reward is administered randomly, or delayed with respect to a drug-tolerant response, less resistance to alcohol-induced impairment is evident (e.g., Beirness & Vogel-Sprott, 1984; Sdao-Jarvie & Vogel-Sprott, 1991).

Some recent research has extended the evidence of the effect of reinforcement under a moderate dose of alcohol to a cognitive information-processing task (Fillmore & Vogel-Sprott, 1997). This research compared the effect of a moderate dose of alcohol on task performance in three groups of drinkers. The groups received either immediate monetary reinforcement for resisting alcohol-induced impairment, an equal monetary incentive for resisting impairment under alcohol, but the money was delayed until the experiment concluded, or no reinforcement. The results showed that immediate reinforcement counteracted the alcohol-induced impairment of information-processing performance, whereas the other two treatments resulted in considerable and comparable impairment.

Reviews of this body of research lead to the conclusion that drinkers display the type of response to alcohol that they expect will be rewarded. If drug-tolerant behaviour is reinforced, drinkers resist the impairing effect of alcohol (Vogel-Sprott, 1992, 1997; Vogel-Sprott & Fillmore, 1999).

Summary

Associative learning interpretations of the effects of positively reinforcing sober performance of psychomotor and information-processing tasks under alcohol suggest that positive reinforcement might also reduce the impairing effect of the drug on inhibitory control of behaviour. If this is the case, then immediate positive reinforcement for a sober level of response inhibition

under alcohol should decrease the disinhibiting effect of the drug. This thesis is designed to test this hypothesis using a stop-signal paradigm that manipulates the presence or absence of positive reinforcement for response inhibition under alcohol. In the preliminary investigation, a procedure was developed that manipulated reinforcement and tested the effects of reinforcement on alcohol-induced impairment of inhibitory control. The results of the preliminary work encouraged a further investigation of the reinforcement hypothesis, and suggested that another factor, the novelty of the experimental setting, might also have an impact on the intensity of the effect of alcohol on response inhibition. The main experiment in the thesis was based on the results of the preliminary study and was designed to test: 1) the effect of alcohol on response inhibition in a novel or a familiar setting, and 2) the effect of immediate positive reinforcement, monetary incentive and no reinforcement on response inhibition under alcohol in a novel or a familiar setting.

PRELIMINARY INVESTIGATION

In this work, a procedure was devised for administering reinforcement of inhibitions on a go-stop task, and the effect of reinforcement on response inhibition under a moderate dose of alcohol was also explored. The effect of alcohol (A) on response inhibition was tested in four groups that received different treatments. Previous research on motor skills indicated that immediate reinforcement (R) in the form of money (M) or approving verbal feedback (V) was effective in reducing the effect of alcohol. Both reinforcers were tested in this investigation. One group (ARM) was reinforced with money (25 cents) immediately after every test on which participants displayed at least their drug-free level of response inhibition. Another group (ARV) was immediately reinforced with approving verbal feedback.

In order to control for possible incentive effects of the money, another group (AM) was told they would receive 25 cents for every test under alcohol on which they maintained their drug-free level of response inhibition, and the money would be paid after the experiment was completed. A fourth group (A) served as a control, and performed the task under alcohol without any reinforcement or monetary incentive.

Learning theory suggests that immediate positive reinforcement of a drug-tolerant response provides the best opportunity to learn that this response yields a favourable consequence. The acquisition of this expectation should increase the drug-tolerant response and thus counteract the drug effect. If immediate positive reinforcement weakens the depressing (i.e., impairing) effect of alcohol on response inhibitions, then groups that receive reinforcement (groups ARM and ARV) should show less impairment than groups without reinforcement (groups AM & A). If immediate reinforcement in the form of money or verbal feedback are equally effective in counteracting the

effect of alcohol, then the response inhibitions of groups ARM and ARV should not differ. On the basis of prior research on the effect of a moderate dose of alcohol on the performance of the go-stop task (Mulvihill et al., 1997), the response reaction time to go-signals was not predicted to change under alcohol.

Method

Participants

Participants were obtained from a group of male undergraduates who volunteered to participate in psychological experiments. The sample contained twenty-eight students between 19 and 26 years of age (mean=20.7, SD=1.6). All participants were healthy social drinkers who were not taking prescription medication. They agreed not to drink any alcohol for 24 hours, and to fast for 4 hours prior to the experiment. They were paid $15.00 for their participation. Ethical approval for this research was obtained from the University of Waterloo Office of Human Research.

Seven participants were randomly assigned to one of three treatment groups (ARM, ARV and AM). Because the investigation was exploratory, the control group (A) was drawn from another alcohol study of go-stop task performance (Mulvihill, 1995) in which the experimenter, participant selection, dose administration and alcohol testing were identical to those of the experimental groups in the present investigation.

Apparatus

Go-stop task Response inhibition was measured using a go-stop choice reaction-time task that had been used in prior alcohol research (Mulvihill et al., 1997). The task was programmed on Micro Experimental Laboratory (MEL) software, and was operated by a 386 PC. The go-signals were four 1.5 cm letters (A,B,C, and D). The letters were presented individually in the centre of the

computer screen for 500 msec. A preparatory fixation point (#) was presented in the middle of the computer screen for 500 msec before each letter appeared. The fixation point and the presentation of the letter were separated by 500 msec during which time the computer screen was blank. The participant rested his index and middle fingers on two adjacent keys on the computer keyboard, and responded to a letter by pressing one of the two keys. If the A or C appeared, the left key was pressed. If the B or D appeared, the right key was pressed. Participants were instructed to press the appropriate key in response to go-signals as quickly as possible.

In addition to responding to go-signals, participants were also instructed to withhold their response whenever a stop-signal sounded during the presentation of a go-signal. The stop-signal consisted of a 900 Hz tone presented for 500 msec. The tone occurred infrequently, and followed the onset of a go-signal at one of four delays (50, 150, 250, and 350 msec after the onset of a go-signal). The delay intervals occurred with equal frequency, in an unpredictable random sequence.

Each go-signal presentation constituted one trial, and trials were separated by 2.5 s during which no stimuli were presented. One test consisted of 176 trials, presented in two blocks of 88 trials, separated by a 30 s rest period. A test required approximately 10 minutes to complete. Stop-signals occurred on 48 trials (i.e., 12 stop-signals at each delay in a test), and occurred with equal frequency for each of the four letters. Each participant performed the task alone in a room. The computer controlled the presentation of the tests, generated the stop-signal delays, the go-signals, and recorded the data.

Measures

Performance Response inhibition was measured by the number of inhibitions to the 48 stop-signals during a test. Responding was measured by the mean RT (ms) to go-signals when no tone was

presented (i.e., the time from a letter presentation until a computer key press).

Choice errors were measured by the number of incorrect key presses to go-signals during a test. Other research using this task indicated that choice errors seldom occur. Tests on the task conducted drug-free and under alcohol indicated that on average only about 5% of responses to go-signals are errors (Logan et al., 1984; Mulvihill et al., 1997). Although the number of choice errors would likely to be too small to justify statistical analysis, their incidence was recorded.

Drinking Habits Participants completed the Personal Drinking History Questionnaire (PDHQ) (Vogel-Sprott, 1992). This questionnaire (Appendix A1) provided three measures of current alcohol use: frequency (i.e., the number of drinking occasions per week), dose (i.e., ml of absolute alcohol/kg typically consumed during a single drinking occasion), and duration (i.e., the number of hours that spanned a typical drinking occasion).

Blood Alcohol Concentrations (BACs) BACs were determined from breath samples measured by Smith & Wesson model 900A and Stephenson model 900A breathalyser machines.

Procedure

When participants arrived, the experimenter ensured that the requirements for participation in the experiment were understood before informed consent was obtained. Participants were weighed and were then required to provide a breath sample to familiarize themselves with the breathalyser and to verify that they were alcohol-free. In order to familiarize participants with the task and ensure that the task instructions were understood, they practiced the task for 3 minutes while the experimenter remained in the room.

After the familiarization practice, participants in groups ARM and ARV were instructed about their positive reinforcement. The reinforcement procedure was modeled on that used in prior

research with psychomotor tasks (e.g., Sdao-Jarvie & Vogel-Sprott, 1991). Group ARM was informed that they would receive 25 cents, and be told "yes" immediately after the completion of their next 10-minute test, if they withheld the same or a greater percentage of responses as they did during their familiarization practice. Group ARV was informed that they would be told "yes" if they inhibited as well as, or better than they did during their familiarization practice. These participants had a sheet on which they could record the feedback they received after each test.

Individuals in group AM were told that they would receive a 25 cent bonus on every test on which they withheld the same or a greater percentage of responses as they did during their familiarization practice, however, the money was delayed until after the entire experiment was completed. Individuals in group A received no reinforcement or monetary incentive for their performance of the go-stop task.

The reinforcement procedures were explained to participants prior to the drug-free baseline test in order to ensure that their respective reinforcement conditions were understood before alcohol was consumed. In addition, introducing the reinforcement during the drug-free baseline test allowed participants to acquire the expectation of reinforcement for inhibitions. Because participants were drug-free and presumably performing as well as they could, there would be no reason to expect that the reinforcement would significantly affect performance on their baseline test. Finally, the administration of positive reinforcement following the baseline test also ensured that alcohol was the only new factor that could affect performance on subsequent treatment tests.

All participants then performed a 10-minute test on the go-stop task in order to provide a drug-free baseline measure of performance. Immediately after this test, participants in groups ARM and ARV received positive reinforcement. Participants then completed the PDHQ and received

0.62 g/kg alcohol. The dose was served in three drinks, mixed in a ratio of one part alcohol to two parts carbonated soft drink plus 15 ml of lemon juice for flavouring. Participants were given one minute to finish each drink. After the first drink was consumed, they rested and read magazines. The second and third drinks were served at 20 and 40 minutes, respectively. This dosing regimen was identical to that employed in prior research (Mulvihill et al., 1997) that showed an average peak BAC of 73 mg/100 ml tended to occur 70 minutes after drinking commenced.

Before performing the task under alcohol, participants in groups ARM and ARV were reminded that they would continue to receive reinforcement at the completion of each test on which they matched or increased the number of inhibitions they had achieved on their drug-free baseline test. Group AM was reminded that they would receive 25 cent bonuses at the end of the study for any tests that matched or increased their drug-free baseline score on inhibitions.

All participants performed a total of five tests that commenced at 23, 43, 63, 83 and 103 minutes after the onset of drinking. Each test was preceded by a BAC measure. Additional BAC measures were obtained at 70 minutes during the 30 s rest interval of the third test, and after all five tests were completed, 120 minutes after drinking had commenced. At the completion of the five tests, participants were fully debriefed and paid for their participation in the experiment. The temporal sequence of events is shown in Appendix A2.

Criterion for reinforcement & incentive

The administration of positive reinforcement to participants in groups ARM and ARV was determined by comparing the number of inhibitions a participant displayed on a test under alcohol to the number of inhibitions he displayed on his drug-free baseline test. Reinforcement was provided whenever the number of inhibitions a participant displayed matched or exceeded his drug-free

baseline score. A match was defined as coming within two inhibitions of the drug-free baseline score. This definition was based on prior research, which showed the number of inhibitions displayed on repeated drug-free tests on the go-stop task did not change significantly with practice, and tended to fluctuate ±2 inhibitions about a participant's mean score on tests (Mulvihill et al., 1997). The same criterion was used to determine the money bonuses paid to participants in the AM group at the conclusion of the investigation.

Because participants could deliberately increase their inhibitions simply by delaying their response to the go-signal and waiting for a tone, the task instructions warned participants that they had to maintain their RT to the go-signal during a test or their inhibitions could not be reinforced (i.e., no verbal feedback or money for that test would be administered). Prior research that tested males drug-free on five go-stop tests indicated that a participant's inhibitions remained stable, even though his RT to go-signals increased or decreased by 40 and 57 msec, respectively (Mulvihill, 1995). Because it appeared that changes in RT to go-signals in this range could occur without affecting response inhibitions, deliberate slowing of the response to go-signals was defined as a mean RT that was 50 msec, or more, slower than the participant's mean RT on his baseline test.

Experimental design & data analysis

The investigation was designed as a between-group factorial experiment. All participants performed the go-stop task under a moderate dose of alcohol. Two groups received immediate positive reinforcement (ARV & ARM) for maintaining their drug-free level of response inhibition under alcohol, whereas the other two groups (AM & A) received no such reinforcement. Groups AM and ARM received a monetary incentive, and groups ARV and A received no money.

The total number of inhibitions, mean RT and total number of choice errors were measured

for each participant on every test. Treatment effects were assessed by analyzing inhibitions and RT separately. The degree to which alcohol changed inhibitions from a participant's drug-free baseline was of primary interest. The change was calculated separately for inhibitions and RT, by subtracting a participant's drug-free baseline test score from each of his test scores under treatment. These change scores were used in 2(reinforcement) X 2(money) X 5(test) analyses of variance (ANOVAs). Treatment effects were also confirmed by a covariance analysis (ANCOVA) of the actual test scores, using the drug-free baseline scores as a covariate. The average number of inhibitions, and the mean RT on all five tests under alcohol were analyzed separately in 2(reinforcement) X 2(money) ANCOVAs. The ANCOVAs provide statistically adjusted scores and are shown in appendices. The ANOVA using change scores are presented in this thesis because they show the actual degree of change from drug-free baseline performance.

RT Data Measures of response-times are often analyzed for outliers (i.e., RTs that are thought to represent "noise"). The RTs in this data set were trimmed for outliers using a non-recursive procedure with moving criterion described by Van Selst & Jolicoeur (1994). The procedure followed to trim outliers in the RT data is described in Appendix A3 together with the analyses of the trimmed and untrimmed RT. The conclusions using the trimmed RT data set were essentially identical to those found using the entire RT data set. As a precaution against outliers and to reduce noise associated with outliers, the trimming procedure for RTs will be adopted in this thesis.

The RT measure could be based on correct responses to go-signals, or on all go responses (both correct and incorrect key presses) to go-signals. Very few incorrect responses occur, and both ways of measuring RT were analyzed, and resulted in identical conclusions (Appendix A4). In the interest of measuring RT to go-signals with the least amount of noise from error responses,

the analysis of correct responses to go-signals that exclude incorrect key presses is the measure of RT analyzed in this thesis. Thus, the measure of RT reported and analyzed in this thesis is the trimmed response-time when participants responded correctly to go-signals.

Results

In order to check that the groups did not differ at the outset of the study, group differences on several variables were examined before analyzing response inhibition.

Because an individual's drinking habits might influence behaviour under alcohol, the four groups were compared using one-way analyses of variance (ANOVAs) of the three drinking habit measures obtained from the PDHQ. Two participants did not provide information on the duration of their drinking occasions, and this resulted in the loss of their data for this measure. There were no significant group differences in any of the drinking habit measures (ps>.393) (Appendix A5). The entire sample reported a mean (SD) drinking frequency of 1.3 (1.2) times per week, with a mean (SD) dose per occasion of 1.4 ml/kg (0.72). For a man weighing 75 kg, this dose would approximate 6 beers containing 5% alcohol/volume. The mean (SD) duration of drinking was 4.4 (1.3) hours.

The drug-free baseline number of inhibitions of the groups was compared by a 2(reinforcement) X 2(money) ANOVA (Appendix A6, Table 1a). This analysis revealed no significant main effect of money or reinforcement and no interaction (ps>.256). The mean number of inhibitions to the 48 stop-signals during the baseline test was 27.8 (SD=10.9). This represented inhibitions to 58% of the stop-signals during a test. The mean (SD) number of inhibitions for each group is presented in Appendix A6, Table 1b.

Typical drug-free performance on the go-stop task shows that individuals display

progressively fewer inhibitions as stop-signal delays increase. Participants in this investigation also displayed this pattern of performance. At the shortest delay (50 msec from the onset of the go-signal), a mean of 10.1 (SD=2.1) inhibitions was displayed. As the delay was increased from 150 to 250 msec, mean inhibitions dropped progressively from 8.8 (SD=2.8) to 5.8 (SD=3.8). At the longest (350 msec) delay, a mean of only 3.1 (SD=3.4) was displayed. Twelve stop-signals were presented at each stop-signal delay so the percentage of successful inhibitions at each stop-signal delay were 84, 73, 48 and 26, respectively.

A 2(reinforcement) X 2(money) analysis of the mean RT during the baseline test revealed no significant main effects or interaction (ps>.089) (Appendix A6, Table 2a). The mean RT during the baseline test was 493.98 msec (SD=112.11). See Appendix A6, Table 2b for the mean (SD) RTs of each group.

The mean number of errors during the baseline test was 7 (SD=4.5), and represents an error rate of 5.5%. This low error rate is comparable to that observed in previous research (Mulvihill et al., 1997; Logan et al., 1984).

Treatment Effects

The measures of a participant's BAC obtained at seven intervals during the alcohol session were tested by a 4(groups) X 7(time) ANOVA (Appendix A7, Table 1a; see Table 1b for the mean (SD) BACs of each group)[2]. The ANOVA only revealed a significant main effect of time, $F(1,6)=114.12$, $p<0.01$. Table 1 presents the seven mean BACs (SD) of the four groups. The table shows the main effect of time reflected the rise and decline in BACs during the session.

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[2] Due to malfunction of the Smith & Wesson breathalyser machine, the data for three participants were lost.

The table indicates that the mean peak BAC of 79 mg/100 ml occurred 80 minutes after drinking commenced, during the fourth test under alcohol.

Table 1: Mean BACs (SD) of four groups at seven time intervals in relation to the five tests on the task.

Test	1	2	3		4	5	
Minutes After Drinking	20	40	60	70	80	100	120
Mean BAC (mg/100 ml)	21	40	62	76	79	71	63
(SD)	9	13	16	13	19	13	10

Inhibitions The change in inhibitions under alcohol was tested by a 2(reinforcement) X 2(money) X 5(test) ANOVA (Table 2). The analysis revealed a significant main effect of reinforcement, $F(1,24)=6.02$, $p=.022$, and a significant test X reinforcement interaction, $F(4,96)=4.34$, $p=.003$. No other effects were significant.

The mean change in inhibitions, averaged across tests for each group, is shown in Figure 1A. Zero on the vertical axis represents participants' pre-treatment (i.e., drug-free) baseline inhibition score. A negative score indicates a reduction in inhibitions under alcohol, whereas a positive score indicates an increase in inhibitions. The figure indicates that the reinforced groups, ARV and ARM, show little change in response inhibition compared to groups AM and A that received no reinforcement, both of which showed decreases in response inhibition. The mean (SD) change in

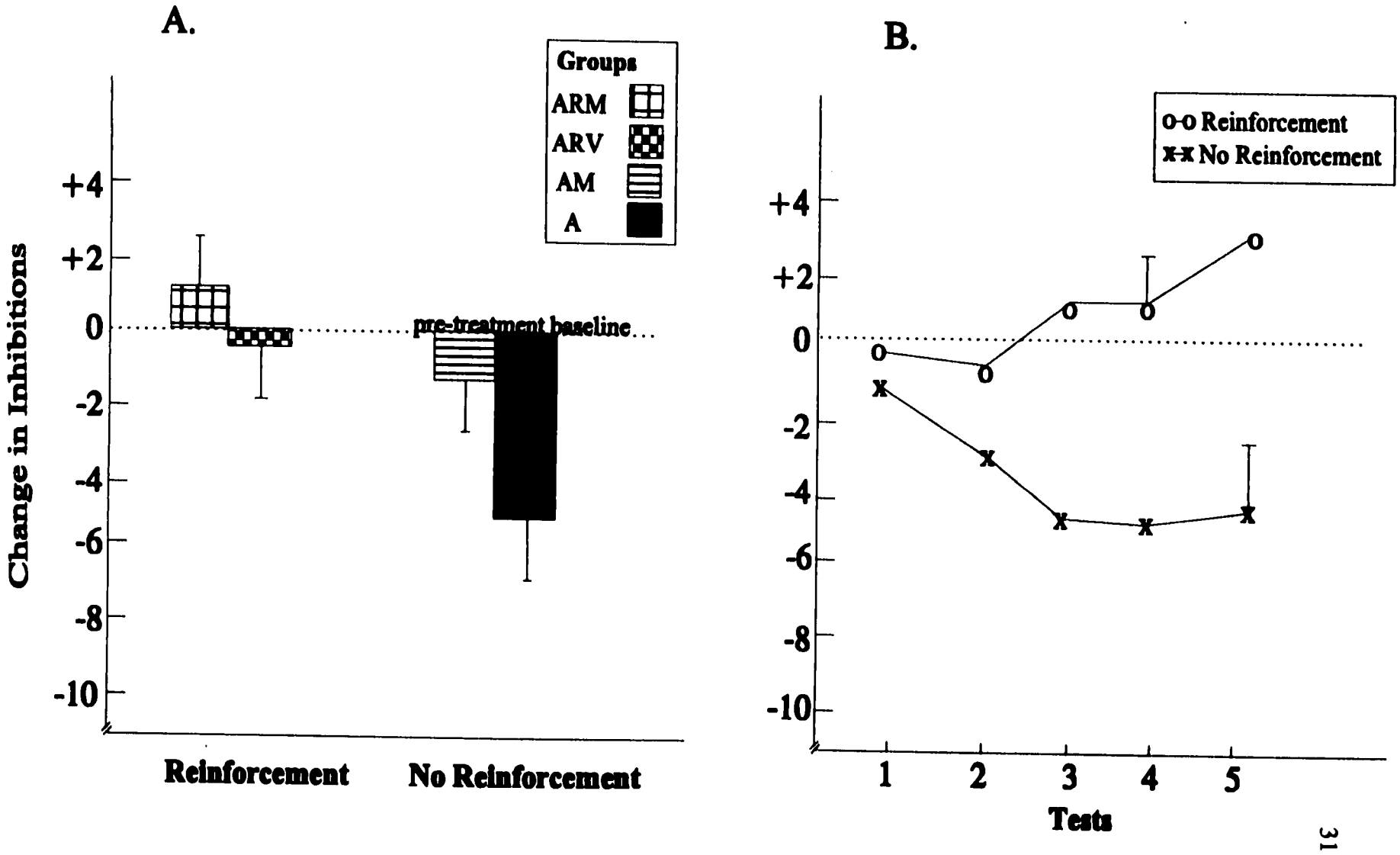
Table 2: ANOVA of the change in inhibitions as a function of reinforcement and money treatments on five tests under alcohol.

Source	df	MS	F	p
Reinforcement (R)	1	528.46	6.02	.022
Money(M)	1	263.31	3.00	.096
R X M	1	35.00	0.40	.534
Error	24	87.81		
Tests (T)	4	8.05	0.71	.589
T X R	4	49.44	4.34	.003
T X M	4	25.76	2.26	.068
TxMxR	4	1.98	0.17	.951
Error	96	11.39		

inhibitions of the ARM and ARV groups was +0.39 (3.9) whereas the mean (SD) change of the AM and A groups was -3.5 (4.7). Although the ARM group showed a slight increase in inhibitions under alcohol and the ARV group showed a slight decline, the group means did not differ significantly, $F(1,24)=0.61$, $p=.444$. Moreover, paired t-tests showed that the means of groups ARM and ARV did not differ from their baseline zero score ($ps>.436$). Thus, as predicted, reinforcement reduced the impairing effect of alcohol on response inhibition and the reinforcement types (i.e., money and verbal feedback) were equally effective in counteracting alcohol's effect.

