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Effects of Alcohol on Emotionally Salient Memory

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July 1997

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfilment of the requirements for the degree of Doctor of Philosophy.

General abstract

Social drinkers (healthy males aged 18-34) participated in three experiments that examined some of the mechanisms that may be responsible for the effects of alcohol on emotionally charged memory. In a study on incidental learning, alcohol enhanced neutral, positive and negative memory, possibly by a nonselective consolidation mechanism. Alcohol's enhancement of memory was found to not be associated (contingently related) with its incentive reward and relief effects. In another study on intentional learning, alcohol enhanced positive memory and/or inhibited negative memory, possibly reflecting a contingent relationship to its incentive effects. When the role of individual differences was examined, relatively little influence was found in mediating the effects of alcohol on incidental memory. However, alcohol's effect on intentional memory was predicted by individual differences in response to some of alcohol's acute incentive effects. The studies demonstrate that alcohol's effects on memory are independent from, and can be combined with, its incentive effects. Further, consumption of alcohol may be influenced by separate effects on memory and incentive, and by their association.

Résumé

Des buveurs sociaux (des hommes en bonne santé physique, âgés de 18 à 34 ans) ont participé à trois expériences examinant certains des mécanismes pouvant être responsables des effets de l'alcool sur la mémoire à charge émotive. Lors d'une étude sur l'apprentissage non-intentionnel, l'alcool a amélioré la mémoire neutre, positive et négative, possiblement par l'entremise d'un mécanisme de consolidation non-sélectif. La facilitation de la mémoire n'est pas associée (par contingence) aux récompenses de la motivation et aux effets soulageants de l'alcool. Lors d'une autre étude sur l'apprentissage intentionnel, l'alcool a facilité la memoire positive et/ou négative inhibitée, reflétant possiblement un raport de contingence avec ses effets sur la motivation. Une fois le rôle des différences individuelles examiné, relativement peu d'influence fut trouvée quant à la modulation des effets de l'alcool sur la mémoire non-intentionnelle. Toutefois l'effet de l'alcool sur la mémoire intentionnelle a été prédit par les differences individuelles en réponse à certains des effets sur la motivation aigüs de l'alcool. Ces études démontrent que les effets de l'alcool sur la mémoire sont indépendants de, et peuvent être combinés à, ses effets sur la motivation. De plus, la consommation peut être influencée par des effets séparés de l'alcool sur la mémoire et sur la motivation, ainsi que par la combinaison de leurs effets.

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Contributions and academic acknowledgements

Dr. **Robert Pihl** supervised the research described in this thesis. When I first met Dr. Pihl and then came to McGill, I was impressed with his frankness and directness. Dr. Pihl always encouraged, respected, trusted and sought out every possible research and financial opportunity for me. I wish to thank him for his resources and resourcefulness, and ability to eliminate red tape for me. His patience, and his pragmatic nature focused me from vague intuitions to immediate, exciting and achievable goals. Dr. Pihl allowed me to entertain and pursue ideas and hypotheses that were originally steeped in confusion and undetermined relevance. I believe he did this merely because he saw they were of keen intrigue to me, and he gambled that we could eventually make them of interest to others as well. (I feel satisfaction that we have and will!) His direction has been

unimposing and constant, and I look forward to the opportunity of working with him again as I have in the past.

Dr. **Maurice Dongier** of McGill's Psychiatry Department and the Douglas Hospital provided me with my first, and also my most recent scientific endeavours since coming to Montreal. Because of his experience and interest in all aspects of alcohol research, discussions and collaborations with him have benefitted me immensely. Dr. Dongier and Dr. **Richard Tremblay** at the Université de Montréal have also provided me with the financial resources this past year to actually sit down and write this thesis. I also thank the other members of the **McGill-Douglas Alcohol Research Group** for the many opportunities to present my ideas and receive important feedback concerning them.

Dr. **Norman White** provided an added framework from which to test some of the ideas we had concerning the incentive effects of alcohol. Dr. White published papers in 1989 (and 1992 with Peter Milner) dealing with how "reinforcement" occurs in animals. Along with Dr. Pihl's work on markers for alcoholism, my reading of Dr. White's papers, and later adapting them to our population, were part of the impetus for the studies contained herein. Dr. White's 1996 paper solidified my confidence that these initial speculations in 1992 about the effects of alcohol for memory and emotion had indeed merited the closer examination.

I thank all the professors at McGill who taught my graduate seminars, and those who supervised my practica and internship courses. Special thanks goes to Dr. **Cam Zacchia**, and Dr. **Sylvain Néron**. They proved to me that those who "can" also "teach". I'd also like to thank Dr. Pihl's other students past and present. They provided much of

the foundation and context for the work described herein. In particular I'd like to single out Dr. **Jordan Peterson** for his unstoppable creativity, interest and enthusiasm. The Résumé, page 3, was translated by my lab colleagues, **Etienne Dubreuil** and **Jean Séguin**.

Drs. **Laurence Becker**, **Gwen Ivy** and **Esther Gelcer** at the University of Toronto provided me with the research experiences that preceded and precipitated my coming to McGill. They were excellent research advisors, and exemplary personal mentors.

I would also like to thank the **Fonds FCAR** (Québec) for pre-doctoral fellowship B2-08A.

I thank my students **Robbie Goddard, Edwin Poon** and **Jennifer Crotogino** for helping me to collect the data in the first study. I also thank my students **Karen Milligan, Tsafrir Vanounou** and **Maria Arvanitakis** for helping collect and enter the data for the second study. I thank my students **John Shestowsky** and **Jamie Mayerovitch** for their reliable assistance and challenging conversations that took place during the second and third experiments.

I thank **Rhonda Amsel** and **Jean Séguin** for keen statistical eyes; their suggestions allowed me to examine and present my findings with the necessary rigor and confidence.

Study 1 herein is currently in press (August 1997). We thank the Journal Editor, the anonymous reviewers, and the APA Copy Editor, all of whose comments are now incorporated in the version of the paper presented here.

Bruce, K. R., & Pihl, R. O. (1997). Forget "drinking to forget": Enhanced consolidation of emotionally charged memory by alcohol. *Experimental and Clinical Psychopharmacology*, *5*(3).

Study 2 is also currently in press. The comments and suggestions of the Journal Editor, and the anonymous reviewers are incorporated in this version of the manuscript.

Bruce, K. R., Pihl, R. O., Mayerovitch, J. I., & Shestowsky, J. S. (in press). Alcohol and memory consolidation: Role of individual differences. *Journal of Studies on Alcohol.*

Study 3 is currently under review at *Experimental and Clinical Psychopharmacology.*

Bruce, K. R., Pihl, R. O., Shestowsky, J. S., & Mayerovitch, J. I. (submitted). Effects of alcohol on intentional memory and subjective ratings for emotionally charged material.

Personal acknowledgements

I thank my parents, **Donna** and **George Bruce,** for their love and support. They instilled confidence and fostered independence early on -- tools that successfully completing a doctoral dissertation necessarily requires. They taught me that self-discipline, persistent hard work, and determination are their own rewards. I thank **my family** too, for their love, support and encouragement.

Thanks and appreciation also go to **Rania Papanas**. Rania has always done more than any other human being could possibly do so that I felt supported, confident, and excited about myself and my work. I'd especially like to thank her for the fantastic job she did in helping me to clarify my ideas throughout the thesis, and for her superb proofreading. To her goes all my love, respect, and admiration. She inspires me to carry out the work I do, so this Ph.D. is as much hers as mine... Thanks Rania!

I also thank Rania's parents, **Dimitrios** and **Ekaterini**. They have treated me better than I ever would have imagined -- as one of their family. Without their unconditional support, my life in Montreal would be much less happy and comfortable.

I thank **Aki Caramanos** for his ability to challenge me to express my ideas with the appropriate rigor and specificity. I also thank Aki's parents for warmly welcoming me. Thanks also to **Dave LeMarquand** for showing me that alcohol research was indeed fun and worthwhile. He is a friend and colleague of great talent and humility. It will be an honour to be a postdoc with him and the rest of the gang at Fernand Séguin.

Statement of originality

This thesis presents information novel and unique in a number of respects. The general introduction's literature review is an update and integration of findings, ideas and theories (some familiar and some new) concerning the effects of alcohol on emotion and memory. Further, a novel proposal is made whereby a theory concerning the actions of "reinforcers" found in the behavioural neuroscience literature (White & Milner, 1992) could be adapted and extrapolated to the problem of describing the effects of alcohol on memory in social drinkers. An original hypothesis is made that alcohol use in humans might involve the same dual (memory and incentive) actions as other conventional and drug reinforcers. A calculated (and ultimately successful) risk is taken whereby the memory actions of alcohol were to be examined by using incidental and intentional memory paradigms We hypothesized that the effect of alcohol on incidental memory would **not** be contingent on (i.e., would be independent of) its desirable incentive properties -- much as Norman White's (Messier & White, 1984) study on the memory enhancing effects of postlearning *sucrose injections* in rats. Further, we advance another original hypothesis that, by contrast, alcohol's influence on intentional memory would reflect a contingent association with its incentive properties -- much as Norman White's study (Messier & White, 1984) on the memory modulating effects of postlearning *saccharin consumption* in rats.

Study 1, begun September 1992, is unique and original for several reasons. First, because of the somewhat daring (i.e., unproven!) methodology, and second, due to the

intriguing findings. The Velten (1968) Mood Induction Procedure is used here in a completely novel context. For the first time, experimental participants are exposed to all 3 statement types, and memory for the statements themselves is tested. An examination of the retrograde effects of alcohol consumption on emotionally charged memory has never been carried out in humans. Thus, hypotheses concerning the relationship between desired effects of alcohol, and the effects of alcohol on memory consolidation are tested for the first time as well. As for the findings, the design includes manipulations whereby some of the mechanisms thought to be involved in the effect (but never examined directly) can be examined. In this regard, the involvement of interference reduction and (a few) individual differences is tested. Most novel, however, is that the results can be explained if White and Milner's (1992) ideas are extrapolated to fit alcohol's effects. Thus, incidental (non-contingent) enhancement of both emotional and neutral memory likely occurs by a nonselective pharmacological effect of alcohol on memory consolidation. Alcohol's contingent incentive reward and relief properties do not seem to be involved in its effects on incidental memory, and alcohol enhances memory much like Norman White's sucrose injections.

Study 2, begun September 1994, is unique in that it represents the first attempt at relating the retrograde consumption of alcohol to individual differences in participants. Kalin (1964) speculated that individual differences might be implicated, but the hypothesis had never been tested. We had included a few variables in Study 1, but had not done so systematically. Variables included here were verbal and memory skills, emotional response style, personality, mood state, subjective and physiological response to alcohol,

and alcohol expectancies. The findings were original in that few individual differences did predict alcohol's effects on incidental memory, suggesting for the first time that alcohol has similar effects on consolidation of incidental memory across individuals.

Study 3, begun September 1995, complements the first two. Instead of an incidental paradigm, an intentional learning paradigm was used. The goal was to examine the effect of changing the instructions given to participants. In so doing, the relationship between memory and alcohol consumption was made explicit, and a contingency between alcohol and memory was established. The uniqueness was in finding that alcohol affected memory for negative experiences differently than memory for positive experiences. Further, measures of alcohol-induced changes salience for the material were used. Again, the results can be explained if White and Milner's (1992) ideas are extrapolated. Thus, intentional (contingent) influences on both types of emotional memory likely occurs by a conditioned motivation. Alcohol influenced memory such that desirable memory outcomes were produced; alcohol acted much as Norman White's studies on postlearning saccharin or sucrose consumption in rats.

Thus, the integration of the studies in this thesis is original in that it was shown that for the first time that alcohol enhances incidental memory by (nonselectively) enhancing the physiological events underlying memory for emotionally charged material. By contrast, we have shown for the first time that alcohol influences intentional memory by conditioned association of its incentive reward and/or relief effects. We also specify for the first time the parameters necessary for the involvement of individual differences in the memory effects.

The successful extrapolation of the White and Milner (1992) paper is particularly original given that traditional "reinforcement" theories of the effects of alcohol cannot explain the incidental memory results (these theories predict relative enhancement of positive over negative memory -- i.e., the conditioned motivation explanation). In addition, semantic network theories of emotion and alcohol expectancies cannot fully explain the incidental results (they would also have predicted relative enhancement of positive over negative memory). An original explanation was required, and has been provided herein.

This thesis attempts for the first time to examine the relationships among emotion, memory, incentive and alcohol in a paradigm specifically designed to model "emotional" memory consolidation. It brings together the memory-based theories of reinforcement, alcoholism and alcohol expectancies in a cognitive-emotional way designed to have both methodological rigor, and some element of external validity.

My own contribution, and the contribution of others, can be described as follows. The introduction is entirely my own, edited by Dr. Pihl.

The first study was designed by me, with input from Dr. Pihl. It was my contention that posttraining experiments could differentiate alcohol's incentive effects from its memory effects. Because others had shown that alcohol enhanced neutral memory, and because we knew that alcohol had acute incentive on emotion, we designed a posttraining experiment with alcohol and long-term effects on emotionally salient memory. I collected and analyzed the data, wrote the paper, and Dr. Pihl edited it. My students Robbie Goddard, Edwin Poon and Jennifer Crotogino assisted me in collecting

the data.

Kalin (1964) had hypothesized that individual differences might be important in the posttraining memory effects. Study 1 herein had examined a few individual difference variables. However, I decided that Kalin's hypothesis had been (unfortunately) overlooked by other researchers, and we proceeded to examine what other alcohol-related individual variables might be implicated. This led me to select a wide range of such variables--anything that might be related to individual differences mediating emotion, memory, and response to alcohol. Here my students Jamie Mayerovitch and John Shestowsky assisted me with the data collection. I decided to make them co-authors because they were particularly helpful during the time when I had a serious but acute medical problem, and was unable to run subjects for several weeks. I wrote the paper, and Dr. Pihl edited it.

The third study was designed by me to complement the first two in terms of examining the effects of a shift in paradigm on verbal memory. I had lingering questions. Were the effects of alcohol different for intentional than for incidental memory? Was the relationship between the incentive effects of alcohol and incidental memory the same for intentional memory as well? What was the effect of making a contingent relationship between memory and alcohol? Were contingent effects of alcohol really like posttraining access to saccharin? Following conversations I had with John Shestowsky, I also decided to examine the role of alcohol-induced changes in salience for the verbal material. Again, John and Jamie assisted in the data collection, I wrote the paper, and Dr. Pihl edited it.

Science must begin with myths,

and with the criticism of myths

-- Karl Popper

Alcohol impairs memory. It can be classified as a sedative or anxiolytic. Alcohol's pleasurable effects show it is "reinforcing".

It will be argued that the preceding statements are *myths* in that, although widely believed, they are at best only half true. In general, intoxicated persons do have difficulty encoding and retrieving information. However, it is an unfortunate myth that alcohol impairs memory, since it actually enhances consolidation. Alcohol has sedative effects, but it must be noted that these typically follow its initial (and often overlooked) stimulant effects. Further, alcohol's "reinforcing" action is not a unitary phenomenon; rather, it involves two separate processes: (a) alcohol has pleasurable subjective and physiological effects: it reduces negative emotions and heightens pleasant emotions, and (b) alcohol enhances the representation of events in memory. Both processes together determine "reinforcement". Support for these claims, among others, will be elaborated in this thesis.

Let's begin the discussion of reinforcement with an analogy. A laboratory rat is situated on a slightly elevated platform. If he steps off the platform on to the floor below (his natural tendency), he receives a mild shock since we have electrified the floor. Our

rat steps back on to the platform. He tries to step down again, and again is shocked. Eventually he stays quietly on the platform, stepping down only occasionally.

Depending on our perspective, we might say that we have "punished" the rat's tendency to step down. Or, we might say that the rat had learned a *new* tendency, to "stay put". The first description may be preferable to some since it requires fewer assumptions about what is "going on" inside our rat, but the latter description conveys more information.

We take our rat from the platform and return him to his cage. While there, we give him access to drink solutions of sucrose or saccharin (equated for desired sweetness according to our rat's prior experience), or a saline solution. The next day, if our rat had access to saccharin, he stays on the platform longer (steps down less often) than if he had access to saline (Messier & White, 1984). Since saccharin has minimal metabolic value, perhaps the sweetness of the saccharin acted as a "reward" for "staying put." A *contingency* between staying put and the subsequent sweet reward would be required for this to be true. (Contingent saccharin consumption can improve maze learning as well as passive avoidance learning; Stefurak & van der Kooy, 1992.) Note that the sweetness did not improve memory per se. Sweetness's incentive had to be associated with "staying put". This association is a separate physiological process. As we will see, incentive is mediated by the ventral tegmental area (VTA) and nucleus accumbens. Conditioning of the sweetness occurs in separate brain areas, likely the amygdala, hippocampus and dorsal striatum.

If our rat had access to sucrose, it stays on the platform even longer than if it had

access to saccharin. Perhaps the "metabolically active" properties of the sucrose (saccharin has no such properties) acted such as to improve memory in addition to the sweetness "reward" (the solutions were equated for sweetness). We might say that both compounds have influenced memory by a contingent association between incentive properties of the substances, and memory for events in the environment (White & Milner, 1992). But we are intrigued as to why the sucrose improved memory still further.