The test X reinforcement interaction is illustrated in Figure 1B. The figure shows that the reinforced groups (ARM and ARV) tended to increase their number of inhibitions as tests were repeated, whereas those with no reinforcement showed some decline in response inhibition across tests.

Figure 1: Mean change in inhibitions of four treatment groups under alcohol. **A:** Overall mean change across five treatment tests. Vertical bars show SEMs. **B:** Mean change on each treatment test for groups that received reinforcement, or no reinforcement. Vertical bars show SEMs.



The conclusions from the ANOVA were compared with those from a 2(reinforcement) X 2(money) ANCOVA of the average number of inhibitions on all five tests under alcohol. The pre-treatment (i.e., drug-free) baseline inhibitions were used as a covariate, and did not interact with any between-factors. Thus the assumption of homogeneity of slopes was not violated and an ANCOVA was valid (Appendix A8, Table 1a). The ANCOVA and adjusted mean number of inhibitions are shown in Appendix A8, Tables 2a and 2b, respectively. The ANCOVA also revealed a main effect of reinforcement, $F(1,23)=7.05$, $p=.014$, which indicated that the adjusted mean number of inhibitions of the reinforcement groups (ARM & ARV) were greater (i.e., less impairment) than the no-reinforcement groups (AM & A).

However, the ANCOVA also detected a significant main effect of money, $F(1,23)=5.10$, $p=.034$, that had only reached $p=.096$ in the ANOVA. Two post hoc comparisons were conducted to investigate the main effect of money obtained in the ANCOVA using Tukey's Honestly Significant Difference post hoc test (Howell, 1992). A comparison of the inhibitions displayed by the group reinforced with money (ARM) with group AM was not significant ($p=.579$), and an additional comparison between the group reinforced with verbal feedback (ARV) and group AM was also non-significant ($p=.956$). Thus, it appeared that simply providing a delayed monetary incentive (AM) was as effective as immediate reinforcement in improving resistance to the effect of alcohol on response inhibition. In addition, a paired t-test revealed that the change in inhibitions in group AM did not differ from the baseline zero score, $t(6)=-1.07$, $p=.328$. Although no effect of a monetary incentive had been predicted, it appears that the effect of the AM treatment on response inhibition under alcohol merits further consideration and examination.

Response RT The results of a 2(reinforcement) X 2(money) X 5(test) ANOVA of change in RT

to go-signals (Table 3) revealed no effects significant at the conventional .05 level. However, the main effect of money approached this level, $F(1,24)=4.12$, $p=.054$. Inspection of the mean RTs of the two groups whose treatment involved money (groups ARM and AM) indicated that their RT became somewhat faster during treatment (mean change=-35.15 msec, SD=28.63) whereas the RT of groups ARV and A showed little change (mean change=-1.61 msec, SD=53.93).

Table 3: ANOVA of the change in RT for reinforcement and no-reinforcement treatments on five tests under alcohol.

Source	df	MS	F	p
Reinforcement (R)	1	9313.66	0.97	.334
Money(M)	1	39363.55	4.12	.054
R X M	1	3513.91	0.37	.550
Error	24	9562.04		
Tests (T)	4	431.71	0.47	.761
T X R	4	505.35	0.55	.703
T X M	4	937.71	1.01	.405
TxMxR	4	306.19	0.33	.857
Error	96	926.63		

There was a concern that participants could delay their response to go-signals in order to increase their number of inhibitions. Thus, the group means were examined to determine whether this might have occurred (Table 4). Table 4 shows that the alcohol group was the only group to slow down their RT to go-signals (mean change = +11.56 msec, SD=71.66) and the other groups tended to respond faster to go-signals during treatment. Although the alcohol group showed a slight increase in RT during treatment, this group also displayed the greatest *decrease* in inhibitions

(see Figure 1). Thus, the mean changes in RT suggested that no group deliberately slowed down in an attempt to increase the number of inhibitions they obtained.

Table 4: Mean change in RT (msec) over all tests by 4 groups.

		Reinforcement (R)	
		Yes	No
Money	Yes	ARM: -38.29 (32.74)	AM: -32.0 (26.10)
	No	ARV: -14.78 (27.59)	A: +11.56 (71.66)

The results from the ANOVA were compared with a 2(reinforcement) X 2(money) ANCOVA of the mean RT on all five tests under alcohol (Appendix A9, Table 1 & 2a). The results from the ANCOVA were consistent with those of the ANOVA (ps>.150) and the adjusted group mean RTs (Appendix A9, Table 2b) also showed that group A had the slowest RT to go-signals.

Choice Errors The mean change in errors indicated that there was little overall change under alcohol. The incidence of errors under alcohol ranged from 2.5% to 8% in the groups. Overall, incorrect key choices tended to occur only 5.5% of the time under alcohol.

Discussion

The preliminary investigation developed an experimental procedure to reinforce inhibitions on a go-stop task, and explored the possibility that immediate positive reinforcement for a sober level of response inhibition on a go-stop task would attenuate the effect of a moderate dose of alcohol on inhibitory control. Some support for the hypothesis was found as there was little decline

in inhibitions under alcohol when immediate positive reinforcement was administered compared to groups with no reinforcement. Reinforcement in the form of money or verbal feedback appeared equally effective in counteracting the reduction in inhibitions under alcohol. These effects could be attributed to reinforcement because differences in RT to go-signals could not account for the results. Consistent with previous research (e.g., Mulvihill et al., 1997), errors remained at a stable low level regardless of the treatments.

Although reinforcement appeared to counteract the impairing effect of alcohol on inhibitions, this conclusion was clouded by the results of the delayed monetary incentive in group AM. The ANCOVA analysis detected a main effect of money (p=.034) that only reached p=.096 in the ANOVA, and post hoc comparisons of adjusted group means indicated that the effect of alcohol on inhibitions in the AM group did not differ from that observed under reinforcement (groups ARV and ARM). However, given the equivocal results of the two statistical analyses, it is difficult to know how much credence to place on these results.

If monetary incentive counteracts the effect of alcohol on response inhibition, this result would be at odds with other research that has found that a monetary incentive does not reduce the effect of alcohol on psychomotor and information-processing tasks (e.g., Fillmore & Vogel-Sprott, 1997; Sdao-Jarvie & Vogel-Sprott, 1991). The inconsistency between the observations of this investigation and those of other studies might suggest that a monetary incentive uniquely affects response inhibition under alcohol. However, it is important to consider other possible explanations for the seemingly unusual effect of the monetary incentive on inhibitions under alcohol.

The measures of RT to go-signals indicated that the AM group did not slow their response in order to increase inhibitions. However, it is also possible to increase inhibitions to stop-signals

by choosing not to respond to go-signals. This possibility was examined by counting the number of times a response to go-signals failed to occur during every test. There was no indication of this strategy in group AM. In fact, the occurrence of "no responses" to go-signals was extremely rare (about 0.2%) during baseline and alcohol tests.

To check the possibility that differences in group BACs might account for the unusual result in group AM, the overall group mean BACs of the groups were examined. The group mean BACs varied slightly (see Appendix A7). However, the difference in BACs did not account for the reduced effect of alcohol on response inhibition in group AM because this group had somewhat higher average BACs. Thus, it seemed unlikely that the BACs or the behavioural strategy of participants in group AM could contribute to the reduction in the effect of alcohol on their response inhibition.

Alternative explanation

There was one procedural difference between the present investigation and the other alcohol research on motor skills and information-processing tasks. The researchers in those studies administered a drug-free training session to allow the task to be learned, and conducted a subsequent session on another day to test the effect of alcohol. Under this procedure, the effect of alcohol was tested *after* participants had become familiar with the task and testing procedures in the laboratory. By contrast, the present work tested the effect of alcohol on the first session. A preceding drug-free training session was deleted because prior research indicated that an individual's performance on the go-stop task does not change significantly with repeated drug-free training (Mulvihill et al., 1997). However, testing the effect of alcohol in one single session in the laboratory measured drug effects in a setting that was new (i.e.,"novel") to the individual. It might

be that the degree of impairment under alcohol was affected by the novelty versus familiarity aspect of the setting. Yet if this hypothesis is correct, it has broad implications for the interpretation of the findings on response inhibition in this investigation.

The possibility that the intensity of the effect of alcohol on response inhibition is affected by such a novel versus a familiar setting factor raises a question about results from the A group in the preliminary investigation. The effect of alcohol on participants in group A had been tested under treatment conditions that were identical to those of all the other groups in this investigation. However, these participants had been drawn from a prior experiment that tested the effect of alcohol *after* individuals had attended a drug-free session at which they were familiarized with laboratory task and testing procedures. In retrospect, the effect of alcohol on response inhibition in group A could be considered to be derived in a familiar setting. If the effect of alcohol is weakened in a novel setting, then the result in group A might have overestimated the impairing effect of alcohol on response inhibition.

The possibility that a weaker effect of alcohol is observed in a novel setting was explored by testing a few additional volunteers under A treatment in one alcohol session (i.e., the setting was novel). Under these conditions little reduction in response inhibition was observed. Moreover, little change in response inhibitions was evident despite the fact that the alcohol dose was consumed more quickly (two drinks instead of three). The faster drinking regimen tends to cause a faster rise in BAC and could be expected to cause greater, not less, impairment (Fillmore & Vogel-Sprott, 1998; Jones & Vega, 1973; Moskowitz & Burns, 1976).

If the impairing effect of alcohol on response inhibition is more intense in a familiar setting, then the apparent resistance to impairment displayed by groups ARM, ARV as well as AM might

be due to a reduced drug-effect owing to the novel setting. This means that the reduced effect of alcohol on response inhibition might be due to reinforcement, or to the novel setting. To clarify the findings, the main experiment was designed to test the effect of reinforcement and a monetary incentive on response inhibition under a moderate dose of alcohol administered when the setting is novel or familiar.

Some aspects of the experimental procedure used in this experiment could be improved by a few modifications. A two-drink dosing schedule appears to have some advantages over the three-drink procedure used in the investigation because it allows the entire dose of alcohol to be consumed before tests on the go-stop task begin. In addition to convenience, the two-drink schedule results in a faster rise in BAC, which could cause stronger effects of the dose and a better test of alcohol effects on response inhibition.

The results suggested that the five-test procedure could be shortened to four without changing the conclusions obtained with five tests. Performance on four tests instead of five might also reduce any effects of fatigue.

After careful consideration of the questions to be addressed in the main study, it was decided that the main study would test the effects of only immediate verbal reinforcement under a moderate dose of alcohol. Verbal reinforcement will be used because it might be a more realistic model of the reinforcement that individuals would receive in a real-life context (i.e., it is more likely that an individual might receive verbal feedback about his behaviour when drinking alcohol than a monetary reward).

Inspection of response inhibitions at each stop-signal delay on the drug-free baseline test showed that few inhibitions were displayed on the longest (350 msec) stop-signal delay. In fact,

30% of the participants displayed no inhibitions at all at that delay. As a result, any further reduction in inhibitions under alcohol might not be detected with a 350 msec stop-signal delay. Thus, the 350 msec stop-signal delay may impose a floor effect on inhibitions and including data from this stop-signal delay could obscure and weaken the evidence of treatment effects. However, deleting the 350 msec delay altogether is inadvisable because four stop-signal delays are used in go-stop tasks to ensure that a participant cannot anticipate when a stop-signal will occur. Thus the main experiment will maintain the 350 msec stop-signal delay in the task, and response inhibition will be analyzed with and without the data from the 350 msec stop-signal delay to determine whether stronger treatment effects are obtained when the 350 msec delay data are excluded from analyses.

Summary & Conclusions

The investigation provided some evidence that seemed consistent with the hypothesis that immediate positive reinforcement of inhibitions increased resistance to the impairing effect of alcohol on response inhibition. However, the interpretation of the results was clouded by observations suggesting that a monetary incentive resulted in similar resistance to the effect of alcohol. Additional consideration of the findings raised the possibility that prior drug-free familiarity with a setting in which alcohol is administered may result in more impairment of inhibitory control, compared to alcohol administered in a novel setting. Thus, the main experiment of the dissertation is designed to test:

1) the effect of alcohol on response inhibition when the setting is novel or familiar, and

2) the effect of positive reinforcement, a delayed monetary incentive or no reinforcement on response inhibition under alcohol in a novel or a familiar setting.

Three changes to the experimental procedure will be adopted in the main study. First, the alcohol dose will be administered in two drinks instead of three. Second, drug effects on the go-stop task will be tested on four tests, and, third, the effect of reinforcement will be tested using approving verbal feedback as positive reinforcement.

MAIN STUDY

The results of the preliminary investigation suggested that the provision of immediate positive reinforcement for maintaining a sober level of inhibitory control under alcohol could reduce the disinhibiting effect of the drug. However, these results were obtained when alcohol was administered the first time that participants were in the laboratory (i.e., the setting was novel). Additional observations indicated that administration of alcohol in a novel setting might result in a less intense drug effect, compared to alcohol administration in a familiar setting. Thus, the main study was designed to test: 1) the effect of alcohol on response inhibition when the setting is novel or familiar, and 2) the effect of positively reinforcing inhibitions under alcohol in each setting.

Drug effects and the novelty or familiarity of the setting

A literature search indicated that the behavioural effect of a drug had rarely been examined in relation to familiarity with the setting in which a drug is administered. However, this possibility received some research attention in the 1960's. The prime goal of these studies was to call attention to the possibility that the pharmacological action of the drug alone cannot explain the behavioural response to a drug. Thus this work was designed to show that non-drug factors, such as prior drug-free experience in the testing setting, could in fact alter the behavioural response to a drug. Although none of these studies used alcohol, the effects of several other drugs were investigated.

Much of this research has examined the behavioural effect of administering a combination of a stimulant and a sedative (an amphetamine and a barbiturate). Research that measured locomotor activity in rats under a dose of these drugs (Steinberg, Rushton & Tinson, 1961) demonstrated that the intensity of the drug effect depended on prior drug-free exposure to the experimental setting. In these studies one group of rats had several drug-free exposures to the experimental setting

before receiving a drug injection (experienced), whereas the other group had never been exposed to the experimental setting before the drug was administered (inexperienced). The results revealed that the inexperienced group showed twice as much locomotor exploration under the drug than did experienced drugged rats. In fact, the drug had little effect in the experienced group because their behaviour was not different from the experienced saline control group. Rushton, Steinberg & Tinson (1963) subsequently showed that even after one short drug-free exposure to the experimental setting, experienced rats showed no significant effect of the drug whereas inexperienced rats showed substantial drug effects. In a study using a similar design, Marriot & Spencer (1965) tested the effects of prior drug-free experience in rats under either chlordiazepoxide or the barbiturate-amphetamine mixture. They also concluded that the intensity of the behavioural effect of these drugs differed in groups of rats with or without prior drug-free familiarity with the setting.

One might have expected that the results of these studies would have prompted investigators to test possible explanations for the effects of prior drug-free exposure to the setting on drugged performance. However, research by psychologists at that time was focussed on demonstrating that the behavioural effect of a drug cannot be explained solely by its pharmacological action, and that environmental factors also play a role. Thus, instead of testing possible explanations for the effect of prior drug-free exposure to a setting, researchers chose to pursue the investigation of the impact of another type of drug-free experience on drugged performance.

Investigators began to examine the effect of different drug-free reinforcement schedules on subsequent performance of squirrel monkeys and pigeons under a drug (e.g., Barrett, 1977; Barrett & Stanley, 1983; Terrace, 1963). Groups in these experiments were trained drug-free under

different reinforcement schedules. They then received drug-free training under a common reinforcement condition, during which no difference in their performance was observed. However, when they received a drug (either morphine, amphetamine, chlorpromazine or imipramine), the behavioural effect of the drug differed, and depended upon the prior drug-free reinforcement schedule under which the animals were initially trained. For example, a dose of morphine decreased or increased responding, depending on the subjects' prior drug-free reinforcement history (Barrett & Stanley, 1983). Reviews of research indicate that a more intense effect, no effect, or a reversal of "usual" drug effects are seen depending on an animal's prior drug-free experience in the experimental setting (Barrett, Glowa & Nader, 1989; Barrett & Witkin, 1986).

A literature search found no studies that tested the effect of prior drug-free experience on the *behavioural* effects of drugs in humans. However, one study was found that called attention to the possibility that drug-free familiarity with a setting might affect the intensity of *subjective and physiological* effects of alcohol (Newlin & Pretorius, 1991). This research measured self-reported intoxication, and physiological reactions (heart rate, finger and cheek temperature). In this study, one group of social drinkers had drug-free appointments in the laboratory before attending a session to receive alcohol (familiar group). The other group attended one session in the lab and received alcohol during that first visit (novel group). After drug-free baseline measures were obtained, all participants received a 0.60 g/kg dose of alcohol, and the measures were repeated. There were no group differences in the drug-free baseline measures. However, after alcohol consumption there was a greater increase in heart rate, cheek temperature and self-reported drunkenness in the familiar group compared to those in the novel group. These findings led the investigators to conclude that "the alcohol effect is weakened in studies in which alcohol is administered in the first exposure to

the laboratory...[and that the] effects that have been found in a novel environment might be more robust...[after prior drug-free] exposure to that environment" (Newlin & Pretorius, 1991, p. 473). Although the experiment was not designed to test any explanation for the findings, the evidence suggested that social drinkers' prior drug-free familiarity with a setting intensified the effect of alcohol on some physiological responses and a self-report measure of drunkenness. Whether drug-free familiarity with a setting similarly affects the intensity of the effect of alcohol on behaviour is not known. Thus, the first phase of the present research examined the effect of alcohol on response inhibition when the setting was novel or familiar.

Positive reinforcement and the setting

The second phase of the study tested the effects of reinforcing inhibitions (with approving verbal feedback) under alcohol administered in a novel or a familiar setting. Considerable research investigating the impairing effect of alcohol on psychomotor skills and information processing has shown that the intensity of alcohol impairment is reduced when drinkers receive immediate positive reinforcement for maintaining their sober (i.e., non-impaired) level of performance (e.g., Vogel-Sprott, 1992). The close temporal contingency between the reinforcer and non-impaired performance appears to be the crucial factor, because simply promising an incentive for resisting impairment does not reduce the behavioural effect of alcohol (e.g., Fillmore & Vogel-Sprott, 1997; Sdao-Jarvie & Vogel-Sprott, 1991). Thus, it seems possible that immediate reinforcement may also reduce the impairing effect of alcohol on inhibitory control, whereas a monetary incentive may have no effect on alcohol-induced impairment of response inhibition. It is also possible that the effect of alcohol in a novel setting might be too weak to impair inhibitory control, and so the effect of reinforcement for resistance to impairment would not be evident. To examine these possibilities,

three different treatments (reinforcement, monetary incentive or no consequences) were administered to groups who performed the go-stop task in either a novel or a familiar setting under a moderate dose of alcohol.

Hypotheses

A between-subjects design was adopted to test the experimental hypotheses. In order to test the effect of alcohol in a novel or a familiar setting, four groups performed the go-stop task with no environmental consequences associated with performance. In Phase 1 of the experiment one pair of those groups performed under alcohol (A) in a "novel" setting (N), or a "familiar" setting (F). This pair of groups were identified as N-A and F-A, respectively. The other pair of groups received a placebo (P) in the novel or familiar setting. These groups, designated N-P and F-P, respectively, were included to control for any effect of expecting alcohol (see Figure 2A for the design of Phase 1 of the experiment). Alcohol administration in a novel setting was predicted to reduce the impairing effect of alcohol, compared to alcohol administration in a familiar setting. Thus, the reduction in inhibitions should be less in a novel setting (group N-A) than in a familiar setting (group F-A). If the novelty-familiarity aspect of the setting only influences the effect of the drug, then inhibitions displayed under the placebo in the novel or familiar setting (groups N-P and F-P) should not differ. In addition, the F-A group should display a greater reduction in inhibitions (i.e., more impairment) than both placebo groups (F-P and N-P).

In Phase 2 of the experiment a second set of hypotheses was tested that concerned the effect of reinforcing (R) a sober level of inhibitory control under alcohol, in a familiar or a novel setting. This investigation involved three pairs of groups that were tested under alcohol (A) in the N or F setting. One pair of groups received positive reinforcement under alcohol whenever they maintained

a sober level of response inhibition under alcohol (groups F-AR and N-AR).

A second pair of groups received a monetary incentive (M) for maintaining their sober level of response inhibition under alcohol (A), but the money was not paid until the experiment was completed. This pair of groups is identified as N-AM and F-AM.

The third pair of groups performed the task under alcohol (A) with no reinforcement or monetary incentive (this group was described above, in Phase 1 of the experiment). These groups served as controls, providing a measure of the actual effect of alcohol on inhibitions in the novel (N-A) or a familiar (F-A) setting. See Figure 2B for the design of Phase 2 of the experiment.

The effect of reinforcement was predicted to depend on the setting. When alcohol is administered in the familiar setting, reinforcement should weaken the impairing effect of alcohol. Therefore, the group that receives reinforcement for a sober level of inhibitory control should show less impairment than groups without reinforcement (F-AM and F-A). Furthermore, if the temporal contingency between reinforcement and subsequent performance is the important factor that allows drinkers to resist the effect of alcohol on inhibitory control, then the inhibitions displayed by groups F-AM and F-A should not differ. If the drug effect is too weak to reduce response inhibitions in the novel setting, no treatment effects may be evident. In this case, the inhibitions displayed by the three treatment groups in the novel setting (N-AR, N-AM, and N-A) should not differ.

On the basis of accumulating findings on the effect of a moderate dose of alcohol on the performance of the go-stop task, the reaction time to go responses was not predicted to show any systematic change under the treatments administered to the groups.

Figure 2: Experimental Designs

A. Experimental design for Phase 1.

		Treatment Groups	
		Alcohol	Placebo
Setting	Novel	N-A	N-P
	Familiar	F-A	F-P

B. Experimental design for Phase 2.

		Treatment Groups		
		Immediate Reinforcement	Monetary Incentive	Alcohol
Setting	Novel	N-AR	N-AM	N-A
	Familiar	F-AR	F-AM	F-A

Method

Participants

Seventy-two male social drinkers took part in the experiment. They were obtained from a group of undergraduates who volunteered to participate in psychological experiments (Appendix B1). All participants were healthy social drinkers who were not taking prescription medication or over-the-counter drugs. They agreed not to drink alcohol for 24 hours and to fast for 4 hours prior to the alcohol session. They received an honourarium of $15.00 for their participation. Ethical approval for this research project was obtained from the University of Waterloo Office of Human Research.