From the platform, we remove another rat who has learned to "stay put" and return him to his cage. Before doing so we give him an *injection* of sucrose or saccharin (equated for sweetness according to our rat's prior experience), or a saline control. The next day, our rat remains longer on the platform if he had been injected with sucrose than if he had been injected with saccharin or saline (Messier & White, 1984). Perhaps the metabolically active properties of sucrose did indeed have some action such that memory to "stay put" was improved. The saccharin injection had no effect since it has minimal metabolic effects, nor did the rat get "rewarded" by tasting its sweetness. Thus, sucrose enhanced memory even when there is no contingency between its sweetness and "staying put." Further, our rat did not learn anything about the sucrose (i.e., its taste). Thus, we might say that sucrose (but not saccharin) has influenced memory directly by some metabolic process related to enhancement of the memory for behaviours and their relation to events in the environment (White & Milner, 1992).

We continue our analogy and get to the point: We will show in this thesis that, for social drinkers, alcohol can act much as the postlearning *sucrose injection* does in our laboratory rat (influencing memory by its "metabolically" active properties, not via

association with its incentive effects). In this regard, alcohol will enhance memory for neutral, positive and negative material; this will reflect a nonselective memory enhancement independent of alcohol's incentive properties. In addition, we will show that alcohol can also act much as postlearning *saccharin (or sucrose) consumption* (influencing memory contingent on associations between incentive properties and memory for behaviours and events in the environment). In this regard, incentive could mean that alcohol will enhance memory for positive material and/or inhibit memory for negative material; both are memory outcomes with face-valid incentive value.

In this introduction, we will outline alcohol's effects on memory, then alcohol's incentive effects. Theoretical issues important to the studies will be outlined, and the rationale and underlying premises and goals will be stated.

1. Changes in memory by alcohol

a. Learning and retrieval impairments by alcohol.

Before we discuss memory enhancement by alcohol, a discussion of alcohol's more familiar effects is warranted to provide some balance and perspective. Alcohol has perhaps surprisingly heterogeneous effects on the cognitive processes in normal individuals (Hashtroudi & Parker, 1986). There appear to be numerous factors that account for this, not the least of which are drug factors (timing, dose, rate, route of administration) and the particular mental task in question (Pihl, Assaad, & Bruce, in press). Acute alcohol intoxication can impair perceptual motor abilities, such as the

pursuit rotor task which requires sustained hand-eye coordination (Zacchia, Pihl, Young, & Ervin, 1991). Simple decision making may also be slower in intoxicated subjects who are required to give motor responses (Maylor & Rabbitt, 1993), and performance on divided attention tasks, those where the subject is performing a motor and a decision making task simultaneously, can also be impaired (Zacchia et al., 1991).

Psychomotor and visuospatial abilities are often compromised by alcohol (Stokes, Belger, Banich, & Taylor, 1991). Intoxication impairs visual sensitivity, or the accuracy in copying a complex visual stimuli such as the Rey figure (Peterson, Rothfleisch, Zelazo, & Pihl, 1990). Judgement of facial expressions of emotion may be impaired for some emotions (anger and disgust/contempt) but not all (e.g., fear, surprise, sadness, and happiness; Borrill, Rosen, & Summerfield, 1987). Estimation of the passage of time can be impaired by alcohol, and by conditions where intoxication is perceived (Lapp, Collins, Zywiak, & Izzo, 1994). Classification of word meaning or structure may be impaired by alcohol, and intoxication results in more target word "misses" in word recognition tasks (Maylor, Rabbitt, & Kingstone, 1987). Intoxication also impairs verbal, associative and visuospatial learning, as assessed by free recall (Peterson et al., 1990). Verbal learning may be impaired, and some feel this is because information is forgotten more rapidly, rather than alcohol merely disrupting attention (Maylor & Rabbitt, 1987). Intoxicated subjects also have difficulty remembering social events (Tucker, Vuchinich, & Schonhaut, 1987), and this may result in the loss of memory, or the production of false memories (Yuille & Tollestrup, 1990).

Also, whether the subject is intoxicated while learning, while remembering, or

both is critical. Unintoxicated subjects have difficulty recalling material learned earlier while intoxicated, and intoxicated subjects have difficulty recalling material learned earlier while unintoxicated (Goodwin, Powell, Bremer, Hoine, & Stern, 1969; Werth & Steinbach, 1991). However, subjects recall some material (words, but not faces) better learned while intoxicated if they are remembering in an intoxicated, as compared an unintoxicated, state (Goodwin et al., 1969; but cf. Werth & Steinbach, 1991). This phenomenon has been referred to as state-dependent learning, where the idea is that some memories are more accessible in similar than dissimilar states (Goodwin et al., 1969).

Interestingly, alcohol has similar inhibitory effects on recall in animal studies (e.g. Castellano & Pavone, 1988), and alcohol at intoxicating concentrations has inhibitory or preventative effects on the cellular events considered by many to be a necessary for some forms of learning (see Madison, Malenka, & Nicoll, 1991, for a review). The induction of this mechanism, long term potentiation (LTP), is prevented at small and larger doses of alcohol (Blitzer, Gil, & Laudau, 1990; Givens & McMahon, 1995; Morrisett & Swartzwelder, 1993; Wayner, Armstrong, Polan-Curtain, & Denny, 1993a,b; Zhang & Morrisett, 1993; but cf Steffensen, Yeckel, Miller, & Henriksen, 1993).

Alcohol intoxication also impairs performance on some measures of abstraction, or classification, and goal-directed planning. As measured using the Porteous Maze series, and Thurstone's word fluency, a self-directed word search task, intoxicated subjects are impaired on these abilities (Peterson et al., 1990). The degree to which alcohol modulates deficits in planning and some forms of new learning may be an important factor contributing to its use (Pihl & Bruce, 1995).

b. Cognitive abilities unaffected by alcohol.

Alcohol does not consistently impair all cognitive abilities. Abilities apparently not always sensitive to alcohol's effects include the performance on the Information, Vocabulary and Digit Symbol subtests of the Wechsler Adult Intelligence Scale - Revised (Peterson et al., 1990; but cf. Nelson, McSpadden, Fromme, & Marlatt, 1986). Reaction time is not consistently impaired by alcohol (Peterson et al., 1990; Salame, 1991), and nor are planning and working memory, as indicated by tests including the Wisconsin Card Sorting Task, and easy paired associate learning (Nilsson, Backman, & Karlsson, 1989; Peterson et al., 1990).

Emotional recognition and classification from slides of human faces is not always impaired by alcohol (Baribeau, Braun, & Dube, 1986). And although alcohol consumed *prior to* exposure may reduce reactivity to negative and positive emotional slides, it does not appear to affect conditioning of the emotional reactions to other stimuli (Stritzke, Patrick, & Lang, 1995); these results are quite distinct from the effects of alcohol consumed *after* exposure to emotional material as will be shown in this thesis.

Alcohol intoxication does not appear to affect implicit memory either (Lister, Gorenstein, Risher-Flowers, Weingartner, & Eckardt, 1991; Nilsson et al., 1989). Implicit memory is inferred from performance where memory is assessed less directly, and does not require the subject to have "conscious" awareness of the procedure. Finally, visually-presented digit memory is not consistently affected, and neither is picture recognition memory (Roache, Cherek, Bennett, & Schenkler, 1993).

Some mediators and moderators of the degree of alcohol-induced performance

inhibition include simultaneous food ingestion (Millar, Hammersley, & Finnigan, 1992), specifically sucrose (Zacchia et al., 1991) and tryptophan (Westrick, Shapiro, Nathan, & Brick, 1988). Other substances can also moderate the degree of alcohol-induced impairment (Brioni, McGaugh, & Izquierdo, 1989; Castellano & Pavone, 1988; Castellano & Populin. 1990). Dose of alcohol is also important (Jubis, 1986, 1990), with some studies finding memory facilitation at mild intoxication, but impairment at moderate-to-heavy doses. Intelligence (Maylor, Rabbitt, James, & Kerr, 1990), gender (Haut, Beckwith, Petros, & Russell, 1989), task repetition (Rumbold & White, 1987), and environment (Babbini, Jones, & Alkana, 1991; Colbern, Sharek, & Zimmermann, 1986; Miles, Porter, & Jones, 1986) also moderate. Time since drinking is also important as alcohol-induced impairment of immediate (less than 30 seconds delay) verbal memory is more pronounced on the ascending limb of the blood-alcohol curve than at comparable blood ethanol levels on the descending limb; short-, and long-term memory is equally disrupted at both times (30 seconds, and 15 minutes delay respectively; Jones, 1973).

Thus, the effects of alcohol on memory are not universally impairing, and it has been proposed that any such impairments may actually instead result secondarily from effects of the drug on other mental processes such as mood, arousal, perception attention or concentration (Jones, 1973; Maylor & Rabbitt, 1987; Weingartner, Eckardt, Molchan, & Sunderland, 1992). Indeed, since some studies have shown alcohol to actually enhance consolidation of memories, the role of alcohol in forgetting may be more indirect than previously thought. We will consider the evidence from animal studies first.

c. Memory abilities (consolidation) enhanced by alcohol: Animal studies

Much as a sucrose injection, when alcohol is administered to an animal following the learning of a task (i.e., posttraining), memory performance is improved when the animal is tested again hours or days later. Posttraining alcohol enhances passive avoidance learning (Alkana & Parker, 1979) and spatial learning (Melia, Ehlers, LeBrun, & Koob, 1986) in rats.

As described, LTP is a cellular model of learning. Once cells have been potentiated, the information "encoding" component of the model is in place. If an event influences already-induced long term potentiation, perhaps that would demonstrate an effect of consolidation or modulation on the "memory trace." Unfortunately, there is only one study available that explicitly examined the effects of posttraining alcohol on the expression of (already-induced) LTP, and no effect was found (Givens & McMahon, 1995). More research is needed in this area to determine the parameters required for alcohol to affect the expression of LTP.

In addition to sucrose and alcohol, many other compounds and events improve memory when administered posttraining in animals (Izquierdo, 1989). Mention of some of them is warranted since some also are known to have subjectively desirable effects, some are known to have undesirable effects, and others are known to have neither effect. Thus, the relationship between desirable effects and posttraining memory effects of events and drugs suggests that no contingency between the posttraining drug/event and the memory task is required for memory improvement. Instead, changes in metabolic activity may be responsible.

Briefly, some of the posttraining events that improve memory include electric shock to the foot (Netto, Siegfried, & Izquierdo, 1987; Rawlins, 1986; Welsh & Gold, 1985; White & Legree, 1984), prevention of paradoxical sleep (Marti-Nicolovius, Portell-Cortes, & Morgado-Bernal, 1988) or influencing REM sleep (Guerrien, Dujardin, Mandai, & Sockeel, 1989). Postlearning intracranial electrical stimulation enhances memory (Berman & Kesner, 1976; Coulombe & White, 1980, 1982; Huston, Mondadori, & Waser, 1974; Major & White, 1978; Mondadori, Ornstein, Waser, & Huston, 1976; Mueller, Huston, & Mondadori, 1977; Segura-Torres, Capdevila-Ortis, Marti-Nicolovius, & Morgado-Bernal, 1988; Segura-Torres, Portell-Cortes, & Morgado-Bernal, 1991), as do eating lab chow, and immersion in ice water (Mondadori, Waser, & Huston, 1977).

Drug approaches also improve memory. Posttraining administration of certain hormones (Flood, Smith, & Morley, 1987; Gold, 1989; Gold & Van-Buskirk, 1976; Hock & McGaugh, 1985; Izquierdo, Dalmaz, Dias, & Godoy, 1988; Izquierdo & Pereira, 1989; McGaugh, 1989; Mondadori, Ducret, & Borkowski, 1991; Randt, Judge, Bonnet, & Quartermain, 1982), protein synthesis inhibitors (Benloucif, Mortimer, Bennett, & Rosenzweig, 1990), neuropeptides (Aguiar & Tomaz, 1990; Flood, Hernandez, & Morley, 1987; Hasenohrl, Gerhardt, & Huston, 1990; Huston, Hasenohrl, Boix, Gerhardt, & Schwarting, 1993; Huston & Oitzl, 1989; Huston & Staubli, 1979; Kafetzopoulos, Holzhauer, & Huston, 1986; Tomaz & Huston, 1986), and glucose (Gold, 1986, 1987; Hall, Gonder-Frederick, Chewning, Silveira, & Gold, 1989; Lee, Graham, & Gold, 1988; Messier & Destrade, 1988; Messier, Durkin, Mrabet, & Destrade, 1990; Prado de Carvalho, Vendite, & Izquierdo, 1978; Messier & White, 1987; Stone, Rudd, & Gold,

1990; Wenk, 1989) increase memory. Further, posttraining administration of drugs known specifically to increase cholinergic (Flood & Cherkin, 1987; Packard, Regenold, Quirion, & White, 1990; Santucci, Kanof, & Haroutunian, 1989), noradrenergic (Fulginiti, Molina, & Orsingher, 1976; Gold & Van-Buskirk, 1975; Guaza, Borrell, & Borrell, 1986; Stein, Belluzzi, & Wise, 1975), serotonergic (Altman & Normile, 1987; Flood & Cherkin, 1987; Normile & Altman, 1988), and dopaminergic (Acker, Jacobson, & Lishman, 1987; Altman & Quartermain, 1983; Castellano, 1974; Haycock, Van-Buskirk, & Gold, 1977; Introini-Collison & McGaugh, 1989; Janak, Keppel, & Martinez, 1992; Martinez, Jensen, Messing, Vasquez, & Soumireu-Mourat, 1980; Oscos, Martinez, & McGaugh, 1988; Strupp, Bunsey, Levitsky, & Kesler, 1991; Weinberger, Riedel, Janak, & Martinez, 1992; White, 1988) neurotransmission all can improve memory. Postlearning administration of drugs that modulate GABAergic (Brioni & McGaugh, 1988; Brioni, Nagahara, & McGaugh, 1989; Castellano, Brioni, Nagahara, & McGaugh, 1989; Castellano & McGaugh, 1989a; da Cunha *et al.*, 1991; Izquierdo & Cardoso-Ferreira, 1989; Izquierdo, Cunha, & Medina, 1990; Katz & Liebler, 1978; Swartzwelder, Tilson, McLamb, & Wilson, 1987; Weingartner, Sirocco, Curran, & Wolkowitz, 1995) and opioidergic (Canli, Cook, & Miczek, 1990; Castellano, Introini-Collison, Pavone, & McGaugh, 1989; Castellano, Libri, & Ammassari-Teule, 1988; Castellano & McGaugh, 1989b; Castellano & Pavone, 1983; Izquierdo & Graudenz, 1980; Izquierdo & Netto, 1990; Maisto, Connors, Tucker, & McCollam, 1980; McDaniel, Mundy, & Tilson, 1990; Schulteis & Martinez, 1992; Staubli & Huston, 1980; Tomaz, Aguiar, & Nogueira, 1990) function can also influence memory.

Which mechanism(s) may be involved in memory enhancement that are common to these drugs and events is a controversial subject, with either somatic (peripheral or brain) glucose or norepinepherine utilization being most implicated. A more parsimonious explanation may be an enhancement, promotion or prolongation of the metabolic or cellular events underlying memory consolidation (Gold, 1989; Landauer, 1969; Pfaff, 1969; White & Milner, 1992). In this sense, memory is viewed much as it was by Hebb (1949) where increases in "reverberatory" cellular activity reflected memory processes. Importantly, the incentive properties of the drugs/events are not implicated in enhancing the reverberatory activity.

d. Memory abilities (consolidation) enhanced by alcohol: Human studies

The claim that posttraining administration of alcohol can "stimulate" memory is perhaps surprising. Nonetheless, at the time of writing this thesis, there were eight published reports of primary relevance in the area documenting this effect. These papers will now be reviewed in some detail, in the order in which they were published.

Perhaps the **first** report to investigate the effects of alcohol on consolidation of pre-existing memories was by Kalin (1964) at Harvard University. Kalin tested male undergraduates while they attended fraternity parties. Participants wrote stories in response to the Thematic Apperception Test (TAT) at three points during the parties. At one party the participants drank beer ad libitum (data were collected for 18 of those men, called the "wet" group) while men at another party drank nonalcoholic beverages (data were collected for 17 of those men, called the "dry" group). At Time 1, the men were shown

a few TAT cards, and they gave responses before any beverages had been consumed. They then started drinking. At Time 2, 25 minutes later, participants again were shown a few (different) cards and gave responses, then drank more. Finally, 25 minutes later at Time 3, they again were shown cards, gave responses and then drank again. Kalin went back to the fraternities the next day and recruited the men to come back for more study. The men were given surprise memory tests. They were asked to recall as much of their TAT responses (stories) from the previous day as they could. As he expected, Kalin found that the dry group had better recall of material written at Time 3. There were no surprises here; alcohol intoxication inhibited attention to, rehearsal or learning of (etc.) responses made while "under the influence". In addition, alcohol did not increase subjects ability to recall the TAT cards, nor improve word-for-word exactness of the recall of their TAT responses at Time 1 or Time 2. However, the wet group recalled slightly more material written at Time 1 (about 5 percent more). Although the group difference was not statistically different, Kalin found that the amount a subject had drunk at Time 1 (i.e., right after the first responses) was highly correlated with subsequent recall (r=.57, p <.02). Kalin concluded there might be a relationship between the amount drunk following learning, and improvements in subsequent recall.

Kalin's study suffers from many easily-detected limitations in design (uncontrolled environment, uncontrolled doses, etc.) and applicability (representativeness of sample). However it is an important study because it led Kalin to raise the question as to whether alcohol might actually improve memory if consumed after learning. He also asked whether alcohol might improve memory indirectly (perhaps by reducing subsequent

interference), and/or whether individual differences in participants might be implicated in the effect (i.e., did those who drank more also have better memory skills, or did their increased drinking improve their memory by removing more interference)?

The **second** study in the area was conducted some sixteen years later by Parker, Birnbaum, Weingartner, Hartley, Stillman, and Wyatt (1980) at the U.S. National Institute of Mental Health. This experiment was the first in humans to examine the memory effects of consumption of alcohol only *after* learning. It was thus the first designed specifically to target the effects of alcohol on memory consolidation. In a two-part incidental learning experiment, Parker and her colleagues showed that posttraining consumption of alcohol facilitated recognition of photographic slides, as well as free- and cued recall for word lists. In the first experiment, 16 healthy men recruited from the community viewed slides then consumed 1.0 ml/kg alcohol. In a different session, the same men consumed a placebo. Recognition memory for the slides was tested unexpectedly 3 hours later. Alcohol enhanced memory relative to placebo by 12 percent ($p<.05$). In the second part of the experiment, a sample of 72 healthy men from the community consumed 1.0 ml/kg alcohol or placebo following the presentation of word lists. Memory was tested unexpectedly 24 hours later. Alcohol enhanced free recall by 30 percent ($p<.05$) and cued recall by 15 percent ($p<.02$) relative to placebo. This study was critical in that Parker et al. showed that alcohol could enhance memory if consumed only posttraining, and because it was the first to speculate that alcohol might improve memory by stimulating consolidation instead of (or in addition to) reducing retroactive interference.

The **third** study was also conducted by Parker and her colleagues (Parker,

Morihisa, Wyatt, Schwartz, Weingartner, & Stillman, 1981) as a follow-up. The goal was to demonstrate a dose-response effect. Sixteen healthy male subjects recruited from a university newspaper participated in four experimental conditions spaced by 1 week where they consumed placebo or 0.25-, 0.5- or 1.0 ml/kg alcohol. Drink order was counterbalanced. Participants viewed slides, then consumed the beverage. Memory was tested 7 hours later in each session. Participants may have therefore expected memory to be tested 7 hours later in at least the second, third and fourth sessions. In this sense, the learning paradigm would likely have been a mix of intentional and incidental.

Parker and colleagues found that 0.5- and 1.0 ml/kg alcohol increased slide recognition (by 11 and 12 percent respectively) relative to placebo; the 0.25 ml/kg dose did not increase recognition significantly. The dose-response effect was somewhat supported, but the increases in memory for 0.5 ml/kg and 1.0 ml/kg doses did not differ from each other despite the fact that the 1.0 ml/kg resulted in greater breath alcohol concentrations and a greater subjective "high" than the 0.5 ml/kg dose. Parker and colleagues then raised the question of the relationship between subjective "high" effects and memory facilitation, and speculated in two subsequent papers (Esposito, Parker, & Weingartner, 1984; Parker & Weingartner, 1984) that the two were isomorphic processes reflecting brain stimulation and "reinforcement" from alcohol. If so, it would be necessary to clarify why the 0.5 and 1.0 ml/kg groups had equal memory improvement despite unequal subjective highs. Nonetheless, Parker's work is the most central of these studies in that she raised the question of whether alcohol enhanced memory and if so, were alcohol's incentive effects implicated as well. As we will see, whether incentive is

implicated appears to depend on the learning paradigm.

The **fourth** study was by Mueller, Lisman, and Spear (1983) at the State University of New York. That paper represented an attempt to disprove that Parker's notion that alcohol enhanced consolidation. Meuller and colleagues set out to demonstrate that the memory improvement effect was instead due to reduction of retrograde interference by alcohol. The authors cited three premises as central to testing the underlying mechanism. The first was a manipulation in temporal delay between presentation of the materials and subsequent alcohol consumption. The authors argued that while enhancement of materials presented more near in time to the consumption might favour a consolidation view, interference reduction would apply equally regardless of interval. Temporal delay was manipulated by presenting two word lists before alcohol consumption. The second was the amount of list "processing" or memory rehearsal prior to drink consumption. The authors argued that while enhancement of memories processed "more deeply" might favour a consolidation view (if somehow increased rehearsal reflected or required increased consolidation), interference reduction would apply equally regardless of rehearsal depth. Type of rehearsal of the words was manipulated such that participants were asked to repeat the word, or to use it in a sentence, or were asked to count backwards in a numerical distraction task. For the third premise, the authors argued that the consolidation view would predict enhancement whether tested by recall or by recognition, while the interference-reduction view would favour enhancement tested by recall, but not recognition. (Postman & Underwood, 1973, found that retroactive interference affects recall more so than recognition, thus there should be more reduction

of this interference by alcohol when tested by recall).

An intentional learning paradigm was employed in the acquisition of word lists. Thirty-six male and female undergraduates, described as moderate-to-heavy social drinkers participated. As with Kalin's (1964) study, participants learned (word lists in this case) both before and after consuming alcohol (1.0 ml/kg here), or placebo. The results were that alcohol uniformly increased recall (by 46 percent, $p<.05$) regardless of temporal delay. The authors suggest this supports the interference reduction view, but it could easily be argued that both lists were still being equally "consolidated." First, memory consolidation can take hours (Mondadori, Ducret, & Borkowski, 1991). Second, memory is lost gradually over the hours after acquisition (Hart & O'Shanick, 1993) not merely in seconds or minutes. Third, posttraining events up to hours later can influences subsequent recall (Izquierdo & Chaves, 1988).

Mueller et al.'s results also showed that alcohol enhanced memory across rehearsal type. Again, the authors argue this favours the interference reduction view, but it can easily be argued that memory consolidation is not a unitary phenomenon, and that enhanced consolidation could occur regardless of rehearsal type. Finally, the results showed that alcohol significantly enhanced recall, but not recognition. The authors argue this favours the interference reduction view, but as will be shown in short order, alcohol can enhance recognition under some conditions, and it does not protect against interference in all tasks.

The **fifth** study was by Mann, Cho-Young, and Vogel-Sprott (1984). Using a paradigm where learning of word lists occurred both before and after consumption of

alcohol (0.66 g/kg; n=18) or placebo (n=14), the authors found that alcohol enhanced memory for words presented before consumption, and alcohol impaired memory for words presented after consumption. As with Mueller et al., (1983), serial position of the words did not matter; alcohol enhanced memory for all words. In addition, both posttraining recall (64 percent, $p<.05$) and recognition (100 percent, $p<.05$) were enhanced. Mann and colleagues argued that the memory improvement may have occurred via interference reduction, enhanced consolidation, or by alcohol's "rewarding" effects.

The **sixth** study showed some of the outer boundaries of the postlearning paradigm. In a study of 96 male undergraduates, Lamberty, Beckwith, Petros, and Ross (1990) showed that while alcohol (1.0 ml/kg) enhanced incidental recall for prose narratives (4.6 percent, $p<.05$), intentional recall and recognition for word lists was not improved. This suggested that memory for increasingly context-rich ideas was more reliably enhanced than was memory for simple, unrelated words. A more interesting finding than the previous studies, to be sure. Additionally, Lamberty and colleagues found a positive relationship ($r=.18$, $p<.05$) between WAIS-R Vocabulary scores and prose recall; participants with better word knowledge had higher recall scores following drinking. There was thus some preliminary evidence that, as proposed by Kalin, the role of individual participant factors should be investigated more fully. And, as with the fifth study just previously discussed, Lamberty and colleagues argued that the memory improvement may have occurred via interference reduction, enhanced consolidation, or by alcohol's "rewarding" effects. The question of mechanism was still unresolved.

The **seventh** study by Tyson and Schirmuly (1994) is perhaps the most

confounding of the eight reports. Participants were male social drinkers. As with the Mueller et al., (1983) paper, the authors set out to demonstrate that the memory facilitation effect was a secondary effect of reduction of retrograde interference (not a result of enhanced consolidation). Using an extremely complex paradigm, the authors tested the effects of alcohol (0.8 ml/kg; n=10) or placebo (n=10) on incidental and intentional memory for word lists. Five subjects in each beverage group learned a list of 25 words. Twenty minutes later, they participated in an incidental learning task. Twenty minutes after that, they drank. Two hours later, they did another incidental task. Two hours after that, memory for the intentional and incidental tasks was assessed.

The other five subjects in each beverage group first did the incidental task. Twenty minutes later, they did the intentional task. Immediately afterward, they drank. Two hours later, they did another incidental task. Two hours after that, memory for the intentional and incidental tasks was assessed.

The results showed that alcohol enhanced intentional recall (9 percent, $p<.05$) but not intentional recognition. As with the Mueller et al. paper, the authors argue the recall vs. recognition results support the view that alcohol acted via reducing interference. However, as with the Meuller et al. paper, we argue the results do not convincingly rule out the consolidation view. Results also showed that incidental recall for material learned prior to drinking was enhanced (54 percent, $p<.03$) while incidental recall for material learned after drinking was inhibited. The authors also argue this is more consistent with the reduction of interference view than the consolidation view, but this is difficult to reconcile. There was no "no interference" control, so whether alcohol acted by reducing

interference (in either the incidental or intentional memory tasks) is impossible to prove.

Furthermore, the **eighth** experiment, (Hewitt, Holder, & Laird, 1996), showed more conclusively the effect of a controlled interfering task. This experiment showed clearly that alcohol did not protect against interference. In the first part of the experiment, 80 participants learned a visual motor task and drank alcohol (.19 ml/kg; n=25) or placebo (n=25). Alcohol decreased size of errors in the location of components of the task the next day (by 139 percent, $p<.05$). That is, alcohol enhanced memory when there was no interference task. However, in the second experiment different participants (alcohol .19 ml/kg, n=15; placebo, n=15) were tested this time with an interference task given after drinking. Here, alcohol did not facilitate memory. That is, alcohol did not protect against retrograde interference. Together, the results suggest to Hewitt et al. that alcohol acts on consolidation, not by protecting against interference.

e. Implications and future directions.

Overall, the memory enhancement effect seems fairly consistent in humans. Four primary mechanisms of action have been hypothesized for the memory improvements following posttraining alcohol, but there remain few formal "theories" (except for Esposito et al., 1984), and little hard evidence one way or the other. Thus, when designing the experiments in this thesis, there was still the opportunity to examine whether individual differences (in personality, intelligence, etc), or and/or enhanced consolidation, and/or alcohol's incentive effects, and/or reduction of interference were implicated.

There are vast individual differences in memory performance and response to

alcohol. Further, we have been documenting the memory-enhancement and learning-impairment effects of alcohol in both animals and in humans. What is needed next is to determine whether alcohol has incentive effects, and whether and when these relate to memory. We begin with the incentive effects.

2. Alcohol's subjective and physical effects with incentive.

Rodents (June *et al.,* 1992; Myers & Quarfordt, 1991; Reid, Hunter, Beaman, & Hubbell, 1985; Samson, Tolliver, Lumeng, & Li, 1989; Volpicelli, Ulm, & Hopson, 1991), monkeys (Crowley, Williams, & Jones, 1990; Ervin, Palmour, Young, Guzman-Flores and Juarez, 1990; Grant & Johanson, 1988), and people (Mello, Mendelson, Palmieri, Lex, & Teoh, 1990; Rumbold & White, 1987) can all learn to drink appreciable amounts of alcohol. This fact alone leads to the question of "why". Perhaps alcohol is consumed because it produces desirable effects? Does alcohol "incentive" include reduction of undesired states, and induction/enhancement of desired states? Yes, for many, it does.

a. Animal studies

As mentioned, some animals will work for and consume alcohol to the point of intoxication. Whether animals drink alcohol mainly to relieve negative events (White, 1996), or possibly to induce positive events (Wise & Bozarth, 1987), neither, or both are still open questions. Answering these questions is relevant in that it may help demonstrate that alcohol's incentive effects are separable from other drug effects, including alcohol's

effects on conditioning.

It is virtually impossible to comprehensively summarize the animal literature on incentive and motivation in one thesis. The definitions of incentive, motivation, drive, etc. are controversial at best, and woefully confusing at worst. "Motivation" is still among the most actively researched areas in behavioral neuroscience. However, for the purposes of this thesis it will be noted that two properties of alcohol linked to motivation and incentive are "relief" and "reward."

For example, the concept of "relief" as applied to alcohol relates to the idea that animals may consume alcohol in part to the degree that alcohol dampens their heightened responses to ongoing physical pain (e.g., Lewis, 1990) and conditioned cues for this pain, or for hunger (Stewart, Gatto, Lumeng, Li, & Murphy, 1993; Volpicelli & Ulm, 1990). Notably, some investigators have discovered that pain relief (analgesia) is related to stimulation of the VTA. Since the VTA is activated by stimulants such as amphetamine etc, this may explain the stimulant abuse potential of analgesics such as morphine or alcohol (Franklin, 1989; Wise, 1988). Relief appears to require intact functioning of the endogenous opiate system while conditioning of relief to other stimuli may require intact functioning of GABAergic systems likely associated with limbic brain areas (reviewed in Pihl & Peterson, 1995). That alcohol can be a "conditioned reliever" or anxiolytic is a commonly held view. We do not dispute this view per se. Rather, we see it as part of the story. The other part of the story, reward and conditioned reward may be unfamiliar as applied to alcohol. These will therefore be discussed in some detail.

By contrast to relief, "reward" refers to induction or prolongation of desirable

effects. It is defined in animals by elicitation of *approach behaviours* (White, 1989). Some investigators infer that the human equivalent is subjectively desirable internal states (White, 1996); this will be discussed more fully in a later section. Although GABAergic (Rassnick, D'Amico, Riley, Pulvirenti, Zieglgansberger, & Koob, 1992), serotonergic (Pihl & Peterson, 1995) and opioidergic (White, 1989) systems may be involved here, most evidence points to the idea that reward requires brain activation the VTA connections with the nucleus accumbens (White, 1989), most notably release and turnover of the neurotransmitter dopamine in those areas (Bardo, Bowling, Robinet, Rowlett, Lacy, & Mattingly, 1993; Beninger, 1991; Fibiger, 1978; Fibiger & Philips, 1988; German & Bowden, 1974; Nakajima, 1989; Phillips, Pfaus, & Blaha, 1993). Reward was inferred by the "discovery" in 1954 of the tendency of rats to repeatedly bar press for (presumably desirable) electrical stimulation of the brain in certain areas (see Milner, 1989, for a discussion). Later studies confirmed the involvement of VTA and nucleus accumbens dopaminergic neurons in this effect (see Bielajew & Harris, 1991, for a review of recent studies).

Does alcohol elicit approach behaviours? Does alcohol stimulate activity in "reward" centres? If so, is that in part why animals drink it? Again, it seems possible. Alcohol affects open-field locomotion and exploration (i.e., approach) in a biphasic (stimulant/increase then sedative/decrease) manner in mice (Cunningham, Niehus, Malott, & Prather, 1992; Cunningham, Niehaus, & Noble, 1993) and rats (Gill, France, & Amit, 1996; Lewis & June, 1990). In addition, many rats will learn to approach and interact with cues (i.e., to bar press) for access to drink (Ritz, George, deFiebre, & Meisch, 1986;

Samson, Pfeffer, & Tolliver, 1988; Sinclair, 1974) and receive alcohol intravenously (Amit & Stern, 1969; Lewis, 1990). Further, injected or self-administered alcohol can interact with electrical brain stimulation reward (Bain & Kornetsky, 1989; De Witte & Gewiss, 1986; Lewis & June, 1990; Kornetsky, Bain, Unterwald, & Lewis, 1988; but cf., Routtenberg, 1981). Alcohol thus has observable incentive effects in animals.

Alcohol stimulates physiological responses of dopamine-releasing cells in the living intact rat brain (VTA or nucleus accumbens). This occurs whether alcohol is consumed orally (Weiss, Lorang, Bloom, & Koob, 1993), administered systemically (Criado, Lee, Berg, & Henriksen, 1995; Gessa, Muntoni, Collu, Vargiu, & Mereu, 1985; Yoshimoto, McBride, Lumeng, & Li, 1991) or injected directly in to those areas (Yoshimoto et al., 1991). Responding of these brain sites is also enhanced by alcohol in isolated, in vitro cell populations (Brodie, Shefner, & Dunwiddie, 1990).