Apparatus & Measures

Task The experiment used the go-stop choice reaction-time task described in the preliminary study. The program, using Micro Experimental Laboratory (MEL) software, was operated by a 486 PC. A test on the task required 10 minutes to complete and was performed by a participant alone in the laboratory. On each test, the computer recorded the number of inhibitions at each stop-signal delay, as well as the total number of inhibitions, and mean reaction time to go-signals.

The evidence obtained in the preliminary investigation indicated that choice errors were unlikely to yield any meaningful results because they occurred very infrequently drug-free and under alcohol. However, choice errors continued to be recorded to monitor their occurrence.

Drinking Habits Participants completed the Personal Drinking History Questionnaire (PDHQ) (Vogel-Sprott, 1992). This questionnaire (Appendix C1) provided three measures of current alcohol use: frequency (i.e., the number of drinking occasions per week), dose (i.e., ml of absolute alcohol/kg typically consumed during a single drinking occasion), and duration (i.e., the number of

hours that spanned a typical drinking occasion). Participants also reported the number of years, to the nearest month, that they had been drinking alcohol on a regular basis. Two additional questions asked participants if they had ever been charged with impaired driving, and if they had any alcohol-related problems. These two questions were included in order to identify and exclude any participant with any problems related to the use of alcohol. However, no participants reported any alcohol-related problems.

Beverage Rating Scale In order to check the credibility of the placebo, participants rated the strength of the dose of alcohol in the drinks they received by comparing the dose to bottles of beer containing 5% alcohol, or to ounces of liquor containing 40% alcohol (Appendix C2). The scale for bottles of beer ranged from zero to ten in 0.5 increments. The scale for ounces of liquor was identical to that used for beer except that the increments referred to ounces of liquor instead of bottles of beer. For scoring purposes, all ratings were converted to the beer equivalent.

Desire to resist drug effect Participants rated the degree to which they attempted to resist the effect of the drug on a scale that ranged from zero ("Not at all") to 10 ("Extremely"). These data were collected for exploratory purposes (Appendix C3).

Blood Alcohol Concentrations (BACs) BACs were determined from breath samples measured by a Stephenson model 900A breathalyser machine.

Procedure

Potential volunteers were contacted by the experimenter in a phone call that provided a general description of the experiment and its requirements. Individuals who agreed to participate were randomly assigned to one of eight groups (n=9). If an individual had been assigned to one of the four groups whose response inhibition would be tested in a familiar setting, he was informed

that the study consisted of an initial drug-free appointment, followed by the alcohol session on another day (Appendix B2). If the participant had been assigned to one of the four groups to be tested in a novel setting, he was informed that the experiment involved one alcohol session (Appendix B3).

At the beginning of the study, the experimenter ensured that the requirements for participation in the experiment were understood (Appendix B4 and B5), and informed consent was subsequently obtained from participants in the F (Appendix C4) and N settings (Appendix C5).

The individuals in the F groups had a drug-free appointment in the laboratory prior to attending an alcohol session. During this session the go-stop task was explained and participants performed the task for 3 minutes to become familiar with the task (Appendix B6). The experimenter remained in the room during this time to ensure that the task instructions had been understood. To introduce them to the testing procedure, they also performed the go-stop task for 10 minutes alone in the laboratory (Appendix B7). Thus, this session provided participants with a drug-free experience that acquainted them with the laboratory, the go-stop task, testing procedure and the experimenter. When this session concluded, participants were reminded about the requirements for the next session under alcohol (Appendix B8).

Individuals in the N groups attended the laboratory once. Before that session started, they received the task instructions and performed the task for 3 minutes in the presence of the experimenter to ensure that they understood the task instructions (Appendix B6).

The participants in the familiar setting returned to the laboratory for their second session while those in the novel setting continued to the next stage in the experiment. Before the experiment continued, however, the experimenter checked that participants had complied with the fasting

requirements. They were then weighed and they provided a breath sample to verify that their BACs were zero (Appendix B9). Participants in the novel and familiar settings were then reminded of go-stop task instructions (Appendix B10 and B11) and were then introduced to the treatment condition for their group.

Immediate Positive Reinforcement Participants were introduced to positive reinforcement by instructions that explained they would be told "yes" immediately after this test, if they inhibited their response to stop-signals as well as or better than they had done on their previous drug-free test (Appendix B12).

Monetary Incentive Participants were told that they would receive a monetary bonus (25 cents) if they maintained or improved their level of response inhibition, and that the bonus money earned would be paid at the end of the experiment (Appendix B12).

Alcohol These participants received a basic reminder of task instructions before their baseline test (Appendix B12). Because the purpose of this treatment was to test the effect of alcohol only on response inhibition (i.e., to serve as a control for the effects of positive reinforcement or monetary incentive), participants performed the pre-treatment test on the go-stop task with no consequences whatsoever.

Placebo These participants received a reminder of task instructions (Appendix B12) that was identical to the instructions received by alcohol only groups (i.e., F-A and N-A). Because the placebo treatment was included to control for any effect of expecting alcohol, participants performed the pre-treatment test on the task with no positive reinforcement or monetary incentive.

Testing Schedule All groups then performed a 10-minute pre-treatment drug-free baseline test on the go-stop task. After the baseline test on the task, participants in groups F-AR and N-AR received

positive reinforcement. All task participants completed the PDHQ. Participants then received alcohol or placebo beverages.

Those participants assigned to alcohol groups received a 0.62 g/kg dose of alcohol. The dose was served in two drinks of equal volume. Each drink was mixed in a ratio of one part alcohol to two parts carbonated soft drink.

Those participants assigned to the placebo groups received a beverage that consisted of a liquid volume of carbonated soft drink equivalent to that administered in the 0.62 g/kg dose alcohol drinks, divided into two equal drinks. Just before serving each drink, the experimenter floated 5 ml of alcohol on the surface of the beverage and around the rim and sides of the glass, and sprayed the glass with a 50-50% water-alcohol mixture. This appeared as condensation, and produced an alcohol scent that added credibility to the placebo beverage. The negligible amount of alcohol floated on top of the drink produces no detectable blood alcohol level, and this placebo has been used successfully in previous research (e.g., Fillmore & Vogel-Sprott, 1995; Mulvihill et al., 1997; Zack & Vogel-Sprott, 1993).

Each beverage (alcohol and placebo) was consumed within one minute. The second drink was served five minutes after the first drink was finished. After both drinks were consumed, participants rested and read magazines for 15 minutes.

Participants were then reminded of the task conditions appropriate to their group assignment (Appendix B13). Participants in the positive reinforcement groups were reminded that they would continue to receive reinforcement at the completion of each test on which they matched or increased the number of inhibitions they achieved on the pre-treatment baseline test. The monetary incentive groups were reminded that they would receive 25 cent bonuses at the end of the study for

any tests that matched or increased their pre-treatment baseline score on inhibitions. The alcohol and placebo treatment groups received a reminder of the go-stop task instructions. Participants then performed four tests that started at 30, 60, 90, and 110 minutes after drinking had commenced. Each test was preceded by a BAC measure. Additional BAC measures were obtained at 72 minutes at the completion of the second test, and after all four tests were completed, 120 minutes after drinking commenced. The BACs of participants assigned to the placebo groups were also tested, ostensibly to measure their blood alcohol levels. The entire temporal schedule of events during the treatment session is shown in Table 5.

Table 5: Temporal schedule of events during the drinking session.

Minutes After Drinking	
0	Drink 1
6	Drink 2
26	BAC
30	Test #1
56	BAC
60	Test #2
72	BAC
86	BAC
90	Test #3
106	BAC
110	Test #4
120	BAC

After the four tests concluded, all participants completed the "beverage rating" and the "desire to resist the drug effect" scales. They were then fully debriefed and paid (Appendix B14).

Participants were also given a general information sheet about alcohol and its effects (Appendix C6).

Criterion for positive reinforcement and monetary incentive

The criterion for administering positive reinforcement, and the monetary bonuses that were offered as an incentive was identical to that used in the preliminary investigation. Thus the number of inhibitions displayed by a participant on a treatment test was compared to the number of inhibitions he displayed on his pre-treatment baseline test. Whenever the number of inhibitions he displayed on a treatment test matched or exceeded his pre-treatment test score, reinforcement ("yes") was administered, or a monetary bonus (25 cents) was earned and paid at the conclusion of the experiment. A match was defined as coming within two inhibitions of the participant's pre-treatment baseline score.

As a precaution to ensure that participants did not try to match or increase response inhibitions by deliberately slowing the response to go-signals, they were also warned that they had to maintain their RT to the go-signal in order to obtain reinforcement ("yes") or monetary bonuses. Deliberate slowing of the response to a go-signal was defined as a RT that was 50 msec or more slower than a participant's mean baseline RT to a go-signal.

Data Analysis

The degree to which alcohol changed inhibitions from a participant's drug-free baseline was of primary interest. Thus, treatment effects were assessed using an analysis of variance (ANOVA) of change scores. The change in total inhibitions and in mean RT to the go-signal were calculated separately, by subtracting a participant's pre-treatment baseline score from each of his test scores under treatment. Each of these measures was used in factorial analyses of variance (ANOVAs) to

test the experimental hypotheses.

The prediction that the administration of alcohol in a novel setting reduces the impairing effect of alcohol on inhibitory control was tested in Phase 1 of the experiment by using the alcohol (A) and placebo control (P) groups in a 2(setting) X 2(A and P treatment) X 4(test) ANOVA. A priori simple-effect comparisons tested the hypotheses about specific group differences.

The prediction that the effect of positively reinforcing inhibitions under alcohol depends on the setting was tested in Phase 2 of the experiment using the three alcohol treatment groups in a 2(setting) X 3(AR, AM and A treatment) X 4(test) ANOVA, and a priori simple-effect comparisons tested hypotheses about specific groups.

The treatment effects also could be tested by covariance analyses (ANCOVA) of the actual scores, using the pre-treatment baseline score as the covariate. ANOVAs using change scores and ANCOVAs of actual scores were performed on each measure. Because both analyses led to similar conclusions, and the analysis using change scores show the actual degree of change instead of adjusted means, the analyses of change scores are presented in the text. Confirmatory ANCOVAs are included in appendices.

Results

All raw data for each participant are reported in Appendix I. In the course of conducting the experiment, eight participants had to be replaced because they failed to cooperate or understand the task instructions.

Procedural Checks

Drinking Habits Participants were between 19 and 23 years of age (M=19.8, SD=0.97) and reported drinking alcohol on a social basis for an average of 3.1 years. Each of the three drinking

habit measures of the eight groups was compared by one-way ANOVAs. One participant failed to provide information on the "duration of drinking occasions" and this resulted in the loss of his data for that measure. There were no significant group differences in any of the three measures of drinking (ps>.323) (Appendix D1). The sample of participants (N=72) reported a mean (SD) drinking frequency of 1.4 (0.93) times per week, with a mean (SD) dose of 1.2 ml/kg (0.68) per occasion. For a man of average weight (75 kg) this dose would be equivalent to five beers containing 5% alcohol/volume. The mean (SD) duration of drinking was 3.9 (1.5) hours. These drinking habits are similar to those of participants in the preliminary investigation.

Beverage Rating All participants who received a placebo (groups F-P and N-P) rated their beverage above zero. Overall the mean rating was equivalent to 1.6 bottles of beer. This indicated that participants believed that the beverage contained alcohol. Thus, the placebo appeared to be credible.

Desire to resist drug effect So many participants had difficulty understanding the meaning of this question that the findings are not presented in the thesis.

Pre-treatment Baseline Performance The results of the baseline test of the four groups who would receive different treatments in either the N or F setting was compared. A 2(setting) X 4(treatment) ANOVA of the mean number of inhibitions revealed no significant main effect of setting, $F(1,64)=1.65$, $p=.203$, treatment, $F(3,64)=1.49$, $p=.226$, or setting X treatment interaction, $F(3,64)=0.10$, $p=.962$ (Appendix D2, Table 1a; see Table 1b for the mean (SD) inhibitions of each group). Thus, there were no differences among the groups in their baseline inhibition scores. In the entire sample (N=72), the mean number of inhibitions to the 48 stop-signals presented during the baseline test was 23 (SD=9). Thus, participants inhibited their response to 48% of the stop-signals

presented during the test.

The data from the preliminary investigation had revealed that inhibitions on the baseline test declined when the stop-signal delay was lengthened. In addition, so few inhibitions were displayed at the longest (350 msec) stop-signal delay, that this delay could be imposing a floor effect making it impossible to observe any decrease in response inhibitions. In the present study, an examination of the mean number of inhibitions on the baseline test at each stop-signal delay showed a similar trend. At the shortest (50 msec) delay, a mean of 9.7 (SD=2.3) inhibitions was displayed. As the delay was increased from 150 to 250 msec, mean inhibitions dropped progressively from 7.9 (SD=3.2) to 3.8 (SD=3.2). At the longest (350 msec) delay, a mean of only 1.7 (SD=2.0) was displayed. Twelve stop-signals were presented at each stop-signal delay so the percentage of successful inhibitions at each stop-signal delay were 81, 66, 32 and 14, respectively. Thus, the possibility that the 350 msec stop-signal delay weakened the evidence of treatment effects was examined by analyzing the data on inhibitions with and without the results obtained from the 350 stop-signal delay.

A 2(setting) X 4(treatment) ANOVA of the mean RT to the go-signals during the baseline test revealed no main effect of setting, $F(1,64)=1.91$, $p=.172$, or treatment, $F(3,64)=1.82$, $p=.152$], and no significant interaction, $F(3,64)=0.96$, $p=.418$ (Appendix D2, Table 2a; see Table 2b for the mean (SD) RT for each group). Thus, the groups did not differ in the pre-treatment baseline RT. The mean RT in the entire sample (N=72) was 434.06 msec (SD=66.90).

The incidence of errors displayed by groups during the baseline test ranged from 4 to 12 errors (3.1% to 9.3%) for an overall average of 6.2%.

BAC With the exception of the two placebo groups, the remaining six groups received alcohol and

their BACs were measured six times, at intervals during the drinking session.[3] The comparability of their BACs was tested by a one-way 6 (group) X 6 (time) repeated measures ANOVA (Appendix D3, Table 1a). The analysis revealed only a significant main effect of time, $F(5,235)=42.82$, $p<.001$. The absence of any other effects indicated that the BACs of the six groups did not differ. Appendix D3, Table 1b, presents the mean (SD) BAC of all alcohol groups at each interval, and Figure 3 illustrates these results. The figure indicates that the mean peak BAC of 74 mg/100 ml occurred 56 minutes after drinking commenced, just before the second test began, and remained at that level until this test ended. The figure also shows that the third and fourth tests occurred while the BAC was declining, and the mean BAC was 58 mg/100 ml after all four tests were completed.

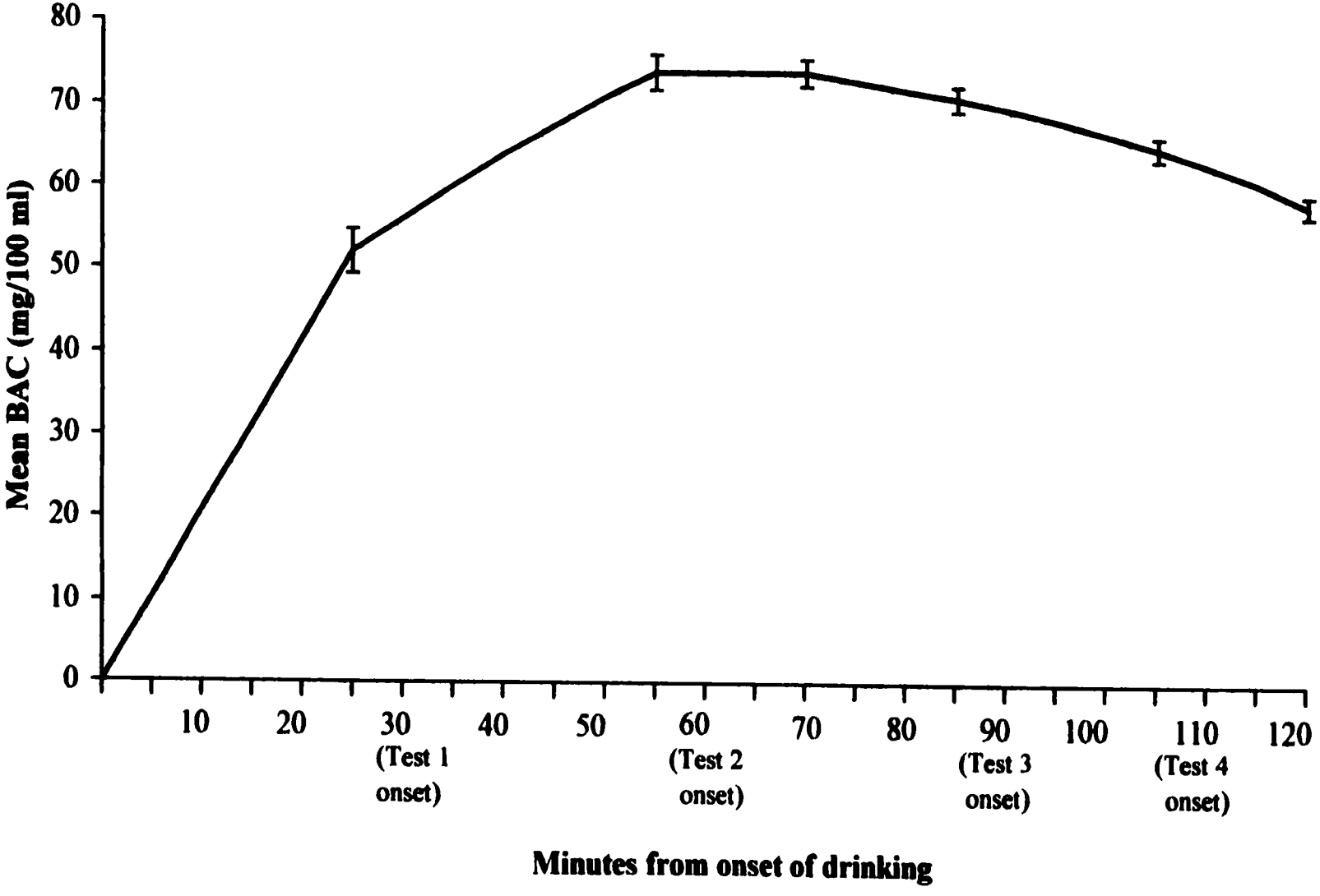
In summary, the results of the procedural checks detected no significant differences among the groups. Thus, drinking habits, pre-treatment baseline performance of the go-stop task, and the BACs of the groups that received alcohol are unlikely to account for any differences in the performance of the groups under their respective treatments.

Phase 1: Alcohol and the setting

Inhibitions The change in inhibitions displayed by A and P groups in the N and F settings was assessed by a 2(setting) X 2(treatment) X 4(test) ANOVA (Table 6). No main effect or interactions reached conventional significance levels (i.e., $p<.05$). The absence of a test X treatment interaction ($F(3,96)=0.34$, $p=.795$) is pertinent to the experimental hypotheses, and indicates that the change in inhibitions displayed under alcohol and placebo did not differ across the four tests. Thus, the mean changes in inhibitions were averaged over all tests and a priori hypotheses were tested by

[3] One participant's data were lost due to a mechanical difficulty with the BAC machine.

Figure 3: BACs of six groups at six time intervals. Vertical bars show SEMs. The onset of go-stop task tests are shown on the x-axis.



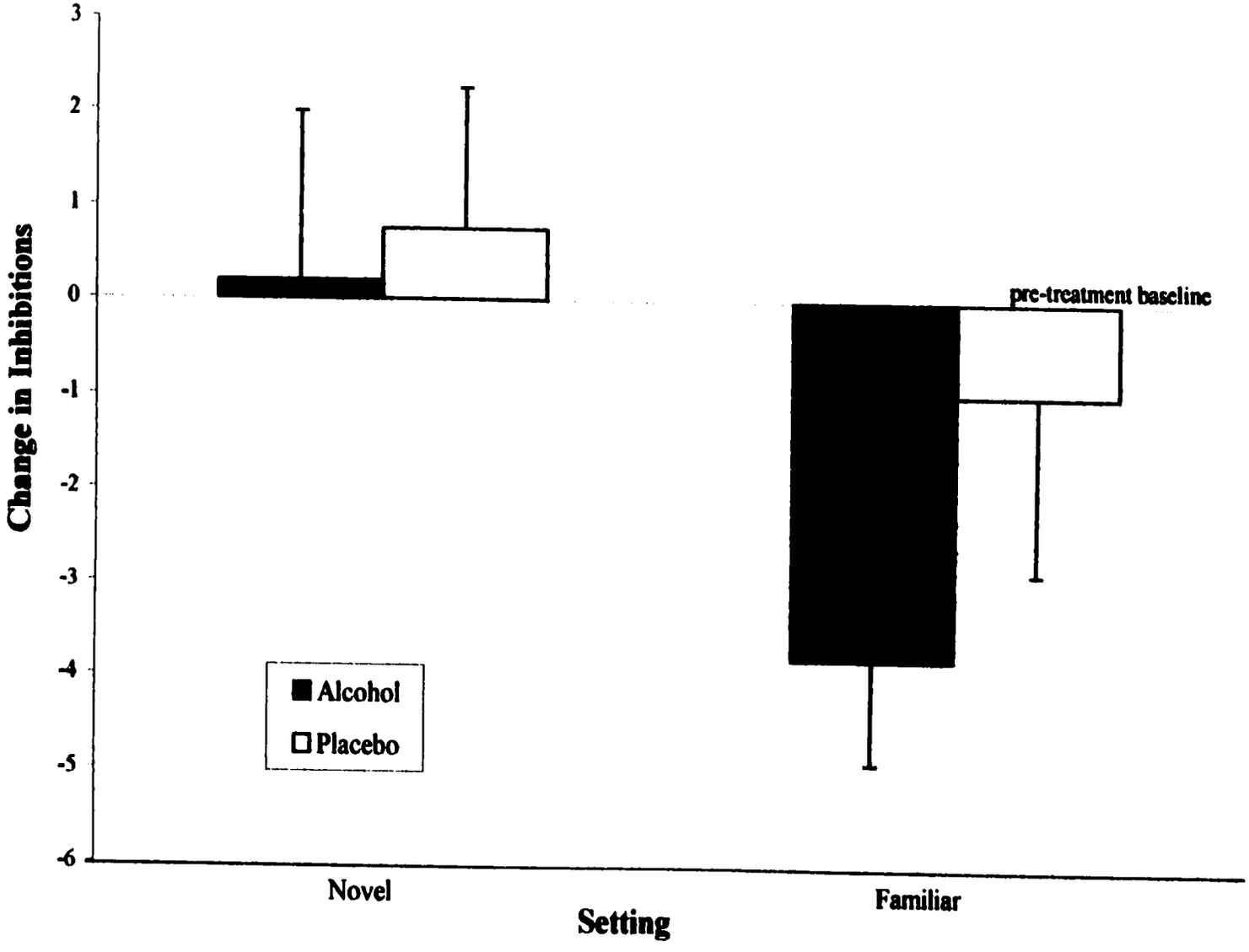
simple-effect comparisons. The between-subjects error term and corresponding degrees of freedom from the ANOVA were used to make these comparisons (Howell, 1992). The mean (SD) change in inhibitions on each test, and the average change on all tests are shown for each group in Appendix E1.