Sober alcohol-preferring rats, who drink larger quantities of alcohol than other rats, have lower levels of dopamine in nucleus accumbens and striatum, but not other areas (Gongwer, Murphy, McBride, Lumeng, & Li, 1989; McBride, Murphy, Lumeng, & Li, 1990). Similarly, acute alcohol administration causes release (Khatib, Murphy, & McBride, 1988; Wozniak, Pert, Mele, & Linnoila, 1991; Yoshimoto & Komura, 1993; Yoshimoto et al., 1991) and turnover (Fadda, Mosca, Colombo, & Gessa, 1990; McBride, Murphy, Lumeng, & Li, 1990; Reggiani, Barbaccia, Spano, & Trabucchi, 1980) of dopamine from VTA and nucleus accumbens neurons as well. The degree to which this occurs relates to cocaine-induced dopamine release in the same animal (Weiss, Hurd, Ungerstedt, Markou, Plotsky, & Koob, 1992). Moreover, acute and chronic alcohol

consumption increases dopamine (and other) metabolite formation in cerebrospinal fluid in monkeys (Ervin et al., 1990).

Chronic alcohol consumption (more than 10 days) affects properties of dopamine receptors in reward areas more so than other brain areas (Hietala, Salonen, Lappilainen, & Syvalahti, 1990; Lograno *et al.*, 1993; Muller, Britton, & Seeman, 1980; Pellegrino & Druse, 1992). The degree to which this occurs reflects the animal's preference for alcohol (Murphy, McBride, Gatto, Lumeng, & Li, 1988). Chronic alcohol consumption also decreases levels of plasma dopamine (Patel & Pohorecky, 1989).

Systemic (Koob & Weiss, 1990; McBride, Murphy, Lumeng, & Li, 1990; Samson, Tolliver, & Schwartz-Stevens, 1990; Weiss, Mitchener, Bloom, & Koob, 1990) and focal (into nucleus accumbens; Samson, Hodge, Tolliver, & Haraguchi, 1993) administration of dopaminergic agonists have been shown to decrease alcohol consumption. Dopaminergic agonists also interact with alcohol-induced locomotor (approach behaviour), modulating alcohol's effect here (Lograno *et al.*, 1993).

Further, systemic administration of dopaminergic antagonists has effects on consumption in some (Amit, Gill, & Ng Cheong Ton, 1991; Hubbell, Marglin, Spitalnik, Abelson, Wild, & Reid, 1991) but not all cases in rats (Linseman, 1990; Nixon & Bowlby, 1996). Injection of dopamine metabolites into the VTA increases consumption in rats (Duncan & Fernando, 1991).

Dopamine-specific destruction of nucleus accumbens has been shown to increase (Quarfordt, Kalmus, & Myers, 1991) or not affect (Lyness & Smith, 1992; Rassnick, Stinus, & Koob, 1993) alcohol consumption. In alcohol-preferring rats,

intracerebroventricular administration of 6-OHDA, a dopamine cell toxin, reduces alcohol consumption (Myers & Melchior, 1975; cited in Ollat, Parvez, & Parvez, 1988). Systemic administration of dopaminergic but not noradrenergic antagonists causes dose-dependent inhibition of alcohol-induced open field approach behaviours at doses that do not directly affect open field behaviour themselves (Strombom, Svensson, & Carlsson, 1977).

Alcohol induces tachycardia in rats. This effect is linked with catecholamine and beta-endorphin response, and sympathetic activation (Peris & Cunningham, 1986). Sympathetic response to alcohol in animals relates strongly to response to other drugs like cocaine and opiates (George, 1993). A discussion of alcohol's tachycardic effects in humans is presented shortly. In sum, we have presented evidence that alcohol indeed appears to have reward effects, and these may be mediated in part by activity in the VTA and nucleus accumbens.

Do alcohol's incentive effects condition neutral stimuli in the long term? As with relief, reward may also be conditioned to neutral stimuli. In this sense, conditioned reward would represent the organism's preparation for (i.e., anticipation of) stimuli with primary desired value such as food, willing sexual partners, etc. and is thought to reflect dopaminergic activity (Phillips, Pfaus, & Blaha, 1993; Pihl & Peterson, 1995). Certain investigators (e.g., White, 1996) argue that the standard paradigm for assessing "reward" in drug studies is the conditioned cue/place preference paradigm, or CPP. In the CPP, an animal is administered a drug then placed (or consumes the drug) in one of two chambers. Saline is paired with the other chamber. The next day, the animal is allowed to freely roam between and within the two chambers. If the animal stays longer in the side paired

with the drug, the experimenter infers that the drug effects were "desirable". If the animal stays longer in the other side, the drug is inferred to have had aversive effects, and if equal time is spent in both chambers, the drug had neither effect.

This procedure does not just measure unconditioned pleasurable effects. Inferring such events is difficult in an animal model. Instead, it can easily be seen that there are two components to the CPP task. First, the animal must experience the effects of the drug (if any), and the animal must associate those effects with a specific chamber. (What is measured is the preference for the chamber.) Failure of one of these processes may falsely imply failure of the other. This is not trivial since animals may vary in the degree to which they respond to the effects of alcohol, and they may vary in the degree to which they associate them with a chamber. This stated, what are the CPP findings?

In mice, alcohol produces a CPP (Cunningham & Prather, 1996), and the degree to which this occurs is related to the degree to which alcohol increases open-field approach behaviour (Cunningham, Niehus, Malott, & Prather, 1992; Cunningham, Niehaus, & Noble, 1993). However, dopaminergic antagonists do not block the mouse CPP (Cunningham et al., 1992), leading White (1996) to conclude that this CPP is not mediated by dopamine. White (1996) states that reward is operationalized by the CPP paradigm, and that the CPP depends on intact dopamine (and perhaps opiate) function. Are alcohol induced CPPs in mice therefore opioidergic? Perhaps, but importantly alcohol's rewarding and conditioned rewarding effects might be mediated in part via a dopaminergic-opiate link (DeNoble, Mele, & Porter, 1985; Di Chiara, Acquas, & Tanda, 1996; Gianoulakis, 1996).

There are other explanations as to why dopaminergic antagonists had no effect in the Cunningham et al., (1992) paper. First, the lack of effect may have resulted because of the mouse strain used, DBA/2J. This strain known to be somewhat less sensitive to alcohol's incentive effects than other strains. If so, the dopaminergic component, including response to antagonists, may be different. Second, in general dopamine agonists are more implicated in reducing alcohol's effects than are dopamine antagonists. Thus, the lack of involvement of dopamine antagonists in the CPP would not be surprising. In sum, it does not rule out dopaminergic mechanisms in alcohol incentive in mice.

The alcohol CPP in rats is different. Alcohol can induce a CPP in some (e.g., Black, Albiniak, Davis, & Schumpert, 1973), but not all rats (e.g., Cunningham et al., 1993; Lai, Carino, & Horita, 1980). Other experimenters have found neither conditioned preference nor aversion (Asin, Wirtshafter, & Tabakoff, 1985) with injections in alcohol-naive rats, but did find preference in the same animals with amphetamine.

Further, several experiments have shown alcohol injections to produce a conditioned place aversion to alcohol in alcohol-naive rats, (e.g., Cunningham, 1979, 1981; Sherman, Hicks, Rice, Rusniak, & Garcia, 1983). Oral alcohol can produce place aversions (Stewart & Grupp, 1986), even while blood alcohol levels were still rising (e.g., Stewart & Grupp, 1989). This is indeed confusing since the rats in these studies were not water deprived, and had a history of consuming alcohol freely. Another possibility is that absolute dose delivered may mediate incentive effects in rats, in addition to experience (van der Kooy, O'Shaughnessy, Mucha, & Kalant, 1983).

In rats, previous exposure to alcohol may be required to develop the CPP (Stewart,

Perlanski, & Grupp, 1988), either to overcome taste (Reid, Hunter, Beaman, & Hubbell, 1985) or novel experience constraints related to systemic injections (Black et al., 1973; Stewart & Grupp, 1985). This suggests that in mice, and some rats, alcohol's reward or relief properties can be conditioned. As we will discuss shortly, that prior experience with alcohol may increase its conditioned motivational properties is highly interesting: Dopamine-mediated conditioned incentive seems important in determining an individuals response to alcohol (Pihl and Peterson, 1995). The same is, of course, true for people: Some find alcohol aversive, others neutral and still others find it pleasurable.

Some (e.g., White, 1996) argue that conditioned incentive from alcohol is unsupported. However, we have shown that alcohol interacts with incentive reward and relief systems. White argues that conditioning of incentive requires the amygdala, and possibly, separately, the hippocampus, If the CPP evidence points away from the amygdala, this is no great tragedy. The alcohol-induced effect on VTA neurons also influences hippocampal afferent connections, suggesting a role for alcohol in hippocampally-mediated incentive reward conditioning (e.g., Criado, Steffensen, & Henriksen, 1994). Further, animals with superior hippocampal functioning (Amit & Smith, 1992) consume large amounts of alcohol voluntarily. Do these animals drink because they have excellent capacity to associate alcohol with events in the environment? If so, to what events? Do these animals drink only to relieve stress? If yes, why do they consume alcohol spontaneously? In other words, perhaps there is a role for hippocampally-mediated conditioned incentive reward, in addition to relief.

Indeed, recent animal theories of alcoholism have dopamine mediated reward as

a central hypothesis (Fibiger, 1978; George & Ritz, 1993; Koob & Weiss, 1990; Ollat, Parvez, & Parvez, 1988; Wise & Bozarth, 1987) while others conclude that dopamine is not involved (Amit & Brown, 1982; White, 1996).

Much of the animal literature that models alcohol's effects in humans has been criticized on grounds of methodological weakness and limited applicability to humans (Tipton, 1988), or as being overly reductionistic (Crabbe, Feller, Terdal, & Merrill, 1990; Kalant, 1990). Clearly, care must be taken in any animal model to ensure a clearly established behavioural or phenomenological equivalent exists in the species (humans) being modeled. Models developed in species who do not drink alcohol or have had little prior experience with it have limited applicability in describing repeated alcohol use in people (Stewart, Perlanski, & Grupp, 1988). In this regard, mice or rats with demonstrated preference for alcohol (Cunningham et al., 1993), as well as adequate associative memory, may serve as better models.

b. Human studies: Subjective incentive effects

As discussed, most humans consume alcohol and many will become intoxicated at some point in their lives. What is the subjective effect of this? What is the physiological effect? People have subjective *beliefs* that alcohol will have both untoward and desirable effects (Earleywine, 1994a,b; Earleywine & Martin, 1993; Pihl & Smith, 1993). The belief that alcohol will produce desirable effects is much more predictive of real-world alcohol consumption than the belief that alcohol will have untoward effects (Christiansen, Smith, Roehling, & Goldman, 1989; Goldman, 1994; Rather & Goldman,

1994; Rather, Goldman, Roehrich, & Brannick, 1992; Smith, Goldman, Greenbaum, & Christiansen, 1995). Thus, exploring alcohol's desirable effects will be our focus.

People's expectations largely appear to reflect what is actually experienced when individuals are intoxicated. Alcohol's desirable effects, more common when blood alcohol levels are rising and peaking, can indeed include "relief" (stress-reducing and relaxing) effects (Connors & Maisto, 1979; Freed, 1978; Josephs & Steele, 1990; Mayfield, 1968; Mayfield & Allen, 1967; Steele & Josephs, 1988; Williams, 1966). Subjective relief is more reliable if alcohol consumption precedes rather than follows the stressor (Sayette, 1993).

Alcohol's incentive effects are not limited to relief, however. In addition, there are desirable reward effects "added" by alcohol. Included here are subjective warmth/glow, "dynamic" feelings, feeling elated, excited, stimulated, talkative, up, euphoric, vigorous, energetic and friendly (Connors & Maisto, 1979; Fromme, Katz, & D'Amico, 1997; Lewis, 1990; Martin, Earleywine, Musty, Perrine, & Swift, 1993; McCaul, Turkkan, Svikis, & Bigelow, 1990).

Interestingly, desired emotional states are actually more likely to promote alcohol consumption than are undesired emotional states (e.g., Pihl & Yanofsky, 1979). However, the effects of alcohol on subjective emotion are by no means uniform or universal (Gustafson, 1987, 1991), and they depend in large part on the experimental demands and procedures (Pihl & Smith, 1983). Other indicators are needed here as well. Thus, are there physiological factors that determine or reflect subjective effects in a given environment?

c. Human studies: Physiological incentive effects

The literature on the physiological effects of alcohol is vast indeed since alcohol affects virtually all bodily systems (e.g., Loke, 1992). Relevant to alcohol's incentive effects, it is of interest to note that alcohol has demonstrated ability to dampen the psychophysiological impact of stressors and threats when it is consumed before (Finn & Pihl, 1987, 1988; Finn, Zeitouni, & Pihl, 1990; Sher & Walitzer, 1986; Stewart, Finn, & Pihl, 1992; Vogel & Netter, 1990; Zeichner, Edwards, & Cohen, 1985), but is less consistent when consumed after (Sayette, 1993) their occurrence.

As with subjective effects, alcohol's physiological incentive effects are not restricted to stress dampening (i.e., relief). Alcohol produces dose-dependent changes in electroencephalographic (EEG) activity. Alcohol-induced subjective euphoria has been found to be strongly associated with alpha wave activity while blood alcohol levels are rising, and associated with theta wave activity while blood alcohol levels are falling (Mendelson, Mello, Lukas, & Woods, 1989). These results are also related to levels of ACTH in plasma (Lukas & Mendelson, 1988). In addition, these authors have implicated the effects of alcohol on EEG alpha over the frontal cortex regions of the scalp most strongly in the euphoria effect. EEG is not the only tool to suggest brain activity is related to alcohol's desirable effects.

Brain imaging technology is among the most exciting of tools for the alcohol researcher. Although some of the tools are so new that subjective effects of alcohol have yet to be studied using them (e.g., with functional magnetic resonance spectroscopy (Spielman, Glover, Macovski, & Pfefferbaum, 1993), recent studies utilizing the more

familiar brain imaging tool positron emission tomography (with multiple doses of alcohol) confirm the earlier EEG findings. Brain activation, as measured by regional cerebral glucose utilization, is changed by alcohol in a dose-dependent fashion, and the effects are related to subjective increases in positive mood (de Wit, Metz, Wagner, & Cooper, 1990). As with the EEG work, the frontal cortex was particularly associated with the positive mood changes. Subsequent research on regional cerebral blood flow confirmed this, demonstrating that biphasic stimulant and sedative effects of alcohol are most related to changes in prefrontal cortex and temporal lobes (containing hippocampus, amygdala and related limbic and memory structures) as well as the tachycardic effects of alcohol (Sano, Wendt, Wirsen, Stenberg, Risberg, & Ingvar, 1993). The tachycardic effects of alcohol on individuals at rest deserves further consideration as it may be relevant to alcohol's incentive effects.

There is no proven "average" response to alcohol (Reed, 1985). In some instances, alcohol increases resting heart rate. The effect is stronger when blood alcohol level is rising and if alcohol was consumed more rapidly. Further, individuals deemed genetically "at risk" for alcoholism by virtue of a positive family history of alcoholism (Pihl & Peterson, 1992) show stronger heart rate responses when blood alcohol levels are rising (Conrod, Peterson, Pihl, & Mankowski, 1997; Finn & Pihl, 1987, 1988; Finn et al., 1990) in a dose-dependent manner (Stewart et al., 1992). Active alcoholics have a similar heart rate response as nonalcoholics with a family history of alcoholism (Peterson et al., 1996). In addition, resting heart rate change predicts alcohol consumption in a taste-test situation (Pihl, Giancola, & Peterson, 1994), and is associated with self-reported consumption in

the real world (Peterson, Pihl, Séguin, Finn, & Stewart, 1993). The finding that heart rate increases can predict consumption may indicate a biological marker, but does not by itself confer incentive or appetitive significance to the effect.

Other evidence links heart rate responses to incentive. Controlled for food value, anticipation of more palatable (preferred) food results in larger heart rate increases (Kostarczyk & Fonberg, 1982). Waiting for your favourite food is more exciting. Further, the alcohol findings are made more interesting because alcohol and cocaine (the classic euphoria-inducing stimulant) both increase resting heart rate, and the drug combination has additive tachycardic effects (Carroll, Krattiger, Gieske, & Sadoff, 1990; Higgins, Rush, Bickel, Hughes, Lynn, & Capeless, 1993; McCance-Katz, Price, McDougle, Kosten, Black, & Jatlow, 1993). Other dopaminergic stimulants also cause tachycardia (e.g., Ackerman, Holcomb, & Dykman, 1984).

More interesting still is that magnitude of heart rate has been studied in a series of experiments examining the response to monetary incentive. Heart rate was shown in these studies to reflect the motivational significance (here, the amount of money a participant could win) of the task at hand, despite the somatic (movement) and negative emotional (frustration, anxiety) demands of the task on heart rate (Fowles, 1983a; Fowles, Fisher, & Tranel, 1982; Jennings, 1982; Perkins, 1984; Tranel, 1983; Tranel, Fisher, & Fowles, 1982). These investigators and others thus inferred that heart rate reflects activity of "behavioral activation" or appetitive motivational brain systems (Fowles, 1980, 1983b, 1987; Gray, 1987; Weitkunat & Schandry, 1990). Note that while some authors view heart rate increases as reflecting disinhibition (e.g., Newlin, Byrne, & Porges, 1990),

Fowles, (1988) argues for a direct behavioral facilitation/activation by alcohol and other drugs of abuse. Perhaps then, with proper controls for subjective experience, somatic activity and anxiety, alcohol induced heart rate changes can reflect incentive from alcohol (Pihl & Peterson, 1995). Further, if alcohol-induced heart rate change did produce/predict real-world conditioning (such as learning related to emotionally-charged stimuli), this would provide exciting evidence for a conditioned incentive marker.

Incentive from alcohol can also be measured to the extent that people consume it. In this regard, it is useful to note as well that many of the larger consumers of alcohol are consumers of other psychoactive drugs as well. A common stimulant mechanism underlying (and perhaps incentive) of so-called addictive drugs may be implicated for these people (Wise & Bozarth, 1987).

d. Alcohol incentive: Use, abuse and dependence

Most people in Western countries have had some direct experience with alcohol. For example, the United States National Comorbidity Survey (Anthony, Warner, & Kessler, 1994) found that 92 percent of the population reported a history of alcohol use. More men are active drinkers than are women, and more caucasians drink regularly than do non-caucasians (Grant, 1994). Alcohol consumption peaks between 18-29 years of age and declines afterward (Fillmore & Midanik, 1984). The majority who consume alcohol do so without becoming "alcoholic," but the reasons that some develop problems while others do not are largely unknown.

Consistently among the most common of the mental disorders, the percentage of

individuals who could be described as alcohol dependent according to criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III-R; American Psychiatric Association, 1987) is about 7 percent (in the preceding 12 months), and about 14 percent (in their lifetime; Kessler *et al.*, 1994). Lifetime prevalence of alcohol dependence is more likely in males (almost 3 times compared with females), younger individuals, caucasians, unemployed persons, those with less education, persons living alone, and nonreligious individuals (Anthony et al., 1994). These findings have been largely confirmed using the more recent *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994) criteria, with the more specific findings that White males age 18-29 are the most likely to experience alcohol related abuse and dependence (Grant, 1992; Grant, Harford, Dawson, Chou, Dufour, & Pickering, 1994). Why this is so is also largely unknown.

3. Rationale for the current studies

The study of the contributing factors to alcohol use, abuse and dependence is important for all populations with access to alcohol, and in particular for minority, under-serviced and disadvantaged populations. Nonetheless, the relevant base rates for consumption and dependence should also be considered if the statistically maximum societal (and research) benefits and impacts are desired. The economic and psycho-social sequelae of not doing so is dangerously immense; alcohol use has associated costs (violence, lost productivity, etc.) in Canadian (Eliany, 1989) and American (Rice, 1993) society in the billions of lost dollars annually, and untold amounts in personal,

interpersonal, familial and employment terms.

On average, young males drink more than other groups. This presents an interesting paradox. Conventional wisdom dating from the Bible ("Let him drink and forget his poverty and remember his misery no more"; *Proverbs 31*:7), and more recently, Conger (1956) informs us that people drink primarily for relief of "stress". Indeed, young men who drink more are also more likely to report the belief that alcohol relieves their tension (Kushner, Sher, Wood, & Wood, 1994). Yet most young (or caucasian) men would be considered by a scant few to be among the most poor, miserable and stressed in our society.

Perhaps then, drinking motives attributed primarily to relieving stress, or drinking "to forget" are an unfortunate myth; there appears to be more to the story. Why in fact do some young males drink more than average, and why are they more implicated in alcohol dependence? Perhaps some drink because alcohol has a subjectively or physically desirable short- or medium-term effect? Unfortunately this notion is fraught with controversy. Further, research efforts devoted to answering this question may be perceived as wasting resources on the privileged at the "expense" of those in more need and may thus be met with some criticism. Nonetheless, answering this question could have benefits to these and other members of society since large numbers of persons are affected.

The goals of science include describing, explaining, predicting and controlling phenomena, and the study of alcohol consumption (and thus dependence) is no exception to this fundamental tenet. There has certainly been no historical shortage of explanations and interventions for harmful alcohol consumption. Indeed, there were well over two

hundred as of 1960 alone (Jelinek, 1960), and the number has surely grown.

Thus, testing some of the more promising theories (and interventions) is desperately needed. Of the firm conviction that a single theory cannot expiain everything, we shall assume that factors contributing to alcohol consumption are multifactorial (environmental, psychological, physiological, etc.) and interactive (Pihl & Peterson, 1992). With this important caveat, we will proceed with the examination of the possibility of extrapolating a promising theory advanced in the behavioural neuroscience literature by White and Milner (1992). The theory concerns events with the capacity to change present and future behaviour. The central idea is that events can change behaviours by enhancing the consolidation of memory, even if there is no contingent relationship formed between the event and the behaviours. Separately, events can also change behaviours if they have desirable subjective or physiological properties *and if* these become contingently associated with the behaviours.

We decided to adapt this theory to determine if alcohol consumption (the event) had these two effects in people. If so, some of alcohol's capacity to influence behaviour contingently and non-contingently would be explained. Can alcohol enhance memory? Does it have subjectively and physiologically desirable properties?

Describing an individual's response to alcohol requires knowing about his or her characteristics prior to-, during-, and following its consumption (when the individual is again sober). Obviously, if the "individual" is an animal, the questions require different tools to answer than for people. Nonetheless, comparisons of animals and humans can be useful to converge on the theory to the extent that suitable models can be validated.

4. Theoretical considerations

Before we discuss theories linking alcohol use to incentive, we begin with a brief presentation of the link between emotion and memory. The relationship between mood state and memory is another topic well outside the constraints of a single thesis. What is emotion? Is emotion primary? These are questions requiring many collections of volumes to answer.

In humans, we might begin with the idea, admittedly somewhat simplistic, that some subjective experiences are desired or wanted, while others are not. There may be a somewhat universal consistency and vocabulary for these states, or they may be entirely individual. In either case, mood states appear to have the common property that they tend to predispose people toward certain physiological states, actions and thoughts (Lang, 1995; Lang, Greenwald, Bradley, & Hamm, 1993). For our purposes, emotional responses to events appear to determine in part how memorable they are later on (Bradley, Greenwald, Petry, & Lang, 1992). Lang conceptualizes emotion in terms of *valence* (subjective positivity -- pleasant states like love and joy; and subjective negativity -- unpleasant states like anger sadness and fear) and *arousal* (the variations in metabolic and neural activity in the systems responding to valence). Lang has shown that an event's arousal (but not valence) is what determines how memorable it is later on.

A different approach is supplied by the so called semantic network theories of emotion. Semantic network theories of cognition (e.g., Anderson & Bower, 1973; Collins & Loftus, 1975; Collins & Quillian, 1969) suppose that thought and memory can be modeled in terms of an associative network of concepts that are used to describe events.

An event or experience is represented in memory by a cluster of "descriptive propositions." These are established in memory by new associated connections among the concepts used to describe the event. The basic unit of thought is seen as the proposition, the basic thought process is activation of the proposition and its concepts. As applied to emotion,

> The semantic-network approach supposes that each distinct emotion...has a specific node or unit in memory that collects together many other aspects of the emotion that are connected to it by associative pointers...Each emotion unit is also linked with propositions describing events from one's life during which that emotion was aroused...These emotion nodes can be activated by many stimuli-- by physiological or symbolic verbal means. When activated above a threshold the emotion unit transmits excitation to those nodes that produce the pattern of autonomic arousal and expressive behaviour commonly assigned to that emotion...Activation of an emotion node also spreads activation throughout the memory structures to which it is connected, creating subthreshold excitation at those event nodes...Thus...excitation [of] the sadness node...will maintain activation of that emotion and thus influence later memories retrieved. (Bower, 1981, page 135.)

Thus, the model predicts two effects: *state dependence* and *mood congruence* (Blaney, 1986; Bower, Gilligan, & Monteiro, 1981; Fiedler & Stroem, 1986; Isen, Shalker, Clark, & Karp, 1978; Laird, Wagener, Halal, & Szegda, 1982; Rholes, Riskind, & Lane, 1987; Small, 1986; Teasdale & Fogarty, 1979). State dependence refers to the idea that semantic encoding in one state is best retrieved in the same state. You remember things that you learned when happy if you are remembering while happy. Mood congruence refers to the notion that memories consistent with your mood state will most likely be encoded. Thus, the theory might predict a relationship between alcohol's pleasurable effects immediately following encoding and facilitated consolidation of pleasurable (but not unpleasurable) stimuli.

5. Theories linking affect, incentive and memory to alcohol use

a. Alcohol expectancy theories

Semantic network theories have been applied to the problem of alcoholism as well (Goldman, Brown, Christiansen, & Smith, 1991; Oei & Baldwin, 1994). The operational definition of this idea is termed the alcohol "expectancy" (Brown, Christiansen, & Goldman, 1987). Briefly, although not the first or only theory to link affect, incentive and conditioning to alcoholism, alcohol expectancy theory is perhaps the most explicitly "cognitive" in its approach and therefore has relevance for predicting the effects of alcohol on memory,

> Because of their potential for tying together a host of psychosocial and biological/genetic variables, and carrying forward the influence of these variables over extended time periods, memory processes (information storage) are now being considered by researchers of all types as one possible "final common pathway" for drinking decisions ...with the alcohol expectancy construct as a central memory element (Goldman et al., 1991; pages 136 and ...143).

Whether expectancies are indeed the central memory element is open to debate. What alcohol does to memory, and what people attribute to alcohol, may be both stored in memory but represent different concepts. Alcohol can affect memory. By contrast, alcohol expectancies are beliefs that people may hold concerning the possible subjective outcomes during- and following alcohol consumption (Brown, Goldman, Inn, & Anderson, 1980). The semantic network or connectionist structure of these beliefs (or cognitions) has been extensively validated (Cooper, Russell, Skinner, & Windle, 1992; Goldman, 1994; Leigh & Stacy, 1993; Rather & Goldman, 1994; Rather, Goldman, Roehrich, & Brannick, 1992; Stacy, Widaman, & Marlatt, 1990) suggesting they can indeed be considered as other conventional or natural memory components.

These beliefs are predictive of current and future alcohol consumption (Christiansen, Smith, Roehling, & Goldman, 1989; Cooper, Russel, Skinner, Frone, & Mudar, 1992; Darkes & Goldman, 1993; Downey & Kilbey, 1995; Hull & Bond Jr.,

1986; Stacy, Leigh, & Weingardt, 1994; Weingardt, Stacy, & Leigh, 1996; Williams & Ricciardelli, 1996; Wood, Sher, & Strathman, 1996). However, whether they are modified by acute or chronic alcohol consumption (in addition to predicting it) remains unknown. Expectancies are often in place before the individual has ever even consumed alcohol (Goldman et al., 1991), and they have a strong genetic basis (Vernon, Lee, Harris, & Jang, 1996); and, although heavier drinkers have different expectancies than lighter drinkers, whether this is cause and/or consequence is unknown.

If expectancies are modified by alcohol, then the degree to which alcohol produces memories for desired outcomes may reflect expectancies. If alcohol causes desirable effects on memory (perhaps by enhancing positive memories and/or inhibiting negative memories), but the effects are not attributed to alcohol, no expectancy will form despite alcohol's effects on the desirable memory. The opportunity of testing the relationship between alcohol's acute effects on emotionally charged memory and expectancies remains.

b. Another approach: Extrapolating White's theories

While expectancy theories are more cognitive (emotions are elements in a computational net), other theories present a different view that emotions and memory are separate processes that can interact. We have adapted the reinforcement model of White & Milner (1992) to the current studies since it presented an opportunity to balance motivational theories with memory constructs. White & Milner (1992) reviewed the animal literature on how "reinforcement" occurs. They concluded that reinforcers (here,

we speculate, alcohol) change the probability of responding in at least two ways. First by *simple/nonspecific/nonselective/pharmacological enhancement*,

> Many memory-enhancing substances also have rewarding motivational properties, but several lines of evidence...lead to the conclusion that the mechanisms of enhancement and motivation are independent. This means that the process by which the consolidation of a memory may be promoted is independent, both in the physiological and informational senses, of the representation of the memory itself. One idea concerning the enhancement phenomenon is that reverberatory activity by the neurons representing the memory may promote consolidation. Any process that prolongs or potentiates this activity...might therefore enhance the memory. The other possibility is that an enhancing event may initiate a process that acts directly on some later stage of consolidation... (page 454).

Second, reinforcers can act such that their incentive properties affect memory directly. This is called *conditioned motivation* or *conditioned incentive*,

> In the normal animal an important function of rewards... is to condition their motivating effects to other brain activity present

> at the time. Data...point to the nucleus accumbens as an important site at which rewards influence responses...Animals learn about these motivating properties; they become associated with neutral stimuli of various kinds, and the presence of these associations influences behaviour when the stimuli are encountered on future occasions. (page 462)

Thus, incentive activity can be conditioned to other events by some unknown memory process. What is this process? Based on earlier work (e.g., McDonald & White, 1993, etc.) showing the dissociability of memory functions in the dorsal striatum (habit learning), amygdala (incentive learning), and hippocampus (declarative learning), White (1996) applied these findings to an animal model of drug reinforcement. Thus, the incentive properties might be conditioned to external events in one or more of these memory systems. Simply put, the dorsal striatum is responsible for stimulus-response learning. Alcohol would enhance habit responses in this system. The role for alcohol in the amygdala system is more controversial. This system is responsible for adding an emotional stamp (appetitive or aversive) to neutral events. White concludes there is little evidence for incentive reward in the accumbens/VTA system. Since these centres are intimately connected to the amygdala, and since the CPP evidence is weak, White concludes that alcohol does not interact with this system. The hippocampal system is for complex learning about multiple stimuli. Included here is the possible implication of the hippocampus in the learning about the relationship between affective states and alcohol.

White concludes that alcohol's role in influencing the hippocampal system may be limited to learning that alcohol helps relieve negative states.

Based on the evidence so far we raise a possibility different from that of White that the hippocampal system (via its own connections with VTA/nucleus accumbens), and possibly the amygdala system as well may be implicated in conditioning alcohol's rewarding effects. Relief and conditioned relief effects could be mediated in hippocampus and/or amygdala, and related structures.

Our use of incidental and intentional paradigms cannot speak to anatomical considerations, but may help determine the relevance of simple/nonselective memory enhancement and conditioned incentive to the effects of alcohol on memory. Adaptation of White's ideas to alcohol provides an excellent, testable model. Further, it allows us to test nonspecific memory enhancement hypotheses vs incentive memory effects hypothesis discussed above and apply them to how alcohol affects different forms of learning.

c. *Another theory: Motivation and alcoholism*

Another exciting theory implicates unconditioned and conditioned motivation in the problem of alcohol use (Pihl & Peterson, 1995). In this sense unconditioned motivation is seen to implicate alcohol's capacity to relieve punishment (e.g., pain, hurt, anger, depression). Further, alcohol's unconditioned effects include satiation (e.g., satisfaction, contentment, and calm). Conditioned (learned or associated) effects of alcohol include relief of threat (e.g., cues for punishment: anxiety and fear) and increased sensitivity to promise (e.g., excitement, curiosity, pleasure and hope). Each of the four

effects is linked in theory with a specific neurotransmitter system (respectively opioid, serotonergic, GABAergic and dopaminergic). In this sense, we obviously are of the belief that the motivational effects of alcohol are not limited just to influencing dopamine.

While to state this is obvious, it is nonetheless important to demonstrate that any theory must balance specificity with validity. We focus in this thesis on dopamine not because it is most important, but because it is tied with incentive reward (our primary interest). Thus, the Pihl and Peterson (1995) conceptualization differs modestly from White (1989). Pihl and Peterson implicate dopamine in conditioned reward while White implicates it (two separate systems) in unconditioned and conditioned reward. Nonetheless, the distinction for our participants is a moot point since all are social drinkers; for them alcohol is already a substance with some history and/or potential for conditioning.

Pihl and Peterson implicate baseline heart rate change to alcohol as one possible indicator of alcohol's conditioned reward potential. If so, linking this event to other indications of alcohol's ability to influence memory would indeed be important. Further, defining parameters whereby alcohol's effects on physiology can be associated with alcohol's effects on memory (e.g., intentional, incidental) seems of primary relevance here. Unfortunately, the experiments in this thesis had little emphasis on incentive relief. The primary reason for this was paradigmatic. To investigate alcohol's relief effects, we would need a laboratory stressor. Adding such a stressor (while participants were concurrently exposed to emotional stimuli) would have complicated the results concerning alcohol's effects on memory. Future experiments may examine the role of incentive relief

further, but it is interesting to note that incentive reward (alcohol-induced heart rate increase) and relief (alcohol-induced stress dampening) can be correlated, at least in the laboratory (Peterson et al., 1993).