Table 6: ANOVA of the change in inhibitions on four tests of A and P groups in a novel or familiar setting.

Source	df	MS	F	p
Setting (S)	1	294.69	3.11	.087
Treatment (T)	1	103.36	1.09	.304
S X T	1	46.69	0.49	.488
Error	32	94.80		
Tests (Tt)	3	29.68	2.48	.065
Tt X S	3	11.53	0.97	.413
Tt X T	3	4.08	0.34	.795
Tt X S X T	3	10.08	0.84	.473
Error	96	11.95		

The hypothesis that the effect of alcohol would be less intense in a novel than in a familiar setting was tested by comparing the mean change in inhibitions of groups N-A and F-A. This one-tailed test revealed a significant difference, $F(1,32)=3.04$, $p=.046$. The result is illustrated in figure 4 in which the overall mean change in inhibitions of each group is shown. Zero on the vertical axis represents participants' pre-treatment baseline inhibition score. A negative change indicates a reduction in inhibitions during treatment, whereas a positive change indicates an increase in inhibitions relative to baseline performance. The figure shows that the alcohol group in a novel

Figure 4: Mean change in inhibitions of alcohol and placebo groups in novel or familiar settings. Vertical bars show SEMs.



setting (group N-A) showed little change in inhibitions compared to the alcohol group in a familiar setting (group F-A). The mean (SD) change in inhibitions of the N-A group was +0.19 (5.5), whereas the mean change of group F-A group was -3.8 (3.3). Thus, in accord with the hypothesis, the impairing effect of alcohol was reduced in a novel setting compared to a familiar setting.

The change in inhibitions displayed by the placebo groups in the N and F settings did not differ, $F(1,32)=0.56$, $p=.458$. Figure 4 shows that the number of inhibitions displayed by these groups changed very little from their baselines. In fact, paired t-tests revealed that their mean change in inhibitions did not differ from their zero baseline ($p>.622$). Thus, setting had no detectable impact on response inhibition when participants were drug-free, even though they expected alcohol. Figure 4 shows that participants in the F-A group displayed a greater reduction in response inhibition than the placebo groups and this was confirmed by a one-tailed comparison between group F-A and the two placebo groups combined, $F(1,32)=3.46$, $p=.036$. Thus, response inhibitions were reduced by alcohol in the familiar setting.

Conclusions from the ANOVA were confirmed by covariance analyses and comparisons based on the adjusted scores from a 2(setting) X 2(treatment) ANCOVA (Appendix E2). Appendix E3 also shows the actual mean number of inhibitions on the pre-treatment baseline test and the average number of inhibitions on the four treatment tests for each group.

A supplementary ANOVA of the change in inhibitions and an ANCOVA of the actual inhibition scores was performed excluding the results from the longest (350 msec) stop-signal delay (Appendix E4). In accord with the notion that the 350 msec delay imposed a floor effect on inhibitions, the results of these analyses provided stronger support for the hypotheses.

Reaction time (RT) to go-signals Table 7 shows the results of a 2(setting) X 2(treatment) X 4(test) repeated-measures ANOVA of change in RT. This analysis revealed that no main effects or interactions reached $p<.05$[4]. The mean (SD) RT on each test, and averaged over all tests is shown for each group in Appendix F1).

A 2(setting) X 2(treatment) ANCOVA of the RT measures (Appendix F2) obtained results that were consistent with those from the ANOVA using change scores. The actual mean RT on the pre-treatment baseline test and the average RT on all four treatment tests for each group are shown in Appendix F3.

The mean number of errors of the groups ranged from 7 to 8, and represents an error rate of 5.5% to 6.3%, for an overall average of 5.9%. Appendix F4 presents the mean (SD) change in number of errors for each group, as well as the mean number of errors during the pre-treatment test and the average errors on all four treatment tests for each group.

[4] The main effect of treatment approached the .05 level, $F(1.32)=3.95$, $p=.055$. The mean change in RT of the groups is shown in appendix F1 along with a discussion of these data that explains why they cannot account for the change in inhibitions displayed by the groups.

Table 7: ANOVA of the change in RT (msec) on four tests of A and P groups in a novel or familiar setting.

Source	df	MS	F	p
Setting(S)	1	536.89	0.05	.822
Treatment (T)	1	41212.05	3.95	.055
S X T	1	41.42	0.004	.950
Error	32	10431.97		
Tests (Tt)	3	860.81	1.17	.324
Tt X S	3	1381.65	1.88	.138
Tt X T	3	1276.81	1.74	.164
TtxSxT	3	942.58	1.29	.284
Error	96	733.79		

Phase 2: Alcohol, positive reinforcement and the setting

Inhibitions The change in inhibitions was tested by a 2(setting) X 3(treatment) X 4(test) ANOVA (Table 8). The ANOVA revealed only a significant main effect of setting, $F(1,48)=9.26$, $p=.004$. The main effect of setting indicated that the reduction in inhibitions under alcohol was greater in the familiar setting (mean change=-3.58, SD=3.7) than in the novel setting (mean change=-0.22, SD=4.4). The table also shows that there was no significant main effect of test and no interactions involving tests (ps>.25).

The absence of a test X treatment interaction ($F(6,144)=1.32$, $p>.250$) indicated that the change in inhibitions in the treatment groups did not differ across the four tests on the go-stop task. Thus, the a priori hypotheses could be tested by simple-effect comparisons using the mean change in inhibitions averaged over tests. The between-subjects error term and

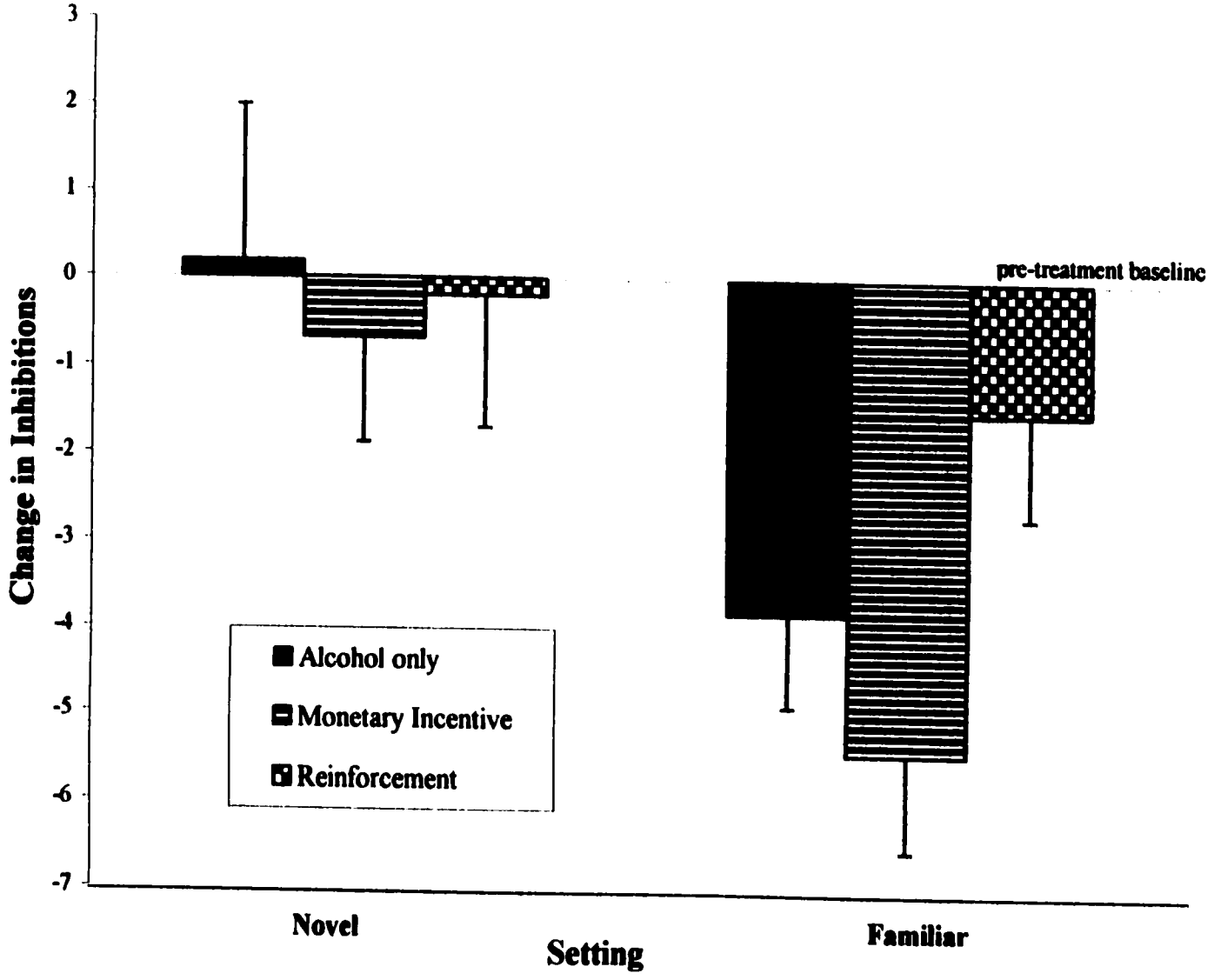
Table 8: ANOVA of the change in inhibitions in three treatment groups on four tests under alcohol in novel or familiar settings.

Source	df	MS	F	p
Setting (S)	1	610.04	9.26	.004
Treatment (T)	2	88.29	1.34	.271
S X T	2	59.76	0.91	.410
Error	48	65.87		
Tests (Tt)	3	6.19	0.49	.691
Tt X S	3	8.86	0.70	.554
Tt X T	6	16.77	1.32	.250
Tt X S X T	6	9.88	0.78	.587
Error	144	12.67		

corresponding degrees of freedom from the ANOVA were used to make these comparisons (Howell, 1992). Appendix G1 presents the mean (SD) change in number of inhibitions on each group on each test, and averaged over tests.

It was predicted that reinforcement in a familiar setting would reduce the impairing effect of alcohol on inhibitions compared to the groups that received a monetary incentive or alcohol *only*. In accord with the hypothesis, the reduction in inhibitions in the monetary incentive (F-AM) and the alcohol (F-A) groups did not differ from each other, $F(1,48)=0.73$, $p=.396$, and a one-tailed comparison of the reinforced (F-AR) group to the other two groups combined was significant, $F(1,48)=3.56$, $p=.033$. This is illustrated in Figure 5, which shows the mean change in inhibitions

Figure 5: Mean change in inhibitions of three treatment groups under alcohol in novel or familiar settings. Vertical bars show SEMs.



of the treatment groups[5]. The mean (SD) reduction in inhibitions of group F-AR was only -1.5 (3.6), whereas groups F-A and F-AM combined displayed an average drop of -4.63 (3.4) inhibitions. In fact, a paired t-test showed that the mean change in inhibitions in the F-AR group did not differ significantly from the zero baseline, ($t(8)=-1.26$, $p=.243$).

The novel setting was predicted to reduce the impairing effect of alcohol on response inhibition to such a degree that no group differences could be detected. This was tested by comparing the mean change in inhibitions displayed by the groups tested in the novel setting. The groups treated with alcohol (N-A) and with an added monetary incentive (N-AM) did not differ, $F(1,48)=0.20$, $p=.655$. When these N-A and N-AM groups were combined and compared to the reinforced group (N-AR) no significant difference was observed, $F(1,48)=0.001$, $p=.980$. Figure 5 shows that the inhibitions of groups tested in a novel setting changed very little from their pre-treatment baseline, regardless of the treatment they received. In fact, the change in inhibitions did not differ from zero in any of the groups ($ps>.593$).

A 2(setting) X 3(treatment) covariance analyses and comparisons based on the adjusted scores from the ANCOVA confirmed these conclusions (Appendix G2). Appendix G3 shows the mean number of inhibitions on the pre-treatment baseline test and the average number of inhibitions on the four treatment tests for each group.

Supplementary ANOVA and ANCOVA analyses of the data on inhibitions that excluded the results of the 350 msec delay are presented in Appendix G4. They also confirmed the evidence presented here using all four stop-signal delays, and provided stronger support for the prediction

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[5] The alcohol treatment is labeled "alcohol only" in the figure in order to be clear that while all groups received alcohol, one received alcohol and reinforcement, another received alcohol and a monetary incentive and another received *alcohol only* (i.e., no reinforcement or monetary incentive).

that reinforcement treatment reduces the effect of alcohol on response inhibition in a familiar setting.

RT to go-signals Table 9 shows the results of a 2(setting) X 3(treatment) X 4(test) repeated-measures ANOVA of change in RT. This analysis revealed that no main effects or interactions reached $p<.05$. The mean (SD) change in RT on each test, and averaged over all tests is shown for each group in Appendix H1.

A 2(setting) X 3(treatment) covariance analysis (Appendix H2) was conducted and revealed results consistent with the ANOVA using change scores. The actual mean (SD) RTs on the pre-treatment baseline test, and on all treatment tests are shown in Appendix H3.

Table 9: ANOVA of the change in RT (msec) in three treatment groups on four tests under alcohol in novel or familiar settings.

Source	df	MS	F	p
Setting(S)	1	890.81	0.11	.743
Treatment (T)	2	23862.88	2.92	.064
S X T	2	3710.70	0.45	.638
Error	48	8182.68		
Tests (Tt)	3	616.35	0.86	.464
Tt X S	3	1456.20	2.03	.112
Tt X T	6	1450.20	2.02	.066
TtxSxT	6	1363.12	1.90	.085
Error	144	717.14		

The incidence of errors on treatment tests of each group ranged from 7 to 17. This represents an error rate of 5.5% to 13.3%, for an overall average of 9.4%. Appendix H4 shows the mean (SD)

change in number of errors for each group, as well as the mean number of errors during the pre-treatment baseline test and the average errors on all four treatment tests for each group.

Summary

This experiment showed that the impairing effect of alcohol on inhibitory control of behaviour can be affected by two different environmental factors: prior drug-free familiarity with the experimental setting in which the drug is administered, and immediate positive reinforcement of inhibitions. The results showed that response inhibitions were reduced by alcohol in the familiar setting, whereas no appreciable drug effect was evident in the novel setting. Comparisons of groups that received alcohol and those that received a placebo in the two settings showed that the setting factor only affected response inhibitions under alcohol.

Evidence from groups tested in the familiar setting supported the prediction that immediate positive reinforcement of inhibitions under alcohol would reduce the drug-induced impairment of inhibitory control of behaviour. In the novel setting, inhibitions were unaffected by alcohol and thus no effects of reinforcement were observed.

No systematic group differences in RT to go-signals were found in this study. Thus, the results showed that alcohol can impair inhibitory control in a familiar, but not in a novel setting. Moreover, alcohol-induced impairment is shown only in a familiar setting when no immediate positive reinforcement for response inhibition is provided.

GENERAL DISCUSSION

This thesis was designed to examine the extent to which, and conditions under which, alcohol reduces inhibitory control of behaviour in social drinkers. The results of the preliminary investigation indicated that reinforcement appeared to improve response inhibition under alcohol and suggested that an additional factor, prior drug-free experience in a setting, might affect the intensity of the effect of alcohol on response inhibition. Thus, the main study was designed to evaluate the effects of reinforcement and the setting on response inhibition under a moderate dose of alcohol.

Phase 1 of the main experiment tested the prediction that the impairing effect of alcohol on inhibitory control would be reduced in a novel setting compared to a familiar setting. Thus, groups of participants received an alcohol or a placebo beverage and performed the go-stop task when the setting was either novel or familiar. The results showed that response inhibitions were not significantly affected by alcohol in the novel setting, whereas the drug significantly diminished inhibitions in the familiar setting. The inhibitions displayed by those who received a placebo did not differ in the novel and familiar settings. These findings indicated that the degree of familiarity with the experimental setting only influenced inhibitory control when participants consumed alcohol.

Phase 2 of the study tested the hypothesis that immediate positive reinforcement would reduce the impairing effect of alcohol on inhibitory control in a familiar setting. Thus, groups of participants in novel or familiar settings performed the go-stop task under alcohol, or with the addition of positive reinforcement, or with a monetary incentive for maintaining a sober level of inhibitory control. Comparisons among groups in the familiar setting showed that inhibitions were reduced when alcohol alone was administered and when a monetary incentive was added. In accord

with the hypothesis, immediate reinforcement (in the form of approving verbal feedback) diminished the impairing effect of alcohol on inhibitory control in the familiar setting. In the novel setting, however, alcohol had no significant effect on inhibitions. In fact, under all the treatments administered in the novel setting, inhibitions showed little change from drug-free levels of performance. Thus, the results demonstrated that the effects of alcohol and of reinforcement on inhibitory control depend on the novelty-familiarity dimension of the setting.

None of the groups showed any systematic changes in response RT to go-signals that could account for these results. In addition, participants' drinking habits and blood alcohol concentrations did not differ. Thus the thesis identified two important environmental factors that influence a social drinker's ability to inhibit a response under a moderate dose of alcohol: the novelty-familiarity dimension of the setting, and immediate positive reinforcement of inhibitions.

Confusion over whether alcohol causes an inevitable reduction of inhibitory control, or whether alcohol only provides an excuse for inappropriate behaviour provided an impetus for this thesis research. The findings indicated that alcohol can impair inhibitory control, but that this loss of inhibitory control is not an inevitable pharmacological effect of alcohol. The results showed that the novelty of the drinking setting and reinforcement for inhibitions determine the extent to which alcohol impairs inhibitory control of behaviour. These findings are important because they indicate that, under a moderate dose of alcohol, individuals are not necessarily hapless victims of the disinhibiting effect of alcohol and, they provide a direct challenge to the claim that alcohol "causes" disinhibition of behaviour (Bushman & Cooper, 1990).

Past research that examined "disinhibition" under alcohol has relied on indirect measures (i.e., an increase in a response such as aggression) to infer a loss of inhibitory control (Steele &

Southwick, 1985; Stuntich & Taylor, 1972). The go-stop task that was used in the present research provided a direct observation of response inhibition. The use of this measure represents a considerable procedural advance in research on inhibitory control. The interpretation of the effect of alcohol on inhibitory control has also been problematic because many experimental paradigms have included rewards and punishments for behaviour under alcohol (e.g., winning a game, getting shocked). Contaminating the results with these environmental factors also might have contributed to the inconsistent results of this work. Although researchers suspected that the inconsistent results of these studies might be due to the interaction of some environmental factors with alcohol, none was identified. The stop-signal paradigm, used in the present research, allowed environmental factors to be excluded or included in the experiment. Thus, the effect of alcohol on inhibitory control could be tested and compared to the effect of alcohol when environmental variables were systematically introduced.

The finding that reinforcement of inhibitions in a familiar setting can reduce the disrupting effect of alcohol on inhibitory control is consistent with an associative learning interpretation. In theory, whenever some environmental consequence follows a response, it provides a basis for learning to expect this outcome whenever the response is displayed. If the expected outcome is favourable, the response will increase. In the present research, the association between a favourable consequence (i.e., reinforcement) for displaying a sober level of inhibitory control reduced the impairing effect of the drug, presumably because inhibitions were expected to yield a positive outcome. A large amount of research, consistent with this interpretation, has shown that reinforcement for performing well under alcohol reduced the impairing effect of the drug (Fillmore & Vogel-Sprott, 1997; Vogel-Sprott, 1992). Moreover, when drinkers were reinforced for

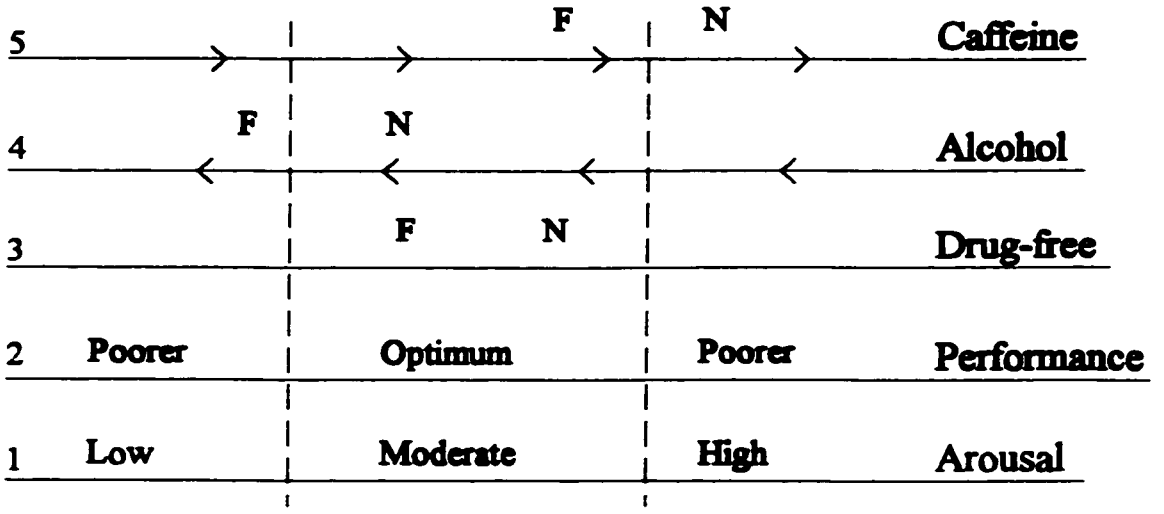
displaying gross impairment under alcohol, their behaviour adhered to this standard (Zack & Vogel-Sprott, 1997), presumably because intoxicated performance was expected to result in a favourable consequence. This previous alcohol research tested the effect of reinforcement on drinkers who performed tasks when the setting was familiar. In the present research, the results when drinkers were tested in a familiar setting were consistent with those findings.

When reinforcement was provided to drinkers in a novel setting, no effect of alcohol was detected. The "setting" in the present research contained several factors (i.e., the testing procedure, the task, the laboratory and the experimenter) that may have been responsible for reducing the alcohol effect. Because all of these aspects of the setting were new to drinkers, it is not clear how many or which novel aspects of a setting reduce the impairing effect of alcohol on response inhibition. It may be that novelty of only one of these factors may be sufficient, or some combination of these novel variables may be required to counteract the effect of alcohol on inhibitory control. The strong impact of novelty in eliminating the effect of alcohol on inhibitions indicates the importance of additional research to identify the exact novel component or components of a situation that influence inhibitory control under alcohol. This research would provide the necessary information to help guide the development of theoretical explanations for the effect of the novelty of the drinking setting on response inhibitions.