This thesis represents a unique opportunity to test some of the hypotheses that these three (i.e., semantic network/alcohol expectancy, White's, and Pihl & Peterson's) theories in regards to the effects of alcohol on memory. As stated, semantic network theory would predict that alcohol would enhance memories according to alcohol's pleasurable effects. By contrast, our extrapolation of Norman White's ideas leads us to the prediction than alcohol might affect incidental memory differently from intentional memory. Finally, our interpretation is that the Pihl & Peterson model would be supported to the extent that alcohol induced heart rate change (and alcohol-induced stress-response dampening, had it been investigated) would predict alcohol's desirable effects on intentional memory, but not alcohol's nonspecific effect on incidetal memory.

6. Some premises underlying the studies contained herein:

a. The degree to which alcohol produces acute changes that are subjectively or physiologically linked to desirability can be measured. Subjective changes can be measured by questionnaires. Physiological changes may be inferred by heart rate response to alcohol.

b. Memory processes are labile for some time after encoding of information, and may be modified by retroactive influences, including, possibly, alcohol.

c. Memory has more has salience if participants refer it to themselves (e.g., Bower &

Gilligan, 1979), and this salience can be inferred by subjective (valence) ratings.

d. Alcohol produces changes in learning, and possibly in memory.

e. Reduction of retrograde interference is one possible mechanism by which alcohol exerts its effects. Minimizing such interference will mean that, if alcohol improves recall or recognition, it did so by means other than reducing interference.

f. If relevant for future behaviour, including predicting drinking behaviour, alcohol's effects on incentive and memory should be related in some meaningful way.

g. According to White's view, non-contingent but contiguous reinforcers can improve memory in animals by simple enhancement. Our premise is that this is embodied in the in an incidental learning paradigm. Alcohol would thus enhance memory much as a non-contingent sucrose injection does in a laboratory rat. In this regard, alcohol would enhance memory nonselectively for neutral, positive and negative material. Further, the degree to which this enhancement occurred for a given individual would be independent of alcohol's incentive reward/relief effects (alcohol-induced heart rate increase, or stress-response dampening for example).

h. According to White's view, contingent reinforcers can influence memory in animals via conditioned incentive. Our premise is contingencies can be "implied" in addition to having people "work" for the alcohol. Our premise is that this is embodied in an intentional learning paradigm. Alcohol would thus influence memory much a contingent saccharin (or sucrose!) consumption in laboratory rats. In this regard, alcohol would affect memory for stimuli so as to produce outcomes with face-valid incentive value: enhancing memory for positive material and/or inhibiting memory for negative material. Further, the

degree to which this occurred for a given individual would be associated with alcohol's incentive reward/relief effects (heart rate increase or stress-response dampening).

7. Goals of the individual studies:

Study 1

a. To determine whether postlearning consumption of alcohol would affect emotionally salient verbal memory as well as neutral memory.

b. If so, to examine alcohol's effects on the emotional stimuli to determine what might be the mechanism(s) implicated in alcohol's effects on incidental memory.

c. To search for possible psychophysiological "markers" for the memory effect, if any.

Study 2

a. To determine which individual (participant) differences, if any, mediated alcohol's effects in an incidental paradigm.

b. To follow up on Study 1 and examine whether desirable and undesirable memories be mediated by the same factors.

Study 3

a. To determine whether alcohol affects intentional verbal memory.

b. If so, to determine which would be the mechanism involved.

c. To complement Study 2 and determine if individual differences are implicated in an

intentional paradigm.

Study 1

We are able to find everything in our memory,

it is like a dispensary or chemical laboratory

in which chance steers our hand

sometimes to a soothing drug

and sometimes to a dangerous poison

-- Marcel Proust

Forget "Drinking to Forget": Enhanced Consolidation of Emotionally Charged Memory by Alcohol

Note: in press at

Experimental and Clinical Psychopharmacology,

Volume 5, number 3, 1997.

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Abstract

Social drinkers (42 men, 18-34 years old) participated in a study of the effects of alcohol consumption on incidental memory for emotionally salient verbal stimuli. Participants rated depressing, elating and neutral statements while sober. Fifteen min later they consumed alcohol or active placebo (1.0 or 0.1 ml/kg) in an environment with minimal retrograde interference. In surprise memory testing 24 hr later, when participants were again sober, the alcohol group had increased recall across statement type. The alcohol group also had better recognition of depressing and elating statements, but recognition of neutral statements did not differ between groups. Findings suggest alcohol produced a nonspecific enhancement of incidental memory, and that alcohol's motivational properties were not implicated.

Alcohol has diverse effects on memory. Intoxication results in performance impairments on some memory tasks (e.g., Kerr, Sherwood, & Hindmarch, 1991; Peterson, Rothfleisch, Zelazo, & Pihl, 1990), leaves performance on others apparently unaffected (Lister, Gorenstein, Risher-Flowers, Weingartner, & Eckardt, 1991; Nilsson, Backman, & Karlsson, 1989; Roache, Cherek, Bennett, & Schenkler, 1993), and actually enhances performance on still other tasks (Loke, 1992). Whether alcohol will result in impairment, no effect, or enhancement of memory appears to depend on several factors. The effects of alcohol on mood, arousal, perception, and attention all can have an influence (Weingartner, Eckardt, Molchan, & Sunderland, 1992). Administration of certain foods (Millar, Hammersley, & Finnigan, 1992), including sucrose (Zacchia, Pihl, Young, & Ervin, 1991) and tryptophan (Westrick, Shapiro, Nathan, & Brick, 1988), can moderate. Other substances with more direct actions in the brain can also moderate, including cholinergic (Brioni, McGaugh, & Izquierdo, 1989) and GABAergic (Castellano & Pavone, 1988; Castellano & Populin, 1990) compounds. The dose of alcohol is also important, with memory facilitation at low doses, but impairment at moderate-to-heavy doses (Jubis, 1986, 1990). Participant variables such as intelligence (Maylor, Rabbitt, James, & Kerr, 1990), gender (Haut, Beckwith, Petros, & Russell, 1989), familiarity with the task (Rumbold & White, 1987), and environment (Babbini, Jones, & Alkana, 1991; Colbern, Sharek, & Zimmermann, 1986; Miles, Porter, & Jones, 1986) can also moderate. Time, rate, and route of alcohol administration are important. Effects of alcohol on memory may be different on the ascending, as opposed to descending, limb of the blood-alcohol curve (Jones, 1973). One must also consider whether alcohol is administered before acquisition

(registration of the stimulus in the central nervous system) or before retrieval because state-dependent effects of alcohol are observed (Goodwin, Powell, Bremer, Hoine, & Stern, 1969; Werth & Steinbach, 1991).

It appears that although alcohol inhibits acquisition and retrieval, reliable evidence suggests that alcohol can improve memory if it is consumed in the interim. For example, Kalin (1964) and Mann, Cho-Young, and Vogel-Sprott (1984) found that although alcohol impairs memory when consumed before acquisition, memory improves when alcohol is consumed after acquisition. Memory improvement also is observed in experiments where alcohol is administered only after acquisition. Under these conditions, alcohol enhances memory in animals (Alkana & Parker, 1979; Melia, Ehlers, LeBrun, & Koob, 1986) and humans (Hewitt, Holder, & Laird, 1996; Lamberty, Beckwith, Petros, & Ross, 1990; Mueller, Lisman, & Spear, 1983; Parker et al., 1980). The enhancement occurs regardless of whether participants are explicitly told to memorize (i.e., whether learning is intentional or incidental), and memory is improved across the verbal, visual, and kinaesthetic domains. Importantly, the effect has been shown to be dose dependent (Parker et al., 1981), suggesting a direct pharmacological effect. The mechanisms for the direct effect are not yet clear, but they may include interactions with individual differences or characteristics of experimental participants (Kalin, 1964), a reduction of postlearning interference (Lamberty et al., 1990; Mann et al., 1984; Mueller et al., 1983; Parker et al., 1980), a nonspecific enhancing effect on trace consolidation (Hewitt et al., 1996; Kalin, 1964; Lamberty et al., 1990; Mann et al., 1984; Parker et al., 1980, 1981), or an enhancement specifically mediated by alcohol's incentive effects (Esposito, Parker, &

Weingartner, 1984; Lamberty et al., 1990; Mann et al., 1984). By the term *incentive effects*, those authors were referring to induction of a rewarding-euphoric sense of well-being or psychomotor stimulation (e.g., Connors & Maisto, 1979; Martin, Earleywine, Musty, Perrine, & Swift, 1993; Sano et al., 1993; Wise & Bozarth, 1987). Under incentive effects, we would also include relief from anxious and depressed moods (e.g., Conger, 1956; Freed, 1978; Mayfield, 1968; Mayfield & Allen, 1967; Pihl & Peterson, 1992; Sayette, 1993; Williams, 1966). The experiment described in this article was an examination of two of the aforementioned mechanisms potentially responsible for the memory improvement. More specifically, while attempting to control for individual differences and retrograde (postlearning) interference, we examined the potential mediating role of incentive effects versus nonselective memory enhancement. We expected that if incentive properties mediate, then alcohol would affect memory outcomes accordingly. Thus, alcohol would facilitate desired (positive) memories and inhibit undesired (negative) ones. Also, we expected incentive effects to predict these outcomes.

By contrast, if nonselective enhancement processes mediate, we expected alcohol to increase both negative and positive memory. These memories should be (positively) correlated, and scores would not be predicted by incentive effects. Finally, we added measures of psychophysiological response to alcohol on the ascending limb of the blood-alcohol curve as potential markers for memory effects. It has been proposed (Parker et al., 1981) that physiological processes on the ascending limb are likely candidates for enhancement of consolidation.

Method

Participants

Forty-two nonalcoholic men (aged 18-34 years) were recruited through newspaper advertisements. Nonalcoholic status was defined as (a) never having met the criteria of the revised third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-III-R*; American Psychiatric Association, 1987) for alcohol dependence or abuse and (b) a score of less than 5 on a shortened version of the Michigan Alcoholism Screening Test (MAST; Pokorny, Miller, & Kaplan, 1972). Participants were also assessed for family history of alcoholism using MAST criteria adapted for family members. Individuals with a diagnosis of alcoholism in their mother, or father, or paternal grandfather were excluded, as were those with a history of alcoholism in any two other relatives. Participants were screened using Family History Research Diagnostic Criteria (FH-RDC; Endicott, Andreasen, & Spitzer, 1975), which were adapted and used to assess the participants' own personal history. Individuals meeting lifetime criteria for a major psychiatric disorder, including substance abuse or dependence, schizophrenia, bipolar disorder, antisocial personality, and anxiety disorders were excluded, as were individuals meeting criteria for depression within the past year. Participants also were excluded if they had a history of traumatic head injury or cardiac condition.

Participants were asked to refrain from drinking alcohol and taking illicit drugs for at least 24 hr and from eating for at least 4 hr before Session 1. They also were asked to refrain from drinking alcohol and taking illicit drugs between sessions. All participants

were tested individually and did not interact with one another. They were paid $5 per hour.

Apparatus

Materials used in the learning paradigm were taken from Velten (1968). Three categories of self-referring statements were used: depressing, elating and neutral. The emotional valence and salience of these statements have been well validated (e.g., Whissell & Levesque, 1988). We printed all 180 (3 x 60) statements onto 7.72 cm x 12.70 cm index cards, one statement per card. Twenty-five statements from each category were randomly selected for use in the learning procedure. The same 75 statements were used for all participants. Distractor items used in the recognition test were taken from the remaining statements.

A Grass (Quincy, MA) Model 7D polygraph was used for physiological recording with two Model 7P4 EKG Tachograph preamplifiers for heart rate and digital blood-volume amplitude (DBVA), a 7P1 preamplifier (on psychogalvanic response setting) for skin conductance measures, and a 7P3 preamplifier (integrated signal) for measures of frontalis muscle tension. Skin conductance measures were obtained from the 7P1 by converting the data (originally in resistance units) to conductance units (microsiemens = reciprocal of resistance) before any calculations or analyses were performed. Medi-Trace pellet electrodes placed bilaterally on the lower chest detected heart rate. A Grass Model PTTI photoplethysmograph attached to the nail of the index finger of the left hand detected DBVA. Two Beckman large bipotential skin electrodes placed on the medial

phalanges of the second and third fingers of the left hand with a conducting medium composed of Glaxal Base cream (a neutral base cream similar to Unibase cream) mixed with physiological saline (Finn & Pihl, 1987) detected skin conductance. Two Medi-Trace pellet electrodes (one placed 1 cm above the left eyebrow centered over the eye and the other place 1 cm above the first) detected muscle tension. Paper records were used for scoring all the polygraph recordings. A Polar Electro (Kempele, Finland) Sport Tester PE300 portable heart rate monitor system was used as a backup measure for each participant.

The alcohol used was 95% U.S. proof. In the alcohol condition, the dosage was 1.0 ml/kg, mixed with 5 parts orange juice. In the placebo condition, an "active" placebo (e.g., as recommended by Stewart, Finn, & Pihl, 1992) was used to disguise flavor; the dosage was 0.1 ml/kg mixed with 0.9 ml/kg Vichy brand mineral water; the alcohol-mineral water mixture was mixed with 5 parts orange juice. A spray bottle was used to add trace amounts (maximum of 2 ml) of alcohol to the top of drinks. An Alco-Sensor III (Intoximeters, Inc., St. Louis, MO) was used to determine the breath alcohol concentration (BAC).

The Inventory of Drinking Situations (IDS; Annis, 1982) is a self-report measure of drinking. The IDS contains subscales of drinking frequency associated with a variety of situations, including drinking in response to pleasant (IDS-P subscale) and unpleasant (IDS-U subscale) emotional states. We therefore used these subscales as proxies for alcohol's incentive properties. This inventory, used in clinical populations, was adapted for use in our nonalcoholic participants (who were expected to drink considerably less)

by changing the heading at the top of the form to "I had at least one drink" from "I drank heavily." Three subtests from the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1974) were also used. The Information and Vocabulary subtests were used to measure general cognitive and verbal abilities, as well as life experience, and the Arithmetic subtest was used as a measure of concentration skill. Two Likert-type scales also were used: Negativity-positivity of the statements in the learning paradigm was rated on a 7-point scale, and certainty in the recognition task was rated on a 10-point scale.

Finally, a questionnaire was used to obtain demographic information such as age, years of formal education, and measures of quantity and frequency of drinking (here, these were collapsed as the estimated average number of drinks consumed each week over the past year). Participants were treated in accordance with the American Psychological Association's (1992) Ethical Principles of Psychologists.

Procedure

Participants were contacted by telephone and asked the MAST and *DSM-III-R* alcoholism questions in a brief interview. The actual experiment was divided into two testing sessions that occurred at the same time on consecutive days. The first session lasted 3.5-5 hr, and the second session 1-2 hr.

Session 1. On arrival, participants were given the screening interview with adapted FH-RDC criteria and, if accepted, read and signed an informed consent. Participants were weighed and had an initial breathalyzer reading taken to ensure a BAC of zero. Psychophysiological electrodes were then attached while participants sat in a reclining

chair. Sport Tester monitors were demonstrated and set to operate. Participants remained in the chair until the end of the postdrinking baseline.

Learning procedure. Participants were not explicitly told that they were participating in a memory study and were not given any instructions to remember the stimulus materials. Instead, an "incidental" learning procedure was used in which the participants were exposed to the stimuli in a card-sorting procedure. Statements were presented one at a time in a standard order across participants. Statements were given in 20-s intervals. Within an interval, participants were required to read the statement aloud, refer it to themselves and sort it in to one of three piles. Piles were respectively labeled *neutral*, *negative*, and *positive*. The original category to which each statement belonged (Velten, 1968) was not disclosed. As a data check, we required that participants sort a minimum of 13 of 25 (52%) of the statements from Velten's categories correctly (i.e., neutral to the neutral pile, depressing to the negative pile, and elating to the positive pile). Participants also were asked to rate negativity-positivity of the statement during the interval. After participants had sorted all 75 cards, they sat quietly for 15 min with the room lights dimmed so a predrinking psychophysiology baseline could be recorded. Participants were randomly assigned to the alcohol or placebo condition during this time. Alcohol or placebo was consumed in three equal drinks. Immediately before presenting each drink to participants in both groups, and unknown to them, the surface of the drink was sprayed with alcohol to enhance the alcoholic flavour and smell. To further enhance placebo effectiveness, the experimenter, who did not know group assignment, told all participants that they were receiving "1.0 ml/kg alcohol, the equivalent of three to five drinks" and

that "because of the purity of the alcohol, the taste and subjective effects may be different (either stronger or weaker)" than they might expect. Participants were required to consume the drinks within 20 min. Twenty minutes after drinking had begun, another 15 min quiet rest period ensued, in which a postdrinking baseline was recorded. After the postdrinking baseline, participants were taken off the recording electrodes and Sport Tester. To prevent boredom -- and to limit new verbal learning-- participants were allowed to watch "National Geographic" nature videos with the sound turned off, listen to classical music, or both. For both groups, BAC was measured 10 min after the drinking period, and each 10 min thereafter, for 5 additional readings. For participants in the alcohol condition, BAC was then measured every 30 min thereafter, until it dropped below 0.04 ng/dl. Participants were then permitted to leave the laboratory. For participants in the placebo condition, BAC was measured once more 30 min later (i.e., 90 min after the drinking period); participants then were permitted to leave the laboratory. No feedback about BAC was given to any participant until the end of the experiment.

Session 2. On arrival for the second session, participants were given a breathalyzer reading to ensure that it was zero and were asked about alcohol or illicit drugs since the previous session. Session 2 was conducted by an experimenter who did not know group assignment. Participants first completed the recall task, which lasted 15 min. They were asked to recall (i.e., to write down) as many of the statements from the previous session as possible and to list each response separately. Participants were encouraged to remember as much as they could word for word when possible. In the recognition task that followed,

target stimuli were selected from the correctly sorted statements in Session 1. Thirteen to 15 targets were randomly selected per category. To these, enough distractor cards were added to ensure that there were 30 cards per category. Participants were presented with the 90 randomly shuffled cards, one card at a time at the participant's own rate. They were asked to decide whether they had seen the card in the previous session and to rate their certainty. Errors (i.e., misses, false- positives) were recorded. After the recognition task, the WAIS-R subtests were administered. Participants then completed the IDS and demographic questionnaires. At the end of the experiment, they were fully debriefed and reimbursed for participation.

Results

Participants

Demographic, cognitive, and drinking behavior measures were used to match groups and limit potential confounds (e.g., Kalin, 1964) to the interpretation of results. Age, years of education, WAIS-R subtests, and the IDS-U and IDS-P subscales were examined for group mean differences using one-way analyses of variance (ANOVAs) or nonparametric Mann-Whitney tests as appropriate. Group matching was determined as an absence of a significant group mean difference. Group means and standard deviations for the demographic variables are shown in Table 1.

----- Insert Table 1 about here -----

Memory measures

Recall and recognition were scored by a single rater (e.g., Kalin, 1964) who did not know the participants' group membership. As determined by one-way ANOVA, the total number of responses to the recall task did not differ between the alcohol and placebo groups. Of the responses, those that accurately resembled the word content original stimulus statements (e.g., Kalin, 1964) were then tabulated for the recall task; confabulated responses were omitted. Accurate resemblance was determined when the response contained at least three of the same key content words as one of the original Velten (1968) statements. Such gist criteria are commonly used in research as measures of accuracy for the entire stimulus phrase (e.g., Goetz, Anderson, & Schallert, 1981). The number of recalled statements was tabulated for each category. Depressing, elating and neutral recall distributions were analyzed for univariate outliers 3.3 *SD*s from the mean (alpha = .001). These were rounded off to the next nearest value (Tabachnick & Fiddel, 1989). Inspection of the distributions revealed satisfactory normality and homogeneity of error variance. Recall was analyzed using a 2 x 3 (Group x Category) ANOVA, with category as a repeated measures variable as participants recalled statements from each category. There were significant main effects of group, $F(1, 80) = 7.27$, $p < .01$, and category, $F(2, 80) = 12.96$, $p < .00001$. The interaction did not reach significance. The group means and standard errors are shown in Figure 1.

----- Insert Figure 1 about here -----

For the recognition task, errors were totaled within each category. Distribution inspection and corrective procedures were carried out as per the recall data. The data for the recognition task were analyzed using a 2 x 3 (Group x Category) ANOVA, with category as a repeated measures variable. The interaction was significant, $F(2, 80) = 3.16$, $p < .05$, as were the main effects of group, $F(1, 80) = 7.84$, $p < .008$, and category, $F(2, 80) = 53.0$, $p < .0001$. Posthoc inspection of the interaction was done using the Newman-Keuls method. Relevant to the current investigation, the analysis revealed that the alcohol group made fewer errors than the placebo group for recognition of depressing, $Q(80) = 4.66$, $p < .01$, and elating, $Q(80) = 3.76$, $p < .01$, statements. However, the group means for neutral statement errors did not differ significantly. Means and standard errors are shown in Figure 2.

----- Insert Figure 2 about here -----

Card-sorting measures were also analyzed. Groups did not differ by Mann-Whitney test comparisons on the proportion of statements correctly sorted or the negativity-positivity rating of the original statements. Certainty ratings for the recognition task also were compared. The groups did not differ as determined by Mann-Whitney test. Means and standard deviations for these three measures are shown in Table 2.

----- Insert Table 2 about here -----

Psychophysiological Measures

Psychophysiological measures were scored by a different rater who also did not know the participants' group membership. Recordings were analyzed for three discrete time periods: during the card-sorting procedure, during the predrinking baseline resting period, and during the postdrinking baseline resting period. To analyze the card sorting period, we averaged the values over the first 90 s of the procedure. For the baselines, we averaged the values over the final 90 s of the baseline. Mean values were calculated by averaging points sampled every 2 s during each discrete 90-s period (e.g., as recommended by Finn & Pihl, 1987). Heart rate was measured in beats per minute, DBVA in arbitrary units, skin conductance in microsiemens, and muscle tension in microvolts. Sport Tester watches were programmed to record mean heart rate every minute. Means for the card sorting period were averaged over the first 5 min of the period; means for the baselines were averaged over the last 5 min of the period.

A multivariate analysis of variance (MANOVA) was used to analyze polygraph psychophysiology. A 2 x 3 (Group x Period) MANOVA was performed with period as a repeated measures variable. Data were natural-log-transformed to improve the normality and homogeneity of error variance. A significant multivariate interaction, Pillai's approximate $F(8, 156) = 3.69$, $p < .001$, and effect of period, Pillai's approximate $F(8, 33) = 16.61$, $p < .001$, were obtained. Because the multivariate effect of group did not reach significance, we did not analyze univariate group effects. We were not interested in univariate period effects, only group effects. However, significant univariate interactions were found for skin conductance, $F(2, 80) = 11.45$, $p < .001$, and DBVA

($F(2, 80) = 4.14$, $p < .02$. The interactions were further examined using posthoc Newman-Keuls tests. Results of interest in our analyses included that the groups differed postdrinking in terms of skin conductance, $Q(80) = 6.74$, $p < .01$, and DBVA, $Q(80df) = 5.22$, $p < .01$. Sport Tester results were significantly correlated ($r = .97$) with the polygraph heart rate data across the two groups and the three periods, suggesting that the Sport Tester is a useful tool for measuring heart rate.

BAC

BAC was analyzed using a 2 x 7 (Group x Time) ANOVA with time (10, 20, 30, 40, 50, 60, and 90 min postdrinking) as a repeated measures variable. A highly significant Group x Time interaction, $F(6, 227) = 9.34$, $p < .001$, and main effects of group, $F(1, 227) = 676$, $p < .001$, and time, $F(6, 227) = 4.43$, $p < .003$, emerged. Average BAC levels for these sampled times were 0.0635 ng/dl ($SD = 0.0002$) for the alcohol group, and 0.0012 ng/dl ($SD = 0.0002$) for the placebo group. As anticipated, alcohol produced much higher BACs than placebo, which itself produced negligible concentrations.

Regression Analyses

To further examine experimental hypotheses, we used regression analysis. We wanted to limit the dependent variables as much as possible because of sample-size constraints. These were collapsed by examining the correlations among the recall and recognition scores. The correlations are shown in Table 3. The recall of depressing and elating statements was correlated, but the recall of neutral statements was more correlated

with recognition than were the other two recall scores. Because the interpretation of this was unclear, we excluded the recall of neutral statements as a variable, and the recall of depressing and elating statements was combined into a single recall variable. Because the recognition variables were highly intercorrelated (and uncorrelated with the depressing and elating recall measures), the recognition variables were added together to form a single variable. The composite recall and recognition variables were examined for univariate outliers and normality. Outliers at the .001 level were rounded to the next nearest value (Tabachnick & Fiddel, 1989), leaving the variables normally distributed.

----- Insert Table 3 about here -----

To ensure an adequate (maximum 1:10) variables-to-participants ratio (Tabachnick & Fiddel, 1989), we wanted to limit the number of independent variables to four. Because we wanted to include the IDS-U and IDS-P subscales, variables measuring psychophysiological response to drinks were restricted to the two variables, (i.e., skin conductance and DBVA), which showed postdrinking group differences. Using stepwise regression, none of the four independent variables was found to significantly predict the recall variable. Analysis of univariate distributions was satisfactory. Stepwise regression results for recognition showed that only the IDS-U measure entered into the equation beyond the intercept (adjusted $R^2 = .13$, $F(1, 40) = 7.52$, $p < .0001$). The correlation matrix (not shown) indicated that IDS-U and recognition errors were positively correlated; those who drink in response to unpleasant emotions had more recognition errors. Analysis of

bivariate and univariate outliers at the .001 level was satisfactory.

Discussion

The principal finding was that alcohol consumed after stimulus exposure increased delayed recall and recognition of emotionally laden verbal memories and increased recall but not recognition of neutral memories. Overall, the findings were most consistent with the nonselective enhancement explanation, not the incentive explanation. First, the recall results suggest that there was no statistical difference in the amount of improvement by alcohol among the three categories of memory. Relative to placebo, alcohol increased the amount of neutral, depressing and elating memories relatively equally. Second, the recall of depressing and elating statements was positively correlated, indicating that alcohol had not produced selective enhancement of elating material or selective impairment of depressing material. Third, alcohol reduced errors on the recognition task for depressing and elating statements. The reduction for these categories was relatively equal. Fourth, the depressing, elating, and neutral recognition scores were positively correlated, indicating that alcohol did not produce selective effects. Fifth, our putative measure of alcohol's incentive rewarding effects did not predict recall or recognition. Thus, self-reported rewarding effects were not associated with the effects of alcohol on memory. Sixth, our putative measure of alcohol's incentive-relieving effects did not predict recall. Thus, self-reported relief effects were not associated with the effects of alcohol on recall. The mechanism responsible for nonselective enhancement is not yet clear, but it may involve

hormonal, neuromodulatory (McGaugh, 1989) or other memory-enhancing processes, possibly in the caudate nucleus and hippocampus and related structures (White, 1996; White & Milner, 1992).

Two findings may be consistent with the incentive explanation. First, alcohol selectively enhanced the recognition of emotionally laden material over neutral material. Thus, it may appear that alcohol had selective effects. However, this may instead have to do with stimulus characteristics. There might have been a floor effect for the neutral statements; neither group made a large number of errors. Therefore, it may be that the neutral statements were easier to recognize in our paradigm. That recall of neutral statements was not highly correlated with depressing and elating recall lends further credence to the possibility that, although our depressing and elating statements were similar, the neutral ones were different. In addition, we did not find that alcohol selectively increased memory for elating over depressing statements. Thus, the meaning of the improved recognition for emotionally laden but not neutral material cannot be attributed easily to alcohol's incentive effects. Second, our putative measure of alcohol's incentive-relieving effects did predict recognition. Thus, self-reported relief effects were associated with detrimental effects of alcohol on recognition. The possible interpretations of this are many. First, as the incentive explanation posits, it may be that individuals who report drinking to relieve negative emotions made more errors and that the relief incentive mediated forgetting. Second, it may be that individuals who drink for relief are habituated to alcohol effects on memory enhancement or are poor learners. Third, since recognition of depressing, elating and neutral material was highly correlated, it appears that any relief-

incentive effects are not restricted to the forgetting of depressing statements. Thus, any incentive effects derived by "drinking to forget" are not easily explained.

The effects of alcohol on recall were observed in the context of several factors. First, alcohol did not increase the overall number of responses given. Thus, the alcohol-induced increase in the number of correct recollections was not attributable merely to an increase in the number of responses given. This, in turn suggests increased accuracy, not merely increased quantity. Second, the results were obtained despite the fact that the groups did not differ on several individual characteristics that might have biased the results (Kalin, 1964). Included here were demographic, cognitive, and drinking characteristics, as well as their initial cognitive and psychophysiological responses to the stimuli. Third, the results were obtained despite the limiting of retrograde verbal learning. Although this is impossible to eliminate, our results suggest that alcohol can improve memory by means other than merely reducing interference (e.g., Hewitt et al., 1996). Fourth, although we did not examine intoxication or withdrawal effects other than BAC during the memory testing, other researchers (e.g., Lemon, Chesher, Fox, Greeley, & Nabke, 1993) have reported that these effects are minimal at 24 hr. Taken together, these factors led us to conclude that alcohol may have enhanced consolidation, likely via nonselective enhancement rather than by mediation through incentive effects, reduction of interference, or individual differences.

To our knowledge, this is the first controlled experiment showing that alcohol can affect consolidation of memory with self-rated emotional salience in human participants. Kalin (1964) found that alcohol improved memory for several Thematic Apperception

Test responses (including some with self-referring sexual content) if the alcohol was consumed after the response was given. However, participants in that experiment consumed alcohol both before and after responding to the test and did not rate salience. Our results for emotionally laden materials parallel findings of other experiments in which neutral material was used (Hewitt et al., 1996; Lamberty et al., 1990; Mann et al., 1984; Mueller et al., 1983; Parker et al., 1980, 1981). Our findings for neutral material also parallel this literature (e.g., Lamberty et al., 1990; Mueller et al., 1983).

We found that memory was not correlated with psychophysiological responses to alcohol. Thus, alcohol-induced changes in skin conductance and DBVA were not markers for the memory effects. Also, because the memory effects occurred despite a lack of group differences muscle tension and heart rate, it would appear that these variables are not markers either. Other physiological markers for the memory effects need to be found.

The findings for elating and neutral stimuli are consistent with motivational theories in the alcohol literature. However, increased memory for depressing stimuli is difficult to reconcile. Reinforcement (e.g., Conger, 1956; Landauer, 1969; Sayette, 1993; Wise and Bozarth, 1987) and expectancy (e.g., Brown, Goldman, Inn, & Anderson, 1980; Goldman, 1994; Goldman et al., 1991; Oei & Baldwin, 1994; Rather & Goldman, 1994; Stacy, Leigh, & Weingardt, 1994; Stacy, Widaman, & Marlatt, 1990) models propose that incentive effects are related to learning and memory. We have speculated that incentive is partially reflected by the frequency of drinking alcohol to regulate emotions, which likely involves memory processes in some way. We have proposed that the effects of alcohol on memory that participants judge as salient also has self-evident motivational

importance. If so, these models must explain how individuals learn that alcohol can regulate emotions in a desired way yet that it can also enhance undesired memories. Part of the answer may lie in our paradigm. Whether similar findings would occur if participants were explicitly told to remember remains to be seen. Perhaps this manipulation would alter the salience of the materials and thus the results. Another explanation may lie in thinking of *incentive* as an association of desired outcomes with drinking. A recent model (White, 1996) has offered some insight into this. Drug-outcome associations can be mediated by so-called "incentive" properties as described earlier, but they also can be formed independently of them. In the case of the former, the incentive properties become directly associated with drinking, forming a kind of alcohol-emotion association. In the case of the latter, any and all preexisting emotional associations, memory traces, and so on are strengthened by direct pharmacological effects of the drug. Thus, existing associations, some desired and some not become strengthened nonselectively by alcohol. This produces incentive effects separable from the direct alcohol-emotion ones. In the words of one reviewer, "the process responsible for enhanced memory for emotionally charged material may be much different from the process that operates on memories for changes in affect produced by alcohol." Nonetheless, because both processes appear to have incentive value and may at times be in opposition, this issue needs to be resolved. Thus, the relationships among the effect of alcohol on emotionally laden memories and the formation of desired alcohol-outcome associations awaits further investigation.

Finally, our attempts at controlling experimental confounds could not be

exhaustive. Further study must be given to other important variables such as alcohol expectancies, personality, and the experimental paradigm, among others. Further attention also must be paid to whether alcohol affects subjective appraisal of emotional stimuli in addition to increasing its quantity.

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This research was supported by grants from the Medical Research Council of Canada, and by a personal fellowship from the Fonds pour la Formation de Chercheurs et l'Aide de la Recherche (Quebec). We thank Robbie Goddard, Edwin Poon and Jennifer Crotogino for help with data collection. We also thank Jean Séguin for statistical recommendations.

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Table 1

Group Means for Demographic, Cognitive, and Self-Report Data

	Placebo		*Alcohol*		
Measure	M	SD	M	SD	p
Age	23.0	3.7	22.0	4.3	ns[a]
Years of education	14.8	2.4	14.0	2.1	ns[b]
Alcohol consumption (drinks per week)	7.8	7.9	5.0	7.5	ns[a]
WAIS-R subscale					
Information	19.8	5.7	20.5	4.8	ns[a]
Vocabulary	52.6	12.3	52.4	8.9	ns[a]
Arithmetic	13.3	3.0	14.6	2.3	ns[b]
Self-reported drinking associated with					
Pleasant emotions	14.1	5.5	12.3	6.1	ns[b]
Unpleasant emotions	7.4	6.6	5.8	8.4	ns[b]

Note. WAIS-R = Wechsler Adult Intelligence Scale-Revised.