The small amount of animal research that exists on the effect of novelty of the setting on drug effects has suggested that a novel setting may increase fear and anxiety (Rushton et al., 1963; Steinberg et al., 1961). Fear and anxiety are generally associated with an increase in arousal. Arousal can vary on a continuum from low to high levels. According to the Yerkes-Dodson Law, performance has an inverted "u"shape relation with arousal such that individuals at a moderate

level of arousal demonstrate optimal performance and individuals at high and low levels demonstrate poorer performance (Yerkes & Dodson, 1908, as cited in Dworetzky, 1985). This is illustrated by lines 1 and 2 in the drawing below.

Possible change in arousal and performance in novel (N) and familiar (F) settings:



Because the novel and familiar groups in this research did not differ on their drug-free inhibitory control, their level of arousal was likely within the moderate range. However, the participants in the novel setting could have had a somewhat higher level of arousal compared to those in the familiar setting (see line 3). As both settings still allow arousal to remain within the moderate range, no group difference in inhibitions would be expected, and none was seen. When alcohol is administered, its depressant-sedative effects should reduce arousal. This should shift the position

of both groups to the left, as shown in line 4. This shift could result in lower arousal in the familiar setting to a degree that is no longer within the moderate range. This could result in poorer performance under alcohol (i.e., fewer inhibitions) in the familiar setting. In contrast, even though alcohol reduces arousal, the shift to the left in the novel group could still be within the moderate range of arousal and so no alcohol effect on performance is observed. This interpretation could be tested using the go-stop task and administering a stimulant, such as caffeine. For example, when caffeine is administered, its stimulating and excitatory action should increase arousal, shifting the position of the novel and familiar groups to the right, as shown in line 5. This shift could result in raising the arousal level in the novel group to a degree that is no longer within the moderate range. This could result in poorer performance under caffeine (i.e., fewer inhibitions) in the novel setting due to the high level of arousal. Previous research has indicated that administration of caffeine might affect performance by altering arousal levels (e.g., Anderson & Revelle, 1982).

To date, it appears that only one other alcohol experiment with humans has examined the effect of the novelty-familiarity dimension of a setting (Newlin & Pretorius, 1991). These researchers measured physiological responses and self-reported measures of drunkenness and found that the drug effect was reduced in a novel setting (Newlin & Pretorius, 1991). The present research extends this finding to response inhibition, and raises the possibility that the novelty of a drinking setting may also reduce the effect of alcohol on other cognitive or motor skills.

The evidence that response inhibition under alcohol can also be influenced by reinforcement raises questions about other reinforcement procedures that might influence inhibitory control under alcohol. Negative reinforcement (i.e., the avoidance of an unfavourable consequence) for a sober level of performance has been shown to result in less impairment of motor skills under a moderate

dose of alcohol (Zack & Vogel-Sprott, 1995). In addition, drinkers' motor skill performance will conform to either a sober or intoxicated standard of performance depending on which one yields a favourable outcome (Zack & Vogel-Sprott, 1997). The possibility that the effect of alcohol on response inhibition can be intensified or diminished, and that this may be achieved by various reinforcement procedures, remains to be investigated.

Past research with motor skills has also indicated that the withdrawal of reinforcement for tolerance results in a loss of tolerance to the impairing effects of alcohol (Mann & Vogel-Sprott, 1981; Zack & Vogel-Sprott, 1993). These results could be explained by associative learning theory. When the favourable outcome for a sober level of performance no longer occurs, the expected reward for resisting the drug effect is contradicted and in the absence of this expectancy, impairment is displayed. It would be important to determine if the withdrawal of immediate positive reinforcement for inhibition under alcohol will similarly result in the return of impairment.

The results of this research on inhibitory control are based on undergraduate male social drinkers under moderate doses of alcohol. Future research that administers higher doses of alcohol and tests heavier drinkers outside a university community could determine whether the effects of reinforcement and the novelty-familiarity dimension of the drinking setting apply to higher blood alcohol levels and different drinking populations. The generality of the present findings should also be tested with women. Although little research has been conducted on the effects of alcohol on female drinkers, some research has shown that the impairment of inhibitory control in women is similar to that shown by men under a moderate dose of alcohol (Mulvihill et al., 1997). Thus, the go-stop task and the procedure developed in this thesis could be applied to assess the possibility that inhibitory control of women under alcohol could be improved by reinforcement or altered by

the novelty-familiarity dimension of the setting.

The direct measure of inhibitory control provided by the go-stop task might be of interest to researchers attempting to understand the relationship between inhibitory control and corresponding changes in the brain. Recent research has used functional MRI (magnetic resonance imaging) and PET (positron emission tomography) technology (Kawashima et al., 1996; Konishi et al., 1999) to examine the correspondence between performance on a go/no-go task and changes in activity to different areas of the brain, in drug-free participants. Use of the go-stop task could add to this research by providing a measure of inhibition of an ongoing response rather than measuring separate go and stop responses as provided in the go/no-go task. Furthermore, the go-stop task could be used with participants who are drug-free as well as under a dose of alcohol while brain activity is monitored. This research could begin to provide evidence that links alcohol-induced changes in brain activity with corresponding changes in inhibitory control of behaviour.

The finding that individuals are affected differently by alcohol in novel and familiar drinking settings might have practical "real world" implications. It is likely that most drinking occurs in settings with which drinkers are familiar (e.g., a local pub, at a friend's house). Given the results of the thesis, these settings should result in less inhibition under alcohol unless some reward is expected for sober behaviour. There are also some occasions when drinking may occur in a new situation (e.g., visiting a strange bar on a blind date). In this novel setting, the results of the present research suggest that behavioural inhibition is less likely to be impaired by alcohol. This resistance to the disinhibiting effect of alcohol might be adaptive to some degree in that a person may be less likely to "lose control" and say or do something inappropriate in a new situation.

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APPENDICES

Appendix A: Preliminary Investigation

Appendix A1

Personal History Drinking Questionnaire (PDHQ):

#_____

Below are some questions which are primarily concerned with your personal drinking. Most ask you to answer according to what is most typical or usual for you. Please try to answer each question as honestly as possible.

1) Age _____ Weight ____ Height ____

2) How often, on average, do you drink alcohol? (Choose only **one**)

A) Only on special occasions, how many times per year? _____
B) Monthly, how often? _____
C) Weekly, how often? _____
D) Daily, how often? _____

3) What alcoholic beverage do you prefer? _____

4) What alcohol beverage do you usually drink? _____

5) In terms of the beverage indicated in question 4, what is the **AVERAGE** quantity you drink in a single drinking occasion? (Choose only **one**)

A) WINE (estimate ounces 1 2 3 4 5 6 7 8 9 10 or ____
B) BEER (bottles) 1 2 3 4 5 6 7 8 9 10 or ____
C) BEER (draft glasses) 1 2 3 4 5 6 7 8 9 10 or ____
D) LIQUOR (assume 1.5 ounces per drink and estimate the number of drinks) 1 2 3 4 5 6 7 8 9 10 or ____

4) How long does your typical drinking occasion last? (Choose only **one**)

A) _____ MINUTES
B) _____ HOURS
C) _____ DAYS

Appendix A1 (cont.)

Scoring instructions for PDHQ:

Frequency of drinking was calculated as the number of times per week the individual reported consuming alcohol.

Dose was defined as volume of absolute alcohol (ml) per kg body weight consumed on a typical drinking occasion. Total ml of absolute alcohol was determined by multiplying the number of drinks (in ml) reported to be consumed on a typical drinking occasion by the concentration of alcohol in the particular beverage.

Beer (bottles & draft) was taken as 5% alcohol by volume.
Liquor was taken as 40% alcohol by volume.
Wine was taken as 15% alcohol by volume.

A bottle of beer contains 341 ml therefore 341 x 5% = 17.05 ml of absolute alcohol.
Draft glasses are 227 ml x 5% = 11.35.
Liquor 45 ml x 40% = 18.0.
Wine (1 fluid ounce) is 28 ml x 15% = 4.20.
Wine (4 fluid ounces) is 112 ml x 15% = 16.80.

The ml of alcohol was multiplied by the number of standard drinks a participant consumes and this amount was then divided by body weight in kg to produce a dose score for each participant.

e.g., The dose for a participant who weighs 75 kg and typically drinks 5 bottles of beer is:

$$17.05 \times 5 / 75 = 1.14$$

Duration of a Drinking Occasion was measured in hours.

Appendix A2

Temporal schedule of events:

0	Drink 1
20	BAC
21	Drink 2
23	Test 1
40	BAC
41	Drink 3
43	Test 2
60	BAC
63	Test 3
70	BAC
80	BAC
83	Test 4
100	BAC
103	Test 5
120	BAC

Appendix A3

Trimmed reaction times:

Van Selst & Jolicoeur (1994) point out that previous work shows that the traditional calculations for determining outliers are problematic and can be affected by sample size. Thus, other methods have been developed which use a different criterion ("moving criterion") for determining outliers depending on sample size (i.e., number of observations for a participant) (Van Selst & Jolicoeur, 1994). The non-recursive procedure with moving criterion is one of the procedures described by Van Selst & Jolicoeur (1994) and was used to determine which RTs represent outliers in this data set. The criterion cut-off was 2.5 (see Table 4, Van Selst & Jolicoeur, 1994).

Comparison of ANOVAs using untrimmed and trimmed RT (msec) to correct go-signals (correct key presses)* for reinforcement and money treatments on five tests under alcohol:

		Untrimmed RT			Trimmed RT		
Source	df	MS	F	p	MS	F	p
Reinforcement (R)	1	15670.67	1.34	.259	9313.66	0.97	.334
Money (M)	1	49248.00	4.20	.051	39363.55	4.12	.054
M X R	1	4225.77	0.36	.554	3513.91	0.37	.550
Error	24	11724.36			9562.04		
Tests (T)	4	960.55	0.91	.465	431.71	0.47	.761
T X R	4	655.39	0.62	.651	505.35	0.55	.703
T X M	4	1365.52	1.29	.281	937.71	1.01	.405
TxMxR	4	342.84	0.32	.862	306.19	0.33	.857
Error	96	1061.78			926.63		

* These analyses show the RT to correct go responses only.

Appendix A4

Comparison using RT (msec) to all go-signals (including errors, i.e., incorrect key presses) and RT (msec) to go-signals where no errors were made (both RT measures are trimmed):

	RT to All go-signals				RT to Correct go-signals		
Source	df	MS	F	p	MS	F	p
Reinforcement (R)	1	10097.89	1.04	.317	9313.66	0.97	.334
Money (M)	1	41228.38	4.26	.050	39363.55	4.12	.054
M X R	1	4359.91	0.45	.508	3513.91	0.37	.550
Error	24	9675.66			9562.04		
Tests (T)	4	307.28	0.34	.854	431.71	0.47	.761
T X R	4	669.64	0.73	.574	505.35	0.55	.703
T X M	4	976.48	1.06	.379	937.71	1.01	.405
TxMxR	4	468.52	0.51	.728	306.19	0.33	.857
Error	96	918.61			926.63		

Appendix A5

One-way ANOVAs on drinking habit measures of four groups:

Frequency:

Source	df	MS	F	p
Group	3	1.56	1.04	.393
Error	24	1.50		

Dose:

Source	df	MS	F	p
Group	3	0.44	0.82	.494
Error	24	0.53		

Duration:

Source	df	MS	F	p
Group	3	0.04	0.02	.995
Error	22	1.88		

Appendix A6

Drug-free baseline measures:

Table 1a. ANOVA on the number of inhibitions during the drug-free baseline test for reinforcement and money treatments on five tests under alcohol:

Source	df	MS	F	p
Reinforcement (R)	1	1.75	0.01	.907
Money (M)	1	170.04	1.36	.256
R X M	1	0.04	<0.001	.987
Error	24	125.31		

Table 1b. Mean (SD) number of inhibitions on the drug-free baseline test by group:

		Reinforcement (R)	
		Yes	No
Money (M)	Yes	ARM: 30.4(9.5)	AM: 30 (12.3)
	No	ARV: 25.6(6.3)	A: 25 (14.9)

Appendix A6 (cont.)

Table 2a. ANOVA on the mean RT (msec) during the drug-free baseline test:

Source	df	MS	F	p
Reinforcement (R)	1	121.68	0.01	.922
Money (M)	1	39276.81	3.14	.089
R X M	1	4.73	<0.001	.985
Error	24	12497.90		

Table 2b. Mean (SD) RT (msec) on the drug-free baseline test by group:

		Reinforcement (R)	
		Yes	No
Money (M)	Yes	ARM: 533.93(101.03)	AM: 528.93 (149.04)
	No	ARV: 458.20 (62.51)	A: 454.85 (116.90)

Appendix A7

BAC Measures:

Table 1a. ANOVA of the BACs for four groups at seven times during testing:

Source	df	MS	F	p
Group (G)	3	1725.48	2.87	.061
Error	21	601.30		
Time (T)	6	10396.93	114.12	<.001
T X G	18	74.73	0.82	.674
Error	126	91.11		

Table 1b. Mean (SD) overall BAC (mg/100 ml) by group:

		Reinforcement (R)	
		Yes	No
Money (M)	Yes	ARM: 55 (7)	AM: 61 (11)
	No	ARV: 66 (9)	A: 52 (8)

Appendix A8

ANCOVA and adjusted means for inhibitions:

Table 1. The test of interactions among drug-free baseline number of inhibitions and between-subjects factors (Money and Reinforcement):

Source	df	MS	F	p
Reinforcement (R)	1	21.36	1.23	.280
Money (M)	1	1.66	0.10	.760
R X M	1	14.05	0.81	.378
Covariate: Baseline (B)	1	1353.67	78.22	<.001
B X M	1	2.50	0.14	.708
B X R	1	3.37	0.20	.664
B X M X R	1	10.24	0.59	.451
Error	20	17.31		

Table 2a. ANCOVA of the number of inhibitions as a function of reinforcement and money treatments using the drug-free baseline inhibition score as a covariate:

Source	df	MS	F	p
Reinforcement (R)	1	109.63	7.05	.014
Money (M)	1	79.35	5.10	.034
R X M	1	7.15	0.46	.505
Covariate (Baseline)	1	2195.17	141.16	<.001
Error	23	15.55		

Appendix A8 (cont.)

Table 2b. Adjusted mean number of inhibitions for each group:

		Reinforcement (R)	
		Yes	No
Money (M)	Yes	ARM: 29.40	AM: 26.45
	No	ARV: 26.95	A: 21.98

Appendix A9

ANCOVA and adjusted means for RT (msec):

Table 1. The test of interactions among drug-free baseline RT and between-subjects factors (Money and Reinforcement):

Source	df	MS	F	p
Reinforcement (R)	1	1043.58	0.51	.482
Money (M)	1	0.13	<0.001	.994
R X M	1	1936.25	0.95	.341
Covariate: Baseline (B)	1	174920.65	85.91	<.001
B X M	1	169.74	0.08	.776
B X R	1	771.81	0.38	.545
B X M X R	1	1680.82	0.83	.374
Error	20	2036.17		

Table 2a. ANCOVA of RT as a function of reinforcement and money treatments using the drug-free baseline RT as a covariate:

Source	df	MS	F	p
Reinforcement (R)	1	1764.43	0.95	.340
Money (M)	1	4105.76	2.21	.150
R X M	1	714.80	0.39	.541
Covariate (Baseline)	1	240872.73	129.86	<.001
Error	23	1854.85		

Appendix A9 (cont.)

Table 2b. Adjusted mean RT for each group:

		Reinforcement (R)	
		Yes	No
Money (M)	Yes	ARM: 459.84	AM: 465.61
	No	ARV: 475.49	A: 501.47

Appendix B: Main Study Instructions

Appendix B1

Participant recruitment:

Below are the steps followed to recruit participants for the present experiment. Participants were recruited through the "Cognitive Division Subject Pool."

1/ Permission was obtained from instructors in various departments on campus to make an announcement in their class that several psychology experiments were being conducted and that participants were needed.

2/ Students were told that:

(a) studies are being conducted in the PAS building
(b) participants are paid about $6.00/hour
(c) please sign-up on sheets that are being passed around the class, if interested
(d) potential participants would be called and told the details of the study
-at that time individuals can decide to take part or decline

Appendix B2

Familiar setting - phone script:

Hello, _____ I'm Lisa Mulvihill and I am phoning from the University of Waterloo, Dept. of Psychology. You expressed an interest in participating in psychology experiments. I'm calling to tell you a bit more about the research we are doing.

In our lab we are measuring the effects of alcohol on a computerized task that requires responding to visual information on a computer screen. The experiment involves attending 2 sessions, each on a different day, and pays $15.00. The first session takes about 30 minutes and just involves getting familiar with the computer task and the lab. During the second session you will receive alcohol in the form of a mixed drink and perform the task. The second session takes about 2 hours. We are selecting individuals whose body weights fall between a range of 130-200 pounds (50-90 kg) and are at least 19 years of age. Are you interested in participating? Have you ever participated in an alcohol study before or a study that involved any other drugs such as caffeine? What did you do in that study? What was the task?

Although the dose of alcohol used in this experiment is not harmful, alcohol may have some physical effects. Thus, it is important that you do not have any medical problems such as diabetes or epilepsy. Similarly, it is important that you are not taking any medication: this includes regular use of cold or allergy medications, aspirin or antihistamines, or over-the counter drugs such as "wake-up" pills.

During the second session, when you receive alcohol, a breathalyser machine will measure your breath samples in order to estimate your blood alcohol concentration at different times. We use moderate doses of alcohol, which will not make you sick. However, you must not drive after completing the experiment. If you need transportation home it will be provided for you. After the experiment you are advised to remain in the lab until your blood alcohol level returns to a safe level.

Finally it is important that you abstain from drinking alcohol prior to the session when you get alcohol. In addition, you should not eat any food during the 4 hours before the session and abstain from fluids, apart from sips of water, for 2 hours. Your stomach should be empty. Do you have any questions?

Appendix B3

Novel setting - phone script:

Hello, _____ I'm Lisa Mulvihill and I am phoning from the University of Waterloo, Dept. of Psychology. You expressed an interest in participating in psychology experiments. I'm calling to tell you a bit more about the research we are doing.

In our lab we are measuring the effects of alcohol on a computerized task that requires responding to visual information on a computer screen. The experiment takes about 2.5 hours and pays $15.00. We are selecting individuals whose body weights fall between a range of 130-200 pounds (50-90 kg) and are at least 19 years of age. During this experiment you will receive alcohol in the form of a mixed drink. Are you interested in participating? Have you ever participated in an alcohol study before or a study that involved any other drugs such as caffeine? What did you do in that study? What was the task?

Although the dose of alcohol used in this experiment is not harmful, alcohol may have some physical effects. Thus, it is important that you do not have any medical problems such as diabetes or epilepsy. Similarly, it is important that you are not taking any medication: this includes regular use of cold or allergy medications, aspirin or antihistamines, or over-the counter drugs such as "wake-up" pills.

A breathalyser machine will measure your breath samples in order to estimate your blood alcohol concentration at different times. We use moderate doses of alcohol, which will not make you sick. However, you must not drive after completing the experiment. If you need transportation home it will be provided for you. After the experiment you are advised to remain in the lab until your blood alcohol level returns to a safe level.

(ask individual to get a pen and paper in order to write the following information down)

It is also important that you abstain from drinking alcohol for 24 hours before the experiment. In addition, you should not eat any food during the 4 hours before the experiment and abstain from fluids, apart from sips of water, for 2 hours before the study. Your stomach should be empty. Do you have any questions?

I'd like to tell you a little bit about the 4 hour fasting. Your last meal prior to fasting should be a light one. In general, avoid all dairy products and all greasy, fried foods (anything with butter)[6]. Do not drink milk or eat any milk products such as yogurt and do not eat fatty, greasy, or fried foods and avoid fats such as butter, mayonnaise, and peanut butter. Then after your light meal, you eat nothing, that is you fast for 4 hours.

Do you have any questions?

---

[6] Items read from menu (shown below).

Appendix B3 (cont.)

Eat a light meal followed by 4 hours of fasting before you come in for your session. Below is a list of suggested foods and a list of foods to avoid. In general, avoid all dairy products and all greasy, fried foods (e.g. anything with butter). Thank you for your cooperation.

Suggested foods:

- breads, buns, muffins
- fruits, vegetables
- seafood (nothing packed in oil)
- meat or poultry (broiled, baked or barbecued)
- hard or soft boiled eggs

- toast with jam (no butter)
- salad (no dressing)
- sandwiches (luncheon meats, with mustard only)
- soup (not creamed)
- pickles

Foods to avoid:

- all dairy products (e.g., cheese, butter, yogurt, ice-cream, margarine or milk)
- mayonnaise
- fried eggs

- fried hamburgers
- french fries, chips
- bacon
- donuts
- peanut butter

Appendix B4

Familiar setting - explanation of the requirements of the study:

First I'd like to thank you for volunteering to participate in this study. I hope that you will find it to be an interesting experience.

To ensure that everyone has the same understanding about the experiment I am going to read some information to you. While this is formal, it just ensures that everyone has the same understanding about the experiment.

The total time required of you will be about 2.5 hours. Today's session will take about 30 min to complete and will involve practising the task and getting familiar with the lab. During the second session you'll receive alcohol and perform the task. This session will take about 2 hours.

The purpose of this study is to examine the effects of alcohol on the performance of a computerized task. The payment for taking part in this study is $15 which you will receive at the end of the second session. Please remember that you have to come to both sessions to be paid: THERE IS NO PARTIAL PAYMENT.

Timing is very important in this study. You will be asked to perform the task at certain times and drink each of the drinks within a certain time period. You cooperation with the time schedule is very important.

As I told you on the phone, there are some instructions regarding fasting for the second session (i.e., no food for 4 hours and no alcohol for 24 hours). I'll give you more details about is at the end of the session today.

Do you have any questions?

Appendix B5

Novel setting - explanation of the requirements of the study:

First I'd like to thank you for volunteering to participate in this study. I hope that you will find it to be an interesting experience.

To ensure that everyone has the same understanding about the experiment I am going to read some information to you. While this is formal, it just ensures that everyone has the same understanding about the experiment.

The total time required of you will be about 2.5 hours. During the study, you will receive alcohol and perform the task at certain times.

The purpose of this study is to examine the effects of alcohol on the performance of a computerized task. The payment for taking part in this study is $15 which you will receive at the end of the study today.

Timing is very important in this study. You will be asked to perform the task at certain times and drink each of the drinks within a certain time period. Your cooperation with the time schedule is very important.

Do you have any questions?

Appendix B6

Task instructions ABCD task (3-minute familiarization test):

PARTICIPANT SEATED IN FRONT OF COMPUTER SCREEN

While you are performing the task you will sit in front of the computer screen, just as you are doing. You are to place your index finger on this key (indicate key with > and .) and your next finger goes on this key (indicate key with ? and /). You may choose which hand you prefer to use but you must be consistent on all trials.

Presented on the screen will be the letters A,B,C and D, only one of which will appear at a time. If the letter is an A or C, you are to press this left key as quickly as possible (indicate). If a B or D appears, you are to press the right key as quickly as possible (indicate).

Now, before each trial a number sign will appear in the middle of the screen (indicate number sign on keyboard). It serves as a fixation point so that you know where to focus your attention on the computer screen. After the number sign disappears one of the 4 letters (A,B,C,D) will appear on the screen. When you see the letter, respond as quickly as possible by pressing the appropriate key.

You will occasionally hear a tone. This tone means that you are NOT to respond. That is, do not press either key regardless of which letter is displayed. DO NOT wait for the tone as it occurs infrequently and at various time intervals so you might only be able to stop on some occasions when you hear a tone. That is normal, just keep doing your best.

The time to complete this block of trials is about 3 minutes and it serves to familiarize you with the task. I'll stay in the room for this trial to ensure that everything is working ok. So just ignore me and do not talk while you are performing the task. Please ask me any questions about the task when you are done.

Any questions? Ok, I will start the task. So remember to respond as quickly and accurately as possible, do your best to stop when you hear the tone, but DO NOT slow down or wait in anticipation that a tone might occur.

AFTER TRIAL

That was fine, you have the hang of it. Do you have any questions about the task?

Appendix B7

Familiar setting - instructions for drug-free experience test
(give after familiarization test):

Now you will perform a longer trial which will take about 10 minutes. Half-way through the trial you will have a 30 second rest. The computer times this break and will prompt you when it is time to begin the second half of the trial. I'm going the leave you alone to perform this trial and I'll be back as soon as you are done.

Remember that it is important that you respond as quickly and accurately as possible, do your best to stop when you hear the tone, but DO NOT slow down or wait in anticipation that a tone might occur.

Any questions? When I have shut the door start the task by pressing the "y" key.

Appendix B8

Familiar setting - study reminders:

I have a few things to tell you about the next session. First of all, as I said before, it is important that you eat breakfast or a light lunch 4 hours before the start of the second session. For example, if you are coming for a session at 6pm, you would have lunch or a snack at 2pm. There are 2 restrictions on what you eat in that meal. One--do no have milk of milk products such as yogurt or cheese. Two--do no have fatty or greasy food such as fried eggs, bacon, french fries, or fats such as butter, mayonnaise or peanut butter. Here is a menu that specifies what you may eat which you can take home as a reminder.

It is important that you DO eat something light; but after this meal, please remember not to eat or drink anything apart from sips of water, for the rest of the four hours prior to the start of the drinking session. Also, as I already mentioned, please don't take any drugs (such as alcohol, aspirin or antihistamines) for 24 hours before the second session.

At the conclusion of the second session, your blood alcohol level may be above zero, so for safety we caution you against driving. You should make alternative arrangements, and if you have any difficulties in this respect, we can arrange a ride for you. Any questions?

Menu:

Eat a light meal followed by 4 hours of fasting before you come in for the next session. Below is a list of suggested foods and a list of foods to avoid. In general, avoid all dairy products and all greasy, fried foods (e.g. anything with butter). Thank you for your cooperation.

Suggested foods:

- breads, buns, muffins
- fruits, vegetables
- seafood (nothing packed in oil)
- meat or poultry (broiled, baked or barbecued)
- hard or soft boiled eggs

- toast with jam (no butter)
- salad (no dressing)
- sandwiches (luncheon meats, with mustard only)
- soup (not creamed)
- pickles

Foods to avoid:

- all dairy products (e.g., cheese, butter, yogurt, ice-cream, margarine or milk)
- mayonnaise
- fried eggs

- fried hamburgers
- french fries, chips
- bacon
- donuts
- peanut butter

Appendix B9

Check for compliance with fasting requirements:

Familiar setting:

QUESTIONS BEFORE STARTING THE STUDY
Before we get started I have some questions.
When did you last eat?
What did you have?
Did you have any trouble meeting any of the other requirements?
- any medication whatsoever? (even aspirin?)
- over-the-counter drugs?
- any alcohol in the last 24 hours?

During this session you will receive alcohol and perform the task at different times. This session takes about 2 hours.

Please come over here and stand on the scale so we can check your weight. By using a person's weight, we can ensure that all participants received the same standard dose.

Throughout this session I will be asking you to provide breath samples to measure you BAC. Now we'll do a practice breath sample so that you are familiar with the machine. Just blow a nice even flow of air into this mouthpiece for about 10 seconds (I will tell you when to stop). Watch this light, you must be blowing hard enough to turn it on.

Novel Setting:

QUESTIONS BEFORE STARTING THE STUDY:
Before we get started I have some questions.
When did you last eat?
What did you have?
Did you have any trouble meeting any of the other requirements?
- any medication whatsoever? (even aspirin?)
- over-the-counter drugs?
- any alcohol in the last 24 hours?

Please come over here and stand on the scale so we can check your weight. By using a person's body weight, we can ensure that all participants received the same standard dose.

Throughout this session I will be asking you to provide breath samples to measure your BAC. Now we'll do a practice breath sample so that you are familiar with the machine. Just blow a nice even flow of air into this mouthpiece for about 10 seconds (I will tell you when to stop). Watch this light, you must be blowing hard enough to turn it on.

Appendix B10

Familiar setting - baseline task instructions:

All groups in the familiar setting (F-AR, F-AM & F-A) received the following instructions as a reminder of task instructions:

The requirements of the task are the same as they were in the first session. I'm just going to take a minute to review the task with you. You place your index finger on this key and your next finger goes on this key. Letters A,B,C and D will appear one at a time. If the letter is an A or C, you press this left key as quickly as possible. If a B or D appears, you press the right key as quickly as possible.

Occasionally you will hear a tone. This tone means that you are NOT to respond. That is, DO NOT press either key regardless of what letter is displayed.

The trials today are 10 minutes trials. Remember that half-way through the trial you will have a 30 sec rest break. The computer times this break and will prompt you when it is time to begin the second half of the trial. I'm going to leave you alone to perform the trial and I'll be back as soon as you are done.

Remember that it is important that you respond as quickly and accurately as possible, do your best to stop when you hear the tone, but DO NOT slow down or wait in anticipation that a tone might occur. Any questions? When I shut the door, just press the "y" key to begin the task.

Appendix B11

Novel setting - baseline task instructions:

All groups in the novel setting (N-AR, N-AM & N-A) received the following instructions:

Now you will perform a longer trial which will take about 10 minutes. Half-way through the trial you will have a 30 second rest. The computer times this break and will prompt you when it is time to begin the second half of the trial. I'm going the leave you alone to perform this trial and I'll be back as soon as you are done.

Remember that it is important that you respond as quickly and accurately as possible, do your best to stop when you hear the tone, but DO NOT slow down or wait in anticipation that a tone might occur.

Any questions?

Appendix B12

Introduction of treatments on the baseline test:

Participants in the AR and AM treatment groups received the baseline task instructions (familiar groups Appendix B10; novel groups Appendix B11). In addition, they received the following:

Reinforcement Treatment: (F-AR and N-AR):

Now I'll be able to tell you how you are doing on the task.

Familiar setting: Your performance will be compared to how you did on the last 10 minute trial you did last session.

Novel setting: Your performance will be compared to your performance on the trial you just completed.

If you can withhold the same or a greater percentage of responses when tones sound I will tell you "YES". If you do not withhold as many or more responses when tones sound I will tell you "NO". To help you keep track of this, put a Y on this sheet when I say "YES" and an N when I say "NO". Any questions?

In order for these trials to count you have to maintain your speed of responses to the letters. If you respond more slowly to the letters I will say that the trial "CANNOT BE COUNTED". To help you keep track of this, put a 0 on this sheet when I say CBC.

As soon as the trial ends I will come into the room and check your performance on the computer and give you feedback. I will not be in the room while you perform the task. Any questions? When I have shut the door start the task by pressing the "y" key.

Monetary Incentive Treatment (F-AM and N-AM):

Now you have a chance to win 25 cent bonus. In order to win the bonus you have to withhold the same or a greater percentage of responses when tones sound ...

[Familiar setting:] ...as you did on the last 10 minute trial you did last session.

[Novel setting:] ...as you did on the trial you just completed.

I'll keep a record of your performance on each trial so I can keep track of the money you have earned. I can only show you this record at the end of the study and you will receive your bonus money at that time. Any questions?

In order for these trials to count you have to maintain your speed of responses to the letters. If you respond more slowly to the letters I will record that the trial "CANNOT BE COUNTED", meaning that you cannot win a bonus for that trial.

As soon as the trial ends I will come into the room (and check your performance on the computer). I will not be in the room while you perform the task. Any questions? When I have shut the door start the task by pressing the "y" key.

Placebo and Alcohol Treatments (N-P, F-P, N-A, F-A):

Groups F-A and F-P - received reminder of task instructions: see appendix B10

Groups N-A and N-P - received reminder of task instructions: as in appendix B11

Appendix B13

Reminder of treatments under alcohol or placebo:

Reinforcement Treatment: (F-AR and N-AR):

Now you are going to do a number of 10 min trials, separated by the 30 sec rest. And I will still be able to tell you how you are doing on the task. Your performance from now on will always be compared to how you did on the last trial you did right BEFORE you started drinking.

Remember, if you can withhold as many or more responses when tones sound I will tell you "YES". If you do not withhold as many responses when tones sound I will tell you "NO". You can continue to keep track of this by putting a "Y" on this sheet when I say YES and a "N" when I say "NO" (pull the sheet over beside the participant).

In order for these trials to count you have to maintain your speed of response the letters. If you respond more slowly then I will say that the trial "CANNOT BE COUNTED". You can continue to keep track of this by putting a 0 on this sheet when I say CBC.

Remember your performance from now on will always be compared to how you did on the last trial you did right BEFORE you started drinking. As soon as the trial ends I will come into the room and check your performance on the computer and give you feedback. I will not be in the room while you perform the task.

Any questions? When I have shut the door press the "y" key to begin the task.

Monetary Incentive Treatment (F-AM and N-AM):

Now you are going to do a number of 10 min trials, separated by the 30 sec rest. And you still have a chance to win bonuses. In order to win the bonus you have to withhold as many or more responses when tones sound as you did on the last trial you did right BEFORE you started drinking.

In order for these trials to count you have to maintain your speed of response the letters. If you respond more slowly then I will record that the trial "CANNOT BE COUNTED", meaning that you cannot win a bonus for that trial.

I will continue to keep a record of you performance so I can keep track of the money you have earned. I can only show you this record give you your bonus money at the end of the study.

Remember your performance from now on will always be compared to how you did on the last trial you did right BEFORE you started drinking. As soon as the trial ends I will come into the room and check your performance on the computer. I will not be in the room while you perform the task.

Any questions? When I have shut the door press the "y" key to begin the task.

Appendix B13 (cont.)

Placebo and Alcohol Treatments (F-P, N-P, F-A, N-A):

Now you are going to do a number of trials. The trials will continue to consist of 2 blocks separated by a 30-second rest period and will take about 10 minutes to complete. I will not be in the room while you perform the task but I will be back when you are done.

Remember that it is important that you respond as quickly and accurately as possible, do your best to stop when you hear the tone, but DO NOT slow down or wait in anticipation that a tone might occur.

Any questions? When I have shut the door press the "y" key to begin the task.

Appendix B14

Debriefing:

As mentioned before, we require that you remain in the lab area until your blood alcohol concentration falls to as safe level of .03%. Your blood alcohol concentration at this time is _____. You drank the equivalent of _____ bottles of beer (dose divided by 17.04). We remind you not to operate any machinery for the next two hours. Also you must not drive home (this includes riding a bike). Are you planning to remain on campus? IF NOT: How are you planning to get home? (Warn about having another drink, and the typical rate of decline of BAC).

Would you like a coffee, tea or soft drink? Or some cookies to eat?

We are interested in how university students respond to information that is presented visually by computers. We are collecting data from a large number of students that will be used to provide a representative normal sample of respondents so that we can examine different within this group. In particular, we are looking at the accuracy and speed with which people react to information. Drugs like alcohol may affect responses to information in different ways. Alcohol is a depressant drug and may impair the ability to respond accurately and quickly. To examine its effects, we administered a mild/moderate amount of alcohol to test a participant's performance. To understand how alcohol affects performance we compare a participant's performance under alcohol, to his performance drug-free. Any differences between these conditions in responses will help us understand exactly how alcohol affects information processing.

We were also interested in whether feedback or incentive (information/money), or level of familiarity with the drinking setting would have any effect on performance under alcohol.

Do you have any questions? Anything unclear?

Appendix C: Main Study Experimental Materials

Appendix C1

Personal History Drinking Questionnaire (PDHQ):

#_____

Below are some questions which are primarily concerned with your personal drinking. Most ask you to answer according to what is most typical or usual for you. Please try to answer each question as honestly as possible.

1) Please estimate the number of years that you have been drinking alcohol. Estimate to the nearest month.

_____years _____months

2) How often, on average, do you drink alcohol? (Choose only **one**)

A) Only on special occasions, how many times per year? _____
B) Monthly, how often? _____
C) Weekly, how often? _____
D) Daily, how often? _____

3) What alcoholic beverage do you drink? _____

4) In terms of the beverage indicated in question 3, what is the **AVERAGE** quantity you drink in a single drinking occasion? (Choose only **one**)

A) WINE (estimate ounces) 1 2 3 4 5 6 7 8 9 10 or ____
B) BEER (bottles) 1 2 3 4 5 6 7 8 9 10 or ____
C) BEER (draft glasses) 1 2 3 4 5 6 7 8 9 10 or ____
D) LIQUOR (assume 1.5 ounces per drink and estimate the number of drinks) 1 2 3 4 5 6 7 8 9 10 or ____

5) How long does your typical drinking occasion last? (Choose only **one**)
A) _____ MINUTES
B) _____ HOURS
C) _____ DAYS

6) Have you ever been charged with impaired driving? YES NO
7) Have you ever experienced any problems related to your drinking? YES NO
8) Age ____ Weight _____ Height _____ Handedness: RIGHT LEFT

Appendix C2

Beverage rating scale:

#_____

Regarding the alcohol you have consumed, rate the strength of its effect by comparing it to bottles of beer (5% alcohol by volume) **OR** fluid ounces of liquor (40% alcohol by volume). ONE STANDARD DRINK CONTAINS 1.5 OUNCES OF ALCOHOL.

BOTTLES OF BEER (5%)	**OR**	OUNCES OF LIQUOR (40%)
Circle the total number of BOTTLES		Circle the total number of OUNCES
0.0		0.0
0.5		0.5
1.0		1.0
1.5		1.5
2.0		2.0
2.5		2.5
3.0		3.0
3.5		3.5
4.0		4.0
4.5		4.5
5.0		5.0
5.5		5.5
6.0		6.0
6.5		6.5
7.0		7.0
7.5		7.5
8.0		8.0
8.5		8.5
9.0		9.0
9.5		9.5
10.0		10.0

Appendix C3

Desire to resist drug effect scale:

#_____

On this scale, ranging from zero (NOT AT ALL) to ten (EXTREMELY), circle the number which best indicates the degree to which you tried to resist the effect of the alcohol on this task.

0	1	2	3	4	5	6	7	8	9	10
NOT AT ALL										EXTREMELY

Appendix C4

Familiar setting - consent form:

I, _____, age _____ hereby state that I have volunteered to consume a moderate dose of alcohol and to perform trials on a computer task. The purpose of this study is to examine the effect that alcohol has on the ability to respond to information on a computerized task. I understand that I will become familiar with the task during a 30 minute session. I understand that I will then attend a second session which will take about 2 hours, where I will perform the task under a moderate dose of alcohol. I understand that my total participation time will be about 2.5 hours.

I am not currently taking any medication. I will abstain from alcohol for at least 24 hours and fast for 4 hours prior to the alcohol session to ensure that stomach contents do not affect the absorption of alcohol. I also understand that at the conclusion of the second session, my blood alcohol level may be above zero and I am advised to remain in the lab until it returns to a safe level of .03%.

I understand that all records, tests and personal data are confidential, and will be used in research reports that do not disclose the identity of any individual.

I consent to what is proposed to be done. I agree of my own free will to participate in this experiment. The Consent is given freely and I understand that I am free to withdraw from the experiment at any time for any reason.

I understand that I shall receive a remuneration of $15 for taking part in this study.

This research is being conducted by Lisa Mulvihill under the supervision of the Principal investigator, Dr. M. Vogel-Sprott, who may be reached at the Department of Psychology, ext. 2666. This project has been reviewed and received ethics clearance through the Office of Human Research. If you have any questions or concerns about your participation, please call this office at 885-1211, extension 6005.

Signed this _____ day of _____, 19___.

_____
Participant's Name

_____
Participant's Signature

_____
Witness

Appendix C5

Novel setting - consent form:

I, _____, age _____ hereby state that I have volunteered to consume a moderate dose of alcohol and to perform trials on a computer task. The purpose of this study is to examine the effect that alcohol has on the ability to respond to information on a computerized task. I understand that I will become familiar with the task and then perform the task under a moderate dose of alcohol. I understand that my participation time will be about 2.5 hours.

I am not currently taking any medication. I have abstained from alcohol for at least 24 hours and have fasted for 4 hours prior to this study to ensure that stomach contents do not affect the absorption of alcohol. I also understand that at the conclusion of the study, my blood alcohol level may be above zero and I am advised to remain in the lab until it returns to a safe level of .03%.

I understand that all records, tests and personal data are confidential, and will be used in research reports that do not disclose the identity of any individual.

I consent to what is proposed to be done. I agree of my own free will to participate in this experiment. The Consent is given freely and I understand that I am free to withdraw from the experiment at any time for any reason.

I understand that I shall receive a remuneration of $15 for taking part in this study.

This research is being conducted by Lisa Mulvihill under the supervision of the Principal investigator, Dr. M. Vogel-Sprott, who may be reached at the Department of Psychology, ext. 2666. This project has been reviewed and received ethics clearance through the Office of Human Research. If you have any questions or concerns about your participation, please call this office at 885-1211, extension 6005.

Signed this _____ day of _____, 19___.

_____

Participant's Name

_____

Participant's Signature

_____

Witness

Appendix C6

Information for participants:

Despite the wide variety of alcohol beverages, all are composed of ethyl alcohol and water. Because alcohol is already liquid, it does not have to dissolve in the stomach as does a drug in a tablet form. Thus it is rapidly and completely absorbed by simple diffusion across membranes. The rate of absorption is both determined by the amount of food in the gastro-intestinal tract and the nature of the beverage consumed.

In general, the more concentrated the alcohol is the more rapid its absorption, i.e., diluted alcoholic beverages (such as beer) are absorbed more slowly than are concentrated drinks (such as cocktails). Food in the stomach retards the absorption, firstly because it will dilute the concentration of the alcohol and secondly it covers some of the stomach membranes through which alcohol is absorbed. Also, a full stomach will prolong emptying time. Thus blood alcohol levels will rise faster for an individual who has fasted than for a person who has just eaten a large meal. However, the alcohol will still be completely absorbed except that for the person who has eaten, it will be somewhat delayed.

Elimination of alcohol from the organism (e.g. via lungs, liver, and kidneys) is a gradual process. In humans, elimination proceeds in a linear fashion at the rate of approximately 10 ml. of absolute alcohol per hour (about an ounce of liquor). Thus the slope of the blood alcohol curve during the absorption phase, commonly referred to as the ascending limb, is steeper than the slope of the elimination phase (descending limb). Considerable evidence is available which suggests that the effects of alcohol are quite different under ascending as opposed to descending BACs.

BLOOD ALCOHOL CONCENTRATION (BAC)

The following effects of alcohol occur because of its action upon the brain. Alcohol's effects are fairly predictable from the amount in the bloodstream. Therefore, if you know a person's BAC you can roughly predict what effects alcohol will be having upon him or her. Some examples:

- At 20 mg% (.02 BAC) light and moderate drinkers begin to feel some effects. This is the approximate BAC reached after one drink.
- At 40 mg% (.04 BAC) most people begin to feel relaxed.
- At 60 mg% (.06 BAC) judgement is somewhat impaired; people are less able to make rational decisions about their capabilities (e.g., to drive).
- At 80 mg% (.08 BAC) there is a definite impairment of muscle coordination and driving skills; legally impaired in Ontario.
- At 100 mg% (.10 BAC) there is clear deterioration of reaction time and control; legally impaired in most of the United States.
- At 120 mg% (.12 BAC) vomiting occurs unless this level is reached slowly.
- At 150 mg% (.15 BAC) balance and movement are impaired. This BAC level means that the equivalent of one-half pint of whisky is circulating in the bloodstream.
- At 300 mg% (.30 BAC) many people lose consciousness.
- At 400 mg% (.40 BAC) most people lose consciousness, some die.
- At 450 mg% (.45 BAC) breathing stops, death occurs.

From: Miller, W.R. & Munoz, R.F. (1976) *How to control your drinking*, Prentice-Hall, Inc.

Appendix D: Main Study Procedural Checks

Appendix D1

One-way ANOVAs on drinking habit measures of eight groups:

Frequency:

Source	df	MS	F	p
Group	7	0.45	0.49	.840
Error	64	0.91		

Dose:

Source	df	MS	F	p
Group	7	0.54	1.19	.323
Error	64	0.46		

Duration:

Source	df	MS	F	p
Group	7	0.53	0.21	.982
Error	63	2.54		

Appendix D2

Drug-free baseline measures:

Table 1a. ANOVA on the number of inhibitions during the pre-treatment baseline test of groups to receive one of four treatments in the novel or familiar setting:

Source	df	MS	F	p
Setting(S)	1	136.13	1.65	.203
Treatment (T)	3	122.61	1.49	.226
S X T	3	7.90	0.10	.962
Error	64	82.44		

Table 1b. Mean (SD) inhibitions on the baseline test for each group:

	Treatment Groups			
	Immediate Reinforcement	Monetary Incentive	Alcohol	Placebo
Setting: Novel	N-AR: 20.89 (8.3)	N-AM:19.67 (11.9)	N-A: 25.89 (8.7)	N-P: 20.56 (7.7)
Familiar	F-AR: 22.44 (10.7)	F-AM: 22.56 (8.0)	F-A: 27.89 (7.1)	F-P: 25.11 (9.3)

Appendix D2 (cont.)

Table 2a. ANOVA on the mean RT (msec) during the pre-treatment baseline test of groups to receive one of four treatments in the novel or familiar setting:

Source	df	MS	F	p
Setting(S)	1	8178.57	1.91	.172
Treatment (T)	3	7804.99	1.82	.152
S X T	3	4099.47	0.96	.418
Error	64	4279.92		

Table 2b. Mean (SD) RT (msec) on the baseline test for each group:

		Treatment Groups			
		Immediate Reinforcement	Monetary Incentive	Alcohol	Placebo
Setting	Novel	N-AR: 441.76 (42.14)	N-AM: 431.34 (73.69)	N-A: 486.43 (95.58)	N-P: 419.35 (38.01)
	Familiar	F-AR: 423.38 (54.17)	F-AM: 392.90 (53.92)	F-A: 438.07 (52.31)	F-P: 439.26 (88.74)

Appendix D3

BAC Measures:

Table 1a. ANOVA of the BACs of six groups at six time intervals:

Source	df	MS	F	p
Group (G)	5	338.60	0.63	.675
Error	47	534.84		
Time	5	4453.71	42.82	<.001
Time X G	25	93.28	0.90	.610
Error	235	104.01		

Table 1b. The BACs of six groups at six time intervals:

Minutes After Drinking	26	56	72	86	106	120
Mean BAC (mg/100 ml)	52	74	74	71	65	58
SD	19	15	11	11	10	9

Appendix E: Descriptive statistics and supplementary analyses of inhibitions in A & P groups in N & F settings

Appendix E1

Mean change in number of inhibitions on each treatment test for each group in each setting:

Groups	Treatment Tests: Test 1	Test 2	Test 3	Test 4
N-A	-1.89 (6.9)	-0.89 (8.1)	+2.22 (4.7)	+1.33 (5.6)
N-P	0.00 (4.7)	+0.89 (5.1)	+1.00 (5.1)	+1.11 (5.6)
F-A	-4.33 (4.4)	-4.11 (3.6)	-4.56 (5.5)	-2.22 (4.4)
F-P	-1.00 (4.8)	-2.33 (7.0)	-0.78 (6.1)	+0.22 (7.7)

Overall mean (SD) change in inhibitions for each treatment and setting:

Setting	Treatment Groups: Alcohol	Placebo
Novel	N-A: +0.19 (5.5)	N-P: +0.75 (4.5)
Familiar	F-A: -3.81 (3.3)	F-P: -0.97 (5.7)

Appendix E2

The test of interactions among pre-treatment baseline number of inhibitions and between-subjects factors:

Source	df	MS	F	p
Setting (S)	1	27.59	1.07	.310
Treatment (T)	1	28.23	1.10	.304
S X T	1	18.61	0.72	.403
Covariate:				
Baseline (B)	1	1921.75	74.52	<.001
B X S	1	8.39	0.33	.573
B X T	1	16.63	0.65	.429
B X S X T	1	8.69	0.34	.566
Error	28	25.79		

ANCOVA of the number of inhibitions as a function of setting and treatment using the pre-treatment baseline inhibition score as a covariate:

Source	df	MS	F	p
Setting (S)	1	61.35	2.53	.122
Treatment (T)	1	17.86	0.74	.397
S X T	1	13.18	0.54	.466
Covariate (Baseline)	1	1928.15	79.61	<.001
Error	31	24.22		

Appendix E2 (cont.)

Adjusted mean number of inhibitions for each group:

Setting	Treatment Groups: Alcohol	Placebo
Novel	N-A: 25.12	N-P: 25.36
Familiar	F-A: 21.23	F-P: 23.90

Group comparisons of adjusted group means testing a priori hypotheses:

N-A vs F-A:

Source	df	MS	F	p (one-tailed)
Hypothesis	1	67.27	2.78	.053
Error	31	24.22		

N-P vs F-P:

Source	df	MS	F	p
Hypothesis	1	9.12	0.38	.544
Error	31	24.22		

F-A vs N-P & F-P combined:

Source	df	MS	F	p (one-tailed)
Hypothesis	1	64.68	2.67	.056
Error	31	24.22		

Appendix E3

Pre-treatment baseline mean (SD) number of inhibitions (Pre) and the mean (SD) number of inhibitions averaged across the four treatment tests (Post) for each group:

		Treatment Groups			
		Alcohol		Placebo	
Setting	Novel	**N-A:**		**N-P:**	
		Pre	**Post**	**Pre**	**Post**
		25.89 (8.7)	26.08 (9.7)	20.56 (7.7)	21.31 (8.0)
	Familiar	**F-A:**		**F-P:**	
		Pre	**Post**	**Pre**	**Post**
		27.89 (7.1)	24.08 (9.0)	25.11 (9.3)	24.14 (9.8)

Appendix E4

ANOVA of the change in inhibitions based on four tests under alcohol or placebo using the data from the three shortest stop-signal delays:

Source	df	MS	F	p
Setting (S)	1	264.06	3.39	.075
Treatment (T)	1	154.17	1.98	.169
S X T	1	47.84	0.61	.439
Error	32	77.98		
Tests (Tt)	3	17.73	1.80	.153
Tt X S	3	8.66	0.88	.455
Tt X T	3	3.73	0.38	.769
Tt X S X T	3	9.47	0.96	.415
Error	96	9.86		

Appendix E4 (cont.)

Group comparisons testing a priori hypotheses:

N-A vs F-A:

Source	df	MS	F	p (one-tailed)
Hypothesis	1	67.09	3.44	.037
Error	32	19.50		

N-P vs F-P:

Source	df	MS	F	p
Hypothesis	1	10.90	0.56	.460
Error	32	19.50		

F-A vs N-P & F-P combined:

Source	df	MS	F	p (one-tailed)
Hypothesis	1	96.00	4.92	.017
Error	32	19.50		

Appendix E4 (cont.)

ANCOVA of the number of inhibitions under alcohol or placebo using the data from the three shortest stop-signal delays:

Source	df	MS	F	p
Setting (S)	1	51.21	2.59	.118
Treatment (T)	1	25.93	1.31	.261
S X T	1	13.28	0.67	.419
Covariate (Baseline)	1	1209.17	61.20	<.001
Error	31	19.76		

Adjusted mean number of inhibitions for each group:

		Treatment Groups	
		Alcohol	Placebo
Setting	Novel	N-A: 23.17	N-P: 23.71
	Familiar	F-A: 19.50	F-P: 22.48

Appendix E4 (cont.)

Group comparisons of adjusted means to test a priori hypotheses:

N-A vs F-A:

Source	df	MS	F	p (one-tailed)
Hypothesis	1	59.53	3.01	.047
Error	31	19.76		

N-P vs F-P:

Source	df	MS	F	p
Hypothesis	1	6.56	0.33	.569
Error	31	19.76		

F-A vs N-P & F-P combined:

Source	df	MS	F	p (one-tailed)
Hypothesis	1	71.21	3.60	.034
Error	31	19.76		

Appendix F: Descriptive statistics and supplementary analyses of RT and errors in A & P groups in N & F settings

Appendix F1

The main effect of treatment [$F(1,32)=3.95$, $p=.055$] on the mean change in RT during treatment tests for each group, is shown in the table below. A negative change in RT indicates that participants tended to speed up their responses to go-signals, whereas a positive change indicates that participants slowed down. The table shows that participants who received alcohol, (groups N-A and F-A) slowed their response to go-signals, whereas those who received the placebo treatment tended to respond more quickly.

It is important to understand the impact of these changes in RT during treatment because participants could delay their response to go-signals in order to increase their number of inhibitions. Although the placebo groups (N-P and F-P) tended to respond more quickly during treatment, they were able to maintain their pre-treatment (i.e., drug-free baseline) level of inhibitions. Although participants in the alcohol treatment groups (N-A and F-A) tended to slow their response to go-signals, the change in response inhibitions of these groups was significantly different. Group F-A showed a *decrease* in their number of inhibitions despite the fact that they slowed down whereas inhibitions in group N-A showed little change. Thus, the change in RT to go-signals across treatments bore no systematic relationship to the change in inhibitions.

Overall mean (SD) change in RT (msec) for each group:

	Treatment Groups	
Setting	Alcohol	Placebo
Novel	N-A:+17.4 (93.47)	N-P:-17.44 (14.68)
Familiar	F-A:+12.53 (32.02)	F-P:-20.23 (21.32)

Appendix F1 (cont.)

Mean (SD) change in RT (msec) for each group at each treatment test:

Groups	Treatment Tests			
	Test 1	Test 2	Test 3	Test 4
N-A	+4.99 (82.20)	+43.22 (147.15)	+25.64 (102.6)	-3.98 (54.89)
N-P	-18.82 (13.3)	-19.39 (13.79)	-17.22 (24.83)	-15.35 (14.37)
F-A	+8.92 (22.62)	+11.29 (33.02)	+13.64 (41.1)	+16.28 (40.67)
F-P	-26.10 (26.54)	-22.45 (24.85)	-20.21 (20.64)	-12.16 (26.50)

Appendix F2

Test of interactions among pre-treatment baseline mean RT (msec) and between-subjects factors:

Source	df	MS	F	p
Setting (S)	1	1.97	0.001	.979
Treatment (T)	1	219.51	0.08	.782
S X T	1	2904.60	1.03	.318
Covariate:				
Baseline (B)	1	116536.33	41.49	<.001
B X S	1	10.86	0.004	.951
B X T	1	684.39	0.24	.625
B X S X T	1	2903.26	1.03	.318
Error	28	2809.01		

ANCOVA of the RT (msec) as a function of setting and treatment using the pre-treatment baseline RT as a covariate:

Source	df	MS	F	p
Setting (S)	1	140.99	0.05	.820
Treatment (T)	1	9899.05	3.68	.064
S X T	1	15.55	0.01	.940
Covariate				
(Baseline)	1	166747.46	61.95	<.001
Error	31	2691.75		

Appendix F2 (cont.)

Adjusted mean RT (msec) for each group:

		Treatment Groups	
		Alcohol	Placebo
Setting	Novel	N-A: 463.58	N-P: 428.12
	Familiar	F-A: 458.25	F-P: 425.49

Appendix F3

Mean RT (msec):

Pre-treatment baseline mean (SD) RT (**Pre**) and mean (SD) RT averaged across the 4 treatment tests (**Post**) for each group:

Treatment Groups

Setting	Alcohol		Placebo	
Novel	**N-A:**		**N-P:**	
	Pre	**Post**	**Pre**	**Post**
	486.43	503.89	419.35	401.91
	(95.58)	(134.22)	(38.01)	(46.82)
Familiar	**F-A:**		**F-P:**	
	Pre	**Post**	**Pre**	**Post**
	438.07	450.60	439.26	419.03
	(52.31)	(74.60)	(88.74)	(74.18)

Appendix F4

Overall mean (SD) change in errors for each group in N & F settings:

	Treatment Groups	
	Alcohol	Placebo
Setting: Novel	N-A: +1.2 (3.7)	N-P: -1.8 (4.0)
Familiar	F-A: +2.8 (3.1)	F-P: -0.97 (3.8)

Pre-treatment baseline mean (SD) number of errors **(Pre)** and mean (SD) number of errors averaged across the 4 treatment tests **(Post)** for each group:

	Treatment Groups			
	Alcohol		Placebo	
Setting: Novel	**N-A:**		**N-P:**	
	Pre	**Post**	**Pre**	**Post**
	6.3	7.5	9.8	8.0
	(4.6)	(5.2)	(5.1)	(5.7)
Familiar	**F-A:**		**F-P:**	
	Pre	**Post**	**Pre**	**Post**
	4.1	6.9	8.4	7.5
	(2.6)	(3.1)	(6.6)	(4.7)

Appendix G: Descriptive stats and supplementary analyses of inhibitions in alcohol groups under three different treatments in N & F settings

Appendix G1

Overall mean (SD) change in inhibitions for each treatment group and setting:

Setting	Treatment Groups: Immediate Reinforcement	Monetary Incentive	Alcohol
Novel	N-AR: -0.19 (4.5)	N-AM: -0.67 (3.6)	N-A: +0.19 (5.5)
Familiar	F-AR: -1.5 (3.6)	F-AM: -5.44 (3.4)	F-A: -3.81 (3.3)

Mean (SD) change in number of inhibitions on each treatment test for each group in each setting:

Groups	Test 1	Test 2	Test 3	Test 4
N-AR	-1.22 (6.5)	+1.89 (6.5)	-0.67 (5.7)	-0.78 (3.3)
N-AM	-0.89 (5.3)	-0.67 (5.2)	-0.89 (3.7)	-0.22 (5.6)
N-A	-1.89 (6.9)	-0.89 (8.1)	+2.22 (4.7)	+1.33 (5.6)
F-AR	-1.33 (4.5)	-1.22 (3.7)	-1.11 (3.5)	-2.33 (5.2)
F-AM	-4.78 (4.3)	-5.67 (4.6)	-5.11 (3.4)	-6.22 (4.8)
F-A	-4.33 (4.4)	-4.11 (3.6)	-4.56 (5.5)	-2.22 (4.4)

Appendix G2

The test of interactions among pre-treatment baseline number of inhibitions and between-subjects factors:

Source	df	MS	F	p
Setting (S)	1	29.62	1.63	.209
Treatment (T)	2	7.71	0.42	.658
S X T	2	14.29	0.78	.463
Covariate:				
Baseline (B)	1	3710.48	203.54	<.001
B X S	1	1.05	0.06	.812
B X T	2	1.96	0.11	.898
B X S X T	2	10.03	0.55	.581
Error	42	18.23		

ANCOVA of the number of inhibitions as a function of setting and treatment using the pre-treatment baseline inhibition score as a covariate:

Source	df	MS	F	p
Setting (S)	1	150.18	8.93	.004
Treatment (T)	2	22.03	1.31	.279
S X T	2	14.93	0.89	.418
Covariate				
(Baseline)	1	4108.95	244.34	<.001
Error	47	16.82		

Appendix G2 (cont.)

Adjusted mean number of inhibitions for each group:

		Immediate Reinforcement	Monetary Incentive	Alcohol
Setting	Novel	N-AR: 23.03	N-AM: 22.56	N-A: 23.42
	Familiar	F-AR: 21.72	F-AM: 17.78	F-A: 19.42

Group comparisons of adjusted group means to test a priori hypotheses:

N-A vs N-AM:

Source	df	MS	F	p
Hypothesis	1	3.21	0.19	.664
Error	47	16.82		

N-AR vs N-A and N-AM combined:

Source	df	MS	F	p
Hypothesis	1	0.01	0.001	.981
Error	47	16.82		

F-AM vs F-A:

Source	df	MS	F	p
Hypothesis	1	11.74	0.70	.408
Error	47	16.82		

F-AR vs F-A and F-AM combined:

Source	df	MS	F	p (one-tailed)
Hypothesis	1	57.91	3.44	.035
Error	47	16.82		

Appendix G3

Pre-treatment baseline mean (SD) number of inhibitions **(Pre)** and the mean (SD) number of inhibitions averaged across the four treatment tests **(Post)** for each group:

Treatment Groups

Setting	Immediate Reinforcement		Monetary Incentive		Alcohol	
Novel	**N-AR:**		**N-AM:**		**N-A:**	
	Pre	**Post**	**Pre**	**Post**	**Pre**	**Post**
	20.89 (8.3)	20.69 (9.5)	19.67 (11.89)	19.0 (12.9)	25.89 (8.7)	26.08 (9.7)
Familiar	**F-AR:**		**F-AM:**		**F-A:**	
	Pre	**Post**	**Pre**	**Post**	**Pre**	**Post**
	22.44 (10.7)	20.94 (10.4)	22.56 (8.0)	17.11 (8.5)	27.89 (7.1)	24.08 (9.0)

Appendix G4

ANOVA of the change in inhibitions based on four tests in alcohol groups under three different treatments using the data from the three shortest stop-signal delays:

Source	df	MS	F	p
Setting (S)	1	427.85	8.06	.007
Treatment (T)	2	79.437	1.50	.234
S X T	2	50.20	0.95	.395
Error	48	53.06		
Tests (Tt)	3	5.00	0.49	.690
Tt X S	3	6.98	0.68	.563
Tt X T	6	12.71	1.25	.287
Tt X S X T	6	6.05	0.59	.736
Error	144	10.20		

Group comparisons testing a priori hypotheses:

N-A vs N-AM:

Source	df	MS	F	p
Hypothesis	1	4.76	0.36	.552
Error	48	13.26		

N-AR vs N-A and N-AM combined:

Source	df	MS	F	p
Hypothesis	1	0.20	0.02	.904
Error	48	13.26		

Appendix G4 (cont.)

Group comparisons testing a priori hypotheses (cont.):

F-A vs F-AM:

Source	df	MS	F	p
Hypothesis	1	3.34	0.25	.618
Error	48	13.26		

F-AR vs F-A & F-AM combined:

Source	df	MS	F	p (one-tailed)
Hypothesis	1	56.53	4.26	.022
Error	48	13.26		

Appendix G4 (cont.)

ANCOVA of the number of inhibitions in alcohol groups under three different treatments using the data from the three shortest stop-signal delays:

Source	df	MS	F	p
Setting (S)	1	105.60	7.80	.008
Treatment (T)	2	19.81	1.46	.242
S X T	2	12.58	0.93	.402
Covariate (Baseline)	1	3033.11	223.93	<.001
Error	47	13.55		

Adjusted mean number of inhibitions for each group:

		Treatment Groups		
		Immediate Reinforcement	Monetary Incentive	Alcohol
Setting	Novel	N-AR: 21.50	N-AM: 20.81	N-A: 21.82
	Familiar	F-AR: 20.61	F-AM: 17.10	F-A: 17.95

Appendix G4 (cont.)

Group comparisons of adjusted means to test a priori hypotheses:

N-A vs N-AM:

Source	df	MS	F	p
Hypothesis	1	4.35	0.32	.574
Error	47	13.55		

N-AR vs N-A and N-AM combined:

Source	df	MS	F	p
Hypothesis	1	0.20	0.02	.903
Error	47	13.55		

F-AM vs F-A:

Source	df	MS	F	p
Hypothesis	1	3.10	0.23	.635
Error	47	13.55		

F-AR vs F-A and F-AM combined:

Source	df	MS	F	p (one-tailed)
Hypothesis	1	56.15	4.15	.024
Error	47	13.55		

Appendix H: Descriptive statistics and supplementary analyses of RT and errors in alcohol groups under three different treatments in N & F settings

Appendix H1

Overall mean (SD) change in RT (msec) for each group:

Treatment Groups

Setting	Immediate Reinforcement	Monetary Incentive	Alcohol
Novel	N-AR: -27.32 (24.45)	N-AM: -14.29 (14.63)	N-A: +17.46 (93.47)
Familiar	F-AR: -6.70 (25.54)	F-AM: -17.79 (32.37)	F-A: +12.53 (32.02)

Mean (SD) change in RT (msec) for each group at each treatment test:

Treatment Tests

Groups	Test 1	Test 2	Test 3	Test 4
N-AR	-13.34 (30.21)	-14.33 (44.07)	-48.68 (28.71)	-32.91 (38.31)
N-AM	-18.65 (17.50)	-17.11 (10.24)	-17.98 (21.79)	-3.43 (33.19)
N-A	+4.99 (82.2)	+43.22 (147.15)	+25.64 (107.6)	-3.98 (54.89)
F-AR	-2.02 (21.53)	-9.38 (32.56)	-6.47 (24.0)	-8.91 (29.73)
F-AM	-10.04 (29.43)	-24.11 (36.32)	-13.73 (32.34)	-23.3 (41.98)
F-A	+8.92 (22.62)	+11.29 (33.02)	+13.64 (41.10)	+16.28 (40.67)

Appendix H2

The test of interactions among pre-treatment baseline mean RT (msec) and between-subjects factors:

Source	df	MS	F	p
Setting (S)	1	401.19	0.19	.666
Treatment (T)	2	1387.85	0.65	.525
S X T	2	2952.34	1.39	.260
Covariate:				
Baseline (B)	1	136322.22	64.23	<.001
B X S	1	474.43	0.22	.639
B X T	2	2457.11	1.16	.324
B X S X T	2	3220.92	1.52	.231
Error	42	2122.49		

ANCOVA of RT (msec) as a function of setting and treatment using the pre-treatment baseline RT as a covariate:

Source	df	MS	F	p
Setting (S)	1	126.26	0.61	.807
Treatment (T)	2	5806.87	2.78	.072
S X T	2	966.44	0.46	.632
Covariate				
(Baseline)	1	189487.71	90.82	<.001
Error	47	2086.53		

Appendix H2 (cont.)

Adjusted mean RT (msec) for each group:

		Treatment Groups		
		Immediate Reinforcement	Monetary Incentive	Alcohol
Setting	Novel	N-AR: 408.49	N-AM: 421.25	N-A: 454.38
	Familiar	F-AR: 428.65	F-AM: 416.78	F-A: 448.24

Appendix H3

Mean RT (msec):

Pre-treatment baseline mean (SD) RT **(Pre)** and the mean (SD) RT averaged across the four treatment tests **(Post)** for each group:

Treatment Groups

Setting	Immediate Reinforcement		Monetary Incentive		Alcohol	
Novel	**N-AR:**		**N-AM:**		**N-A:**	
	Pre	**Post**	**Pre**	**Post**	**Pre**	**Post**
	441.76	414.44	431.34	417.05	486.43	503.89
	(42.1)	(46.4)	(73.7)	(80.3)	(95.6)	(134.2)
Familiar	**F-AR:**		**F-AM:**		**F-A:**	
	Pre	**Post**	**Pre**	**Post**	**Pre**	**Post**
	423.38	416.69	392.9	375.1	438.07	450.6
	(54.2)	(52.3)	(53.9)	(32.1)	(52.3)	(74.6)

Appendix H4

Overall mean (SD) change in errors for each group in N & F settings:

Setting	Treatment Groups: Immediate Reinforcement	Monetary Incentive	Alcohol
Novel	N-AR: +4.4 (5.8)	N-AM: +4.8 (6.3)	N-A: +1.2 (3.7)
Familiar	F-AR: +2.4 (2.6)	F-AM: +4.8 (4.9)	F-A: +2.8 (3.1)

Pre-treatment baseline mean (SD) number of errors **(Pre)** and the mean (SD) number of errors averaged across the 4 treatment tests **(Post)** for each group:

Treatment Groups

Setting	Immediate Reinforcement		Monetary Incentive		Alcohol	
Novel	**N-AR:**		**N-AM:**		**N-A:**	
	Pre	**Post**	**Pre**	**Post**	**Pre**	**Post**
	10.0 (5.6)	14.4 (11.0)	11.8 (8.3)	16.6 (10.7)	6.3 (4.6)	7.5 (5.2)
Familiar	**F-AR:**		**F-AM:**		**F-A:**	
	Pre	**Post**	**Pre**	**Post**	**Pre**	**Post**
	6.2 (8.7)	8.7 (10.8)	9.2 (6.0)	14.0 (9.6)	4.1 (2.6)	6.9 (3.1)

Appendix I: Main Study - Experimental Data

The raw data are presented by participant.
X = No BAC measure (placebo participant) XX = missing data

Line 1:
1) Age
2) # of months drinking regularly
3) Frequency of alcohol consumption (# occasions per week)
4) Dose of alcohol (ml abs. Alc/kg)
5) Duration of typical drinking occasion (in hours)
6) Drink Rating on beverage rating scale
7) Rating on desire to resist scale
8) BAC #1
9) BAC #2
10) BAC #3
11) BAC #4
12) BAC #5
13) BAC #6

Line 2:
1) Pre-treatment baseline # inhibitions at 50 msec delay
2) Pre-treatment baseline # inhibitions at 150 msec delay
3) Pre-treatment baseline # inhibitions at 250 msec delay
4) Pre-treatment baseline # inhibitions at 350 msec delay
5) Test 1 # inhibitions at 50 msec delay
6) Test 1 # inhibitions at 150 msec delay
7) Test 1 # inhibitions at 250 msec delay
8) Test 1 # inhibitions at 350 msec delay
9) Test 2 # inhibitions at 50 msec delay
10) Test 2 # inhibitions at 150 msec delay
11) Test 2 # inhibitions at 250 msec delay
12) Test 2 # inhibitions at 350 msec delay
13) Test 3 # inhibitions at 50 msec delay
14) Test 3 # inhibitions at 150 msec delay
15) Test 3 # inhibitions at 250 msec delay
16) Test 3 # inhibitions at 350 msec delay
17) Test 4 # inhibitions at 50 msec delay
18) Test 4 # inhibitions at 150 msec delay
19) Test 4 # inhibitions at 250 msec delay
20) Test 4 # inhibitions at 350 msec delay

Appendix I (cont.)

Line 3:
1) Pre-treatment baseline RT
2) Test 1 RT
3) Test 2 RT
4) Test 3 RT
5) Test 4 RT

Line 4:
1) Pre-treatment baseline number of errors
2) Test 1 number of errors
3) Test 2 number of errors
4) Test 3 number of errors
5) Test 4 number of errors
6) Pre-treatment baseline no response to go-signals
7) Test 1 no response to go-signals
8) Test 2 no response to go-signals
9) Test 3 no response to go-signals
10) Test 4 no response to go-signals
11) Setting assignment (1=Novel; 2=Familiar)
12) Treatment assignment (1=Immediate Reinforcement; 2=Monetary Incentive; 3=Alcohol; 0=Placebo)

Appendix I (cont.)

Group: N-AR:

---

21 XX 3 0.992 3 2 3 32 71 84 79 72 63
12 11 6 1 11 10 2 0 12 10 5 1 12 11 1 0 12 11 3 0
417.770 374.500 380.490 346.290 366.760
10 9 18 18 10 0 0 1 0 0 1 1

---

19 20 2 0.934 4 4 7 67 79 71 62 59 55
11 9 8 2 12 12 8 7 12 12 10 6 10 9 3 1 12 12 4 0
504.100 535.070 587.330 425.100 418.630
2 1 1 4 2 0 0 0 0 0 1 1

---

21 56 1.500 2.819 4 7 4 38 59 71 70 69 63
9 1 1 0 8 5 0 0 9 5 1 0 5 5 2 1 7 6 0 0
388.990 380.510 360.440 349.340 396.890
14 20 29 23 10 1 0 0 2 0 1 1

---

23 51 2 0.363 2 2 9 52 63 55 54 48 42
10 4 4 1 8 8 3 0 11 12 6 0 10 10 5 0 9 6 4 1
471.470 494.710 458.830 427.490 420.390
9 7 6 9 14 0 0 0 0 0 1 1

---

19 29 0.115 1.461 3 4 3 56 69 73 69 68 62
11 8 3 2 12 12 5 1 11 11 7 3 12 11 6 2 12 9 9 0
444.540 452.290 458.510 441.880 475.660
5 5 15 4 5 0 0 0 0 0 1 1

---

20 64 2 1.834 5 3.500 5 27 54 64 73 79 74
11 9 3 0 11 10 3 0 12 10 2 0 12 11 2 0 12 6 2 0
400.170 383.250 388.030 386.510 371.660
6 3 8 1 2 0 0 0 0 0 1 1

Appendix I (cont.)

Group: N-AR (cont.):

---

21 75 0.625 3.807 5 1 1 39 68 74 70 66 59
12 10 6 1 10 10 0 0 10 10 2 0 11 10 4 0 12 9 6 1
484.220 433.260 437.890 429.630 472.930
8 9 10 19 12 0 0 2 0 1 1 1

---

20 28 0.750 1.233 4 5 10 29 48 XX XX 50 50
6 4 1 1 2 0 1 0 8 2 0 1 5 6 2 1 3 2 3 0
466.620 418.570 393.520 377.330 388.340
18 26 22 31 28 1 0 0 1 1 1 1

---

19 45 0.500 0.506 2 2.670 5 49 68 61 61 60 59
6 3 1 0 5 1 0 0 2 1 1 0 0 2 0 0 5 2 1 0
397.930 383.590 381.800 354.160 368.320
18 28 29 41 41 1 0 0 1 0 1 1

Appendix I (cont.)

Group N-AM:

---

19 10 1 0.673 2 1.330 6 58 79 63 62 51 44
10 7 5 0 9 3 2 0 9 8 5 2 9 8 4 2 12 10 8 4
453.740 405.300 422.280 427.830 457.720
4 10 17 8 14 2 1 3 0 2 1 2

---

21 80 0.875 0.482 4.500 3 3 47 60 52 51 48 48
12 11 11 5 12 11 10 5 11 10 11 3 12 11 10 6 12 11 11 4
535.050 522.020 525.730 529.730 538.410
1 2 8 2 1 0 0 0 0 0 1 2

---

20 21 1 0.406 2 5 7 48 96 84 82 69 65
5 6 1 3 4 2 3 0 1 3 2 0 6 6 3 1 5 6 0 1
408.540 372.810 385.570 408.570 392.700
9 16 20 14 16 0 0 0 0 0 1 1 2

---

20 24 0.019 0.055 0.083 2.330 4 58 72 79 76 72 68
10 4 2 0 11 9 4 0 9 10 4 0 7 9 4 1 11 5 3 1
386.190 379.710 371.770 392.970 427.740
21 30 28 30 22 3 0 3 3 3 1 2

---

21 64 2 1.859 6 5 6 38 61 71 78 72 68
7 2 2 0 7 4 1 0 5 2 0 0 5 1 0 0 3 1 0 0
392.250 359.920 365.940 363.100 345.380
19 14 16 17 24 0 1 1 1 0 1 2

---

19 42 2 1.901 5 6.500 5 93 110 86 84 81 79
9 4 1 0 7 7 1 0 9 5 1 0 8 3 0 0 8 2 1 0
362.910 349.040 340.830 310.840 318.510
9 16 12 18 19 0 0 1 3 0 1 2

Appendix I (cont.)

Group N-AM (cont.):

---

20 50 3 0.244 1.500 4.500 6 42 89 97 86 69 62
12 12 10 6 12 11 9 6 12 12 11 6 11 12 9 6 11 9 11 11
570.500 578.600 553.190 533.810 613.010
13 6 6 3 3 1 0 2 2 1 1 2

---

20 30 0.750 0.791 6 7 6 50 69 70 71 69 59
5 2 1 0 2 0 0 0 2 1 0 0 1 0 0 0 3 1 0 1
389.170 386.970 376.020 400.220 401.880
5 11 9 15 19 0 0 2 3 6 1 2

---

19 42 2 1.229 6 4 6 33 63 76 87 82 69
6 4 1 1 9 6 1 1 8 6 2 1 9 4 1 0 5 4 0 0
383.730 377.870 386.740 353.200 355.830
25 23 31 50 46 2 2 4 0 4 1 2

Appendix I (cont.)

Group N-A:

---

21 31 2 1.226 4.500 6 3 53 73 69 61 53 49
8 8 2 0 10 9 3 0 12 8 5 0 11 8 2 0 11 8 3 0
420.400 398.040 396.380 394.470 416.040
5 13 8 8 2 0 0 0 1 1 1 3

---

19 26 0.750 0.931 4.500 5 8.500 32 93 87 77 62 59
10 5 6 2 4 2 0 0 3 3 1 0 9 7 1 0 6 4 2 0
456.290 420.060 439.090 436.950 419.690
10 3 5 12 9 10 0 0 0 0 0 1 1 3

---

19 24 1 0.996 4 4 8 35 68 91 78 63 52
12 11 12 5 12 12 11 5 12 12 12 2 12 12 12 9 12 12 11 7
471.070 474.020 510.330 493.240 480.840
1 1 1 2 1 0 0 0 0 0 1 3

---

20 29 2 1.090 3 3 8 51 55 68 71 68 68
11 7 7 4 10 10 10 6 11 10 9 8 7 7 8 7 8 8 6 3
560.230 774.440 982.890 837.040 635.830
0 13 8 11 10 10 0 1 4 1 0 1 3

---

19 59 2 0.259 2 3 7 49 72 72 69 58 45
10 10 3 1 8 9 5 3 10 10 6 1 11 12 5 1 11 12 6 1
441.960 455.530 487.340 485.940 509.070
6 13 4 5 2 0 0 0 0 2 1 3

---

19 63 3 1.323 7 7 7 31 73 82 74 68 54
7 4 2 0 10 2 0 0 9 4 1 0 9 7 0 0 11 7 1 0
412.000 381.860 392.210 377.960 379.560
13 15 20 20 21 4 2 0 1 0 1 3

Appendix I (cont.)

Group N-A (cont.):

---

19 60 2 2.275 4 7 9 68 77 71 69 66 53
11 10 8 7 9 11 7 4 7 7 6 5 8 11 7 6 11 12 7 6
709.670 646.460 633.790 625.990 604.920
4 6 8 8 8 0 0 4 2 2 1 3

---

21 34 0.077 0.669 4 3.330 5 52 78 85 79 78 62
9 6 3 2 8 6 0 0 11 7 1 0 10 10 4 2 9 10 4 1
488.880 450.630 480.880 497.310 468.030
10 5 6 1 10 0 0 0 0 0 0 1 3

---

19 25 0.096 0.467 5 2.500 2 55 71 82 69 62 59
12 12 5 1 12 11 6 1 12 11 8 1 12 12 11 3 12 12 9 2
417.360 421.690 443.900 459.700 428.030
2 1 2 1 3 1 0 0 0 0 1 3

Appendix I (cont.)

Group N-P:

---

23 43 0.106 0.728 3.500 1.500 5 X X X X X X
8 10 4 0 11 9 4 0 12 9 7 2 12 9 5 1 11 11 6 4
465.330 453.080 440.080 462.280 465.390
4 0 4 3 1 1 6 3 1 1 1 0

---

19 24 2 2.051 4.500 1.500 1 X X X X X X
12 10 8 2 12 12 10 3 12 12 11 2 12 12 11 3 12 12 7 3
479.380 476.120 458.900 483.530 469.500
7 0 3 1 1 0 0 0 0 0 1 0

---

21 32 0.875 0.945 4.500 2.500 0 X X X X X X
10 9 3 1 9 5 1 1 10 7 1 0 11 6 0 0 11 7 1 0
416.910 376.370 385.250 369.310 391.140
9 7 8 4 6 0 0 0 0 1 1 0

---

19 51 1.500 1.860 7.500 1 0 X X X X X X
11 11 4 2 12 7 1 0 12 7 2 1 12 11 5 1 12 7 3 2
400.820 389.880 410.520 427.430 402.430
12 11 7 13 4 1 2 1 0 1 1 0

---

20 52 3 1.823 4 1 3 X X X X X X
10 9 3 2 11 10 5 1 9 11 7 0 7 8 4 1 11 9 7 1
452.930 438.250 440.130 452.740 444.840
3 3 5 1 2 0 0 1 0 1 1 0

---

19 45 1 1.343 XX 0.667 0 X X X X X X
6 5 1 2 9 4 2 0 6 4 1 0 6 5 0 0 4 3 1 1
391.970 378.880 383.410 355.140 383.360
18 10 15 14 14 3 2 1 2 2 1 0

Appendix I (cont.)

Group N-P (cont.):

---

19 44 0.375 1.062 3 1 1 X X X X X X
5 4 0 0 8 4 1 0 8 0 0 1 9 4 1 0 10 3 0 0
367.610 336.920 337.100 331.720 328.000
16 8 13 9 13 0 1 1 0 0 1 0

---

21 55 0.058 0.671 1.500 2 4 X X X X X X
11 8 2 1 11 7 2 0 12 8 1 0 12 6 0 1 11 7 0 0
390.800 354.970 358.160 356.370 358.070
12 23 15 18 19 1 0 0 0 1 1 0

---

20 15 0.750 0.861 4 2 3 X X X X X X
6 2 1 2 8 4 1 0 12 2 3 1 9 7 2 1 10 6 2 0
408.430 400.370 395.130 380.670 393.330
7 6 12 6 10 2 1 1 0 0 1 0

Appendix I (cont.)

Group F-AR:

---

19 12 0.250 0.464 1.500 2.500 7 58 91 76 75 68 56
4 2 0 0 6 2 0 0 5 3 0 0 9 3 0 0 7 3 0 0
360.790 369.920 331.940 338.850 330.610
9 7 15 18 20 2 0 0 0 0 2 1

---

19 42 1 0.887 3 3 7 120 87 78 74 62 51
6 2 0 1 4 0 1 0 5 3 0 0 3 1 3 0 4 3 1 1
364.950 375.400 336.640 358.110 361.850
28 39 37 31 33 1 1 1 0 0 2 1

---

19 34 2 1.410 6 6.500 7 43 67 74 62 56 51
10 8 5 1 12 7 4 0 11 11 2 1 12 11 3 0 12 11 5 0
412.940 401.460 396.450 402.460 406.980
2 4 2 1 1 0 0 0 0 0 2 1

---

20 17 1 1.237 4 3 8 29 58 72 72 63 56
8 6 2 1 12 3 1 0 8 1 1 0 10 3 1 0 11 4 0 0
426.780 403.890 387.080 408.410 377.970
8 8 12 7 11 1 0 0 0 0 2 1

---

21 10 0.500 1.090 5 2.670 4 49 67 70 70 72 78
12 12 5 2 11 9 8 4 12 12 10 1 12 11 3 2 11 10 5 1
467.270 471.640 492.900 469.770 483.910
1 3 0 4 5 1 0 0 0 0 2 1

---

19 42 2.500 1.375 3 4 2 61 86 66 47 43 42
12 10 2 1 12 10 4 2 12 9 3 1 11 9 2 2 12 7 1 0
419.510 419.640 425.430 426.880 426.100
3 2 3 2 1 0 0 0 0 0 2 1

Appendix I (cont.)

Group F-AR (cont.):

---

21 42 1.500 2.026 5.500 5.500 5 66 95 91 71 65 60
11 6 1 1 8 3 1 0 9 4 1 1 9 6 1 0 6 2 0 0
366.480 396.510 394.920 396.410 399.630
1 5 4 5 2 0 0 1 0 0 2 1

---

21 44 1 1.074 4 3 4 61 73 67 62 49 45
12 11 8 4 11 10 4 1 12 11 4 2 12 11 5 1 12 10 4 1
508.820 464.780 449.630 455.190 454.130
3 3 3 4 5 0 0 1 0 0 2 1

---

21 67 2 1.769 5 4 2 28 47 59 54 51 49
12 11 9 4 12 11 12 5 11 12 11 2 11 11 8 6 11 12 9 5
482.920 489.040 511.020 496.190 489.080
1 3 4 4 4 3 7 2 7 2 2 1

Appendix I (cont.)

Group F-AM:

---

19 14 2 0.701 4 5.500 8 60 78 85 79 76 66
9 10 3 1 8 4 1 1 10 6 1 0 11 6 3 0 9 6 2 1
367.910 359.960 356.620 361.710 357.800
8 13 12 13 9 2 2 5 1 0 2 2

---

19 21 1.500 0.461 4 2 7 49 80 78 77 68 60
12 10 9 5 12 9 5 0 12 10 3 0 12 12 6 0 12 11 2 0
518.100 438.160 406.600 428.710 419.510
4 7 6 9 7 0 0 0 1 0 2 2

---

20 21 1.500 1.185 5.500 3.500 5 52 89 86 69 59 52
11 8 4 3 11 9 2 0 8 6 1 0 7 5 5 1 8 7 4 3
428.140 441.970 404.040 428.660 437.020
2 4 5 5 3 4 0 0 10 11 2 2

---

19 10 1 0.444 4 4 5 48 72 64 59 57 57
7 4 2 2 6 2 0 0 8 0 1 0 8 4 1 0 7 7 0 0
361.790 353.400 343.250 377.230 360.940
13 16 15 14 15 2 0 0 0 0 2 2

---

19 58 0.500 0.482 1 3 7 58 84 83 83 75 70
8 7 1 0 8 3 1 0 10 4 0 0 8 1 0 0 1 0 0 1
358.190 352.160 357.140 340.600 280.720
16 23 19 37 46 1 1 3 3 0 2 2

---

21 69 0.500 1.375 4.500 3.500 5 31 64 79 76 69 59
9 5 0 0 5 1 0 0 3 0 0 0 2 0 0 0 2 0 0 0
365.100 370.220 379.630 362.010 361.580
2 5 4 5 5 0 0 0 0 0 2 2

Appendix I (cont.)

Group F-AM (cont.):

---

19 42 0.500 2.037 3 5.500 6 87 82 74 67 59 59
11 12 6 1 12 11 3 1 12 10 5 0 12 12 4 0 12 12 4 0
381.740 394.540 380.010 385.630 377.660
7 10 7 2 6 2 1 2 0 1 2 2

---

19 29 1.125 1.049 4.500 7 7 27 39 43 47 54 48
12 12 4 0 10 10 6 0 12 11 5 2 11 10 3 1 12 9 2 0
410.540 381.950 372.580 373.600 361.420
13 18 22 22 30 2 3 2 3 0 2 2

---

19 41 3 1.267 4 5.500 6 33 59 77 75 68 59
11 4 0 0 11 8 0 0 8 3 1 0 9 3 0 0 11 2 0 0
344.570 353.360 319.230 354.350 369.760
18 22 32 17 19 1 2 3 0 2 2 2

Appendix I (cont.)

Group F-A:

---

19 24 1.500 1.062 3 3 6 69 103 77 67 59 56
10 6 0 2 5 3 0 0 6 5 1 0 4 2 1 0 5 0 0 1
446.040 451.640 481.840 489.780 450.820
4 9 7 9 5 2 1 2 0 1 2 3

---

19 28 1.500 1.361 2 4 9 48 62 54 48 48 47
12 11 4 0 12 6 3 1 10 7 0 0 10 6 1 0 12 6 5 2
412.520 421.350 406.550 409.790 420.420
9 8 8 5 8 0 0 1 0 0 2 3

---

20 42 1.250 1.384 3.500 4 4 42 83 88 80 69 59
12 11 10 6 10 9 7 7 11 10 8 7 10 8 4 7 12 10 8 8
539.180 602.890 625.030 612.070 651.070
1 3 3 7 4 0 0 0 0 0 2 3

---

19 16 0.250 1.087 5 3.330 2 57 89 71 69 62 58
12 11 4 1 11 10 3 0 11 8 4 0 10 8 3 1 12 11 5 2
400.420 396.610 396.840 368.000 403.170
4 6 4 6 8 2 1 0 0 1 2 3

---

20 74 2.500 1.187 5 6 6 110 107 104 101 97 81
11 12 8 6 12 12 8 5 12 12 10 5 12 12 10 8 11 11 11 4
491.020 495.260 484.760 531.270 508.450
0 4 2 3 1 2 6 5 2 3 2 3

---

19 54 4 2.111 5.500 5 0 47 68 68 66 58 52
11 11 9 2 12 11 6 2 12 10 3 1 10 11 5 2 11 11 6 3
452.620 432.710 433.450 411.950 433.440
4 11 8 10 8 0 2 0 0 0 2 3

Appendix I (cont.)

Group F-A (cont.):

---

20 48 3.500 0.511 0.500 4 8 33 51 61 79 70 68
12 8 2 0 12 7 4 0 7 10 4 0 12 5 0 1 9 9 6 0
438.030 447.810 423.680 409.240 412.280
4 5 13 16 18 0 2 3 1 1 2 3

---

20 36 0.250 2.308 3 5 7 78 92 69 69 67 51
10 8 4 3 6 5 1 1 9 8 2 1 12 11 2 1 8 10 5 1
388.460 399.200 411.160 436.280 428.850
6 14 8 7 3 1 0 0 2 1 2 3

---

19 36 2 2.251 6 7 8 35 68 78 77 69 59
11 9 2 0 12 7 2 0 11 8 1 0 11 8 2 0 10 6 0 0
374.350 375.410 380.940 396.980 380.640
5 5 6 1 7 0 0 0 0 0 2 3

Appendix I (cont.)

Group F-P:

---

20 40 0.750 0.829 1 2.500 8 X X X X X X
12 12 6 2 12 12 8 4 12 12 9 2 12 12 7 2 10 12 3 1
473.230 452.490 448.070 446.050 471.840
6 3 3 3 0 0 0 0 0 2 2 0

---

19 20 1.500 0.283 2 1 4 X X X X X X
10 9 0 3 12 8 4 1 12 12 8 2 12 12 8 2 12 12 9 2
453.780 443.750 472.970 454.790 463.980
9 9 4 2 7 0 0 1 0 1 2 0

---

19 18 1.500 2.323 5.500 1.500 1 X X X X X X
8 6 0 0 10 5 3 3 10 5 2 0 8 6 1 0 11 10 5 2
410.600 386.620 387.520 389.130 448.950
23 17 21 16 6 0 1 0 0 1 2 0

---

19 30 3 1.695 5 0.500 1 X X X X X X
10 11 3 3 11 9 3 0 10 9 4 0 12 9 2 0 11 8 3 0
400.030 388.250 381.290 361.320 363.740
10 14 14 15 11 0 3 1 1 0 2 0

---

19 12 0.875 0.684 4 2.500 1 X X X X X X
12 12 10 10 11 12 9 6 12 10 8 9 10 12 11 8 12 12 8 8
654.150 560.380 577.630 597.160 600.170
0 2 1 2 0 2 1 2 2 2 2 0

---

19 36 2 1.504 5 1.500 7 X X X X X X
7 6 1 1 6 2 1 0 3 1 0 0 3 2 0 0 5 5 1 0
367.190 357.830 356.180 361.980 354.620
8 7 4 13 5 1 0 0 0 0 2 0

Appendix I (cont.)

Group F-P:

---

20 16 1.500 0.940 3.500 1 2 X X X X X X
8 10 1 0 9 6 2 0 9 3 0 0 9 3 2 0 9 5 1 0
360.770 349.480 338.090 353.850 340.260
9 9 11 9 11 0 0 0 0 0 2 0

---

19 33 0.500 0.620 4 3 1 X X X X X X
10 9 6 0 12 5 3 0 9 8 1 1 8 12 5 2 11 10 3 1
436.040 414.350 422.090 441.690 421.330
10 7 10 6 9 2 0 2 2 1 2 0

---

21 10 0.250 0.406 3 1.500 2 X X X X X X
12 12 3 1 12 12 4 0 12 8 2 0 12 12 2 1 12 12 2 0
397.590 365.310 367.500 365.480 379.020
1 4 4 6 4 0 0 0 0 1 2 0