[a]group difference nonsignificant using a Mann-Whitney two-sample (nonmatched) *U* test. [b]group difference nonsignificant using a one-way analysis of variance ANOVA

Table 2

Card Sorting and Recognition Certainty Measures

Measure	*Placebo*		*Alcohol*		
	M	*SD*	*M*	*SD*	*p*[a]
Card Sorting					
Proportion of cards correctly sorted	0.79	0.10	0.78	0.09	ns
Average rating[b] of stimulus cards	3.97	0.24	4.00	0.14	ns
Recognition task					
Certainty[c]	8.56	3.03	8.29	1.37	ns

[a]nonsignificance determined using a Mann-Whitney two-sample (nonmatched) *U* test. [b]Rating on a 7-point Likert-type scale (1 = *highly negative*, 4 = *neutral*, 7 = *highly positive*). [c]Rating on a 10 point Likert-type scale (1 = *unsure*, 10 = *sure*)

Table 3

Correlations Between Memory Measures

Variable	1	2	3	4	5	6
1. RED	--					
2. REE	.62*****	--				
3. REN	.57*****	.47***	--			
4. RD	-.03	.14	.15	--		
5. RE	-.02	-.09	-.19	.39***	--	
6. RN	-.49****	-.37**	-.40***	.27*	.31**	-

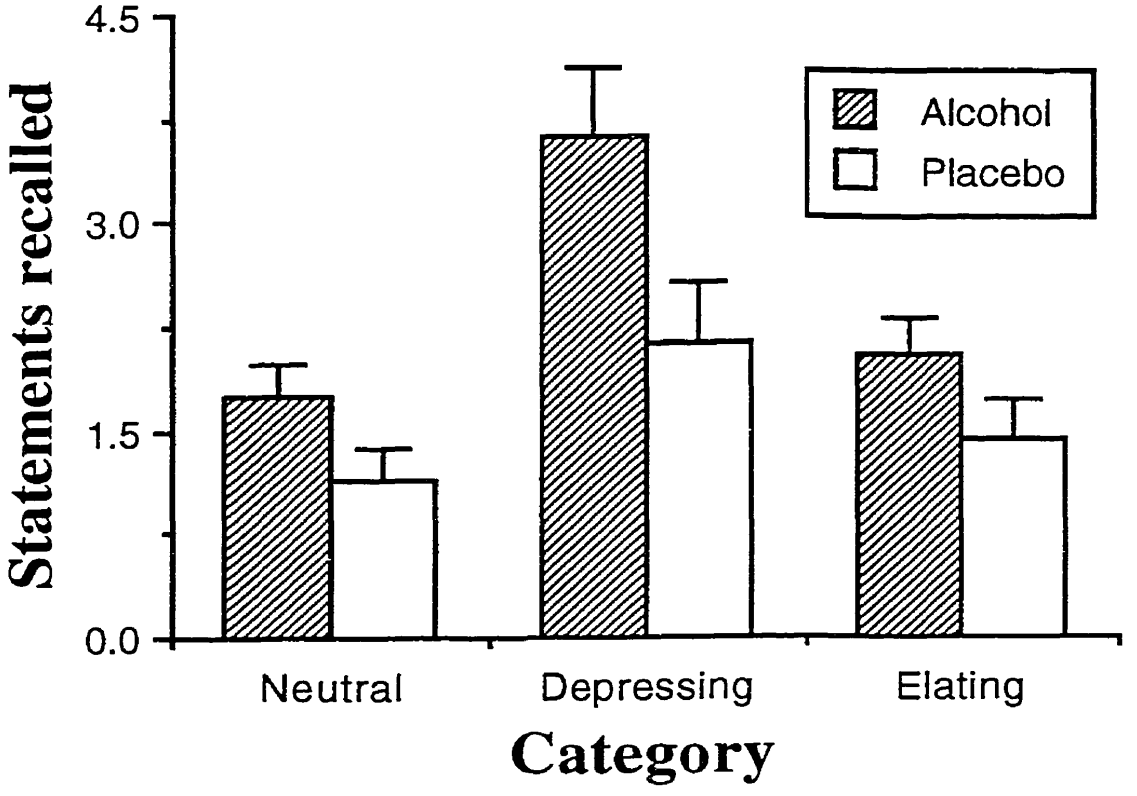
Note. RED = recognition errors for depressing statements; REE = recognition errors for elating statements; REN = recognition errors for neutral statements; RD = recall of depressing statements; RE = recall of elating statements; RN = recall of neutral statements. All probabilities are two-tailed.

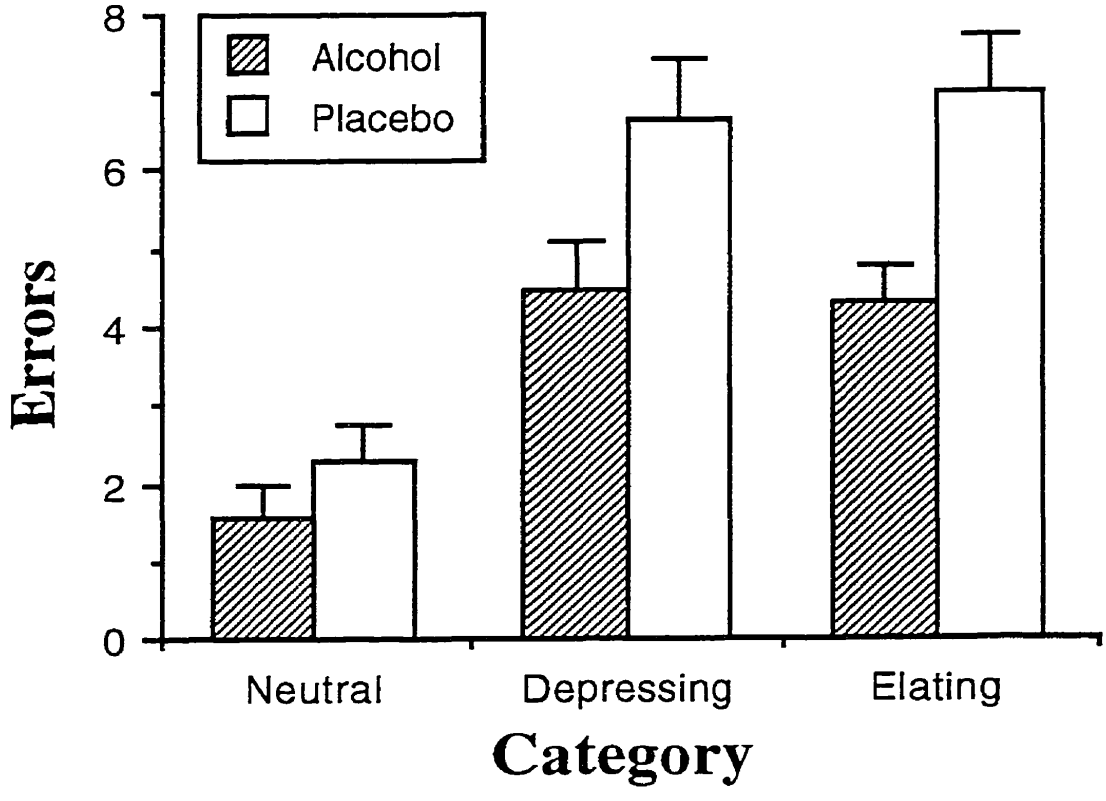
*$p < .1$ **$p < .05$ ***$p < .01$ ****$p < .001$ *****$p < .0001$

Figure Captions

Figure 1. Mean recall of neutral, depressing and elating statements. A minimum of three of the same content words as the original stimulus (Velten, 1968) statement was required. Increased recall of neutral, depressing and elating statements was found for the alcohol group relative to the placebo group, and the amount of increase did not differ across category.

Figure 2. Mean errors (i.e., misses, false-positives) in recognition for neutral, depressing and elating statements. Errors for depressing and elating statements were decreased in the alcohol group relative to the placebo group. Group means did not differ for neutral statements.


4.5
3.0
1.5
0.0
Statements recalled
Alcohol
Placebo
Neutral
Depressing
Elating
Category


Alcohol
Placebo
Errors
8
6
4
2
0
Neutral
Depressing
Elating
Category

Postscript to Study 1 and preamble to Study 2

The first study was important in that it demonstrated that alcohol enhanced incidental memory for stimuli that participants themselves had subjectively appraised. Further, alcohol's effects were independent of the emotional valence of the stimuli. This suggested that the nonspecific enhancement view (an hypothesis advanced, but not tested by Hewitt et al., 1996; Lamberty et al., 1990; Mann et al., 1984; and Parker et al., 1980, 1981) was supported for the incidental paradigm. Alcohol enhanced memory much as the sucrose injections described in our laboratory rat at the outset of this thesis. The incidental paradigm used in this experiment involved contiguous presentation of alcohol: Alcohol was administered close in time to the verbal stimuli, but was not explicitly linked or associated with the stimuli. Memory was enhanced despite this lack of contingency. Also, Study 1 showed that the memory effects were independent of the individual psychophysiological response to alcohol -- indicators of alcohol's incentive properties (Pihl & Peterson, 1995).

Because there was no contingency between alcohol and memory, and because negative memory was enhanced as well as positive and neutral memory, alcohol's incentive properties were unlikely to be implicated. Further, the experiment showed that our incidental paradigm modelled the posttraining experiments used for alcohol (Alkana & Parker, 1979) and other drugs in animals. Our extrapolation of the White and Milner (1992) animal model to the effects of alcohol on memory in people seemed at least in part to be supported.

This was the first report to examine the effects of alcohol on emotionally salient memory. Salience was judged by participants themselves, with some needed data-checks according to Velten's (1968) categories. Although the materials were not used for the purposes of "mood induction" per se as they were originally used by Velten, it was hoped that the self-referent content would at least be "emotionally significant memories" for participants. Judging from their ratings, and their recall and recognition, this appears to have been the case. Notably, we did not "ask" participants to rate their mood, since they were exposed to more than one statement type.

The Velten Mood Induction Procedure was intended by Velten (1968) for use such that participants would be exposed to only one of the three statement categories, and a "mood" change would occur following this exposure. In that context, the alleged mood-inducing properties have been criticized by some authors as nonspecific (Cairns & Norton, 1988) or merely an artifact of self-report and experimenter demand (i.e., "asking" participants to rate their mood; Buchwald, Strack, & Coyne, 1981; Lewis & Harder, 1988). A review of over 46 studies (Kenealy, 1986) concluded that much of the debate seems to have resulted from "imprecise" administrations of the original procedure. Other papers (Berkowitz & Troccoli, 1986; Blackburn, Cameron, & Deary, 1990; Martin, 1990; Polivy & Doyle, 1980; Riskind, Rholes, & Eggers, 1982; Teasdale & Taylor, 1981), including a recent meta-analysis (Larsen & Sinnett, 1991) demonstrated that in the seventy-plus studies in which the VMIP has been used, there are indeed significant mood shifts in the directions predicted (*in addition to* experimenter demand effects and *despite* method of reporting and assessing mood). This suggests the statements do indeed have

an emotional salience and "charge" in the intended directions. Further, the word content of the statements have been validated according to emotional valence (Whissell & Levesque, 1988). It thus appears that our materials have the appropriate emotional validation on top of the face-validity offered by our own participants' ratings.

Participants rated the materials in the first session, and memory was tested in the second. Had participants also rated the materials again, changes in subjective ratings from day 1 to day 2 and their relation to memory, could have been studied. This was an unfortunate omission. Other limitations of the study include a restriction to verbal stimuli (i.e., omission of visual stimuli), the generalizability of the results given the demographically homogeneous sample, and the modest sample size.

As in most experiments in this area, the results were not perfectly as predicted. We found that alcohol did not improve recognition of neutral material. Further, we unexpectedly found that individuals who report a tendency to drink to cope with negative feelings had poorer performance on the recognition task, suggesting that individual differences (or their interaction with alcohol's incentive coping effects) predicted the "forgetting". The possibility that other, perhaps more reliable, individual differences might also mediate the effects of alcohol on incidental memory was raised. Investigation of this possibility was the object of Study 2.

A wide range of individual differences was selected since this was to be the first study of its kind. Individual differences determine drinking patterns (Annis, Graham, & Davis, 1987), and quantity and frequency of drinking (Pihl, Peterson, & Finn, 1990). Thus, if the effects of alcohol on incidental learning have implications for drinking

motives or incentives, then individual differences should be linked to the effects of alcohol on incidental memory.

Study 2

All that is needed is to discover

the laws of nature, then man will

no longer be answerable

for his actions and everything will

be exceedingly easy

-- Fyodor Dostoevsky

Study 2

Alcohol and retrograde memory effects:

Role of individual differences

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This research was supported by grants from the Medical Research Council of Canada to R.O. Pihl, and by a personal fellowship from the Fonds pour la formation de chercheurs et l'aide de la recherche (Québec) to K.R. Bruce. We thank Karen Milligan for assistance collecting, Tsafrir Vanounou and Maria Arvanitakis for assistance entering data.

Note: in press at the *Journal of Studies on Alcohol.*

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Abstract

Objective: When alcohol is consumed following learning, the effect on delayed, sober memory can vary from person to person. We examined a range individual differences to look for predictors of this variability. ***Method***: Sixty-five male social drinkers (average age 23.3 years) were exposed to emotionally-charged verbal stimulus materials while sober. Participants consumed 1.0 ml/kg alcohol immediately afterward, and remained in an environment designed to minimize retrograde interference. Stimulus recall and recognition were tested twenty-four hours later, when participants had breath-alcohol concentrations of zero. Relationship between memory scores and individual differences (in age, education, alcohol consumption, vocabulary, verbal learning, emotionality, mood state 24 hours after learning, response to alcohol, personality, and alcohol expectancies) were determined. ***Results***: Only age and vocabulary were significantly associated with memory score following drinking, likely because they constrained initial understanding of the statements, and mediated the effects of alcohol on memory consolidation. ***Conclusions***: The effects of a given dose of alcohol on emotionally-charged verbal memory are similar for men of equal age and verbal skill, but independent of other individual differences. Alcohol likely affects incidental memory by nonspecific enhancement or interference processes.

Placebo-controlled studies have shown that alcohol can produce retrograde

improvements in both visual (Hewitt et al., 1996; Mueller et al., 1983; Parker et al., 1980; 1981) and verbal (Bruce and Pihl, in press; Kalin, 1964; Lamberty et al., 1990; Mann et al., 1984; Parker et al., 1981; Tyson and Schirmuly 1994) memory. The effect is observed whether the learning is intentional or incidental, whether stimuli are emotionally-charged or neutral, and, in most cases, whether memory is tested by recall or recognition. The mechanism(s) responsible for the improvement are unknown. Reduction of retrograde interference (Lamberty et al., 1990; Mann et al., 1984; Mueller et al., 1983; Parker et al., 1980; Tyson and Schirmuly, 1994) has been proposed. Nonspecific enhancement of memory trace consolidation (Hewitt et al., 1996; Kalin, 1964; Lamberty et al., 1990; Mann et al., 1984; Parker et al., 1980; 1981) and mediation by the conditioned incentive effects of alcohol (Esposito et al., 1984; Lamberty et al., 1990; Mann et al., 1984; Parker and Weingartner, 1984) have been hypothesized. Finally, that the effect may be mediated by individual differences in experimental participants, or their interaction with alcohol (e.g., Kalin, 1964) has been suggested. This hypothesis is important for two reasons. First, it is unknown which individual differences, if any, might be implicated. Would individual differences in basic demographics and cognitive skills mediate? Learning? Mood state? Personality? Beliefs about, or response to, alcohol? Second, although the placebo-controlled studies have shown alcohol-placebo group differences, there is substantial variability within each group, and considerable overlap between them. Thus, are there individual differences responsible for the variability in memory effects following alcohol consumption?

We attempted to examine the role of individual differences in mediating the

variability in memory scores for participants who had consumed alcohol. It was expected that if individual differences did mediate, they would be associated with subsequent memory scores. If they did not mediate, then the effects of alcohol on memory should be similar across individuals who vary on the measures. Individual differences including gender (Haut et al., 1989) intelligence (Maylor et al., 1990), familiarity with task (Rumbold and White, 1987) or environmental cues (Miles et al., 1986) have been shown to affect acquisition and retrieval while participants are intoxicated. However, the role for individual differences in determining the post-learning alcohol-memory interaction is largely unknown. Our own previous study (Bruce and Pihl, in press) found no mediational role for drinking-induced resting psychophysiological changes, but suggested a negative association between a questionnaire measure of *drinking in response to negative feelings* and *incidental memory following drinking*. However, since those individuals forgot more of positive as well as negative memories, the question of more reliable individual differences was raised. Our selection of variables was based on whether they have been shown or were suspected to mediate verbal memory performance and/or individual response to emotional stimuli, and/or alcohol. In this regard, we included demographics, verbal memory, subjective ratings of the stimuli, mood state, personality, response to alcohol, and alcohol expectancies.

Materials and methods

Participants. Sixty-five men between the ages of 18 and 30 were recruited

through newspaper advertisements. Each was required to never have met *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association 1994) criteria for alcohol dependence or abuse, and to have a score of less than 5 on the brief version of the Michigan Alcoholism Screening Test (BMAST; Pokorny et al., 1972). Individuals were also asked to report about their family members, and were excluded if their mother, or father, or paternal grandfather, or any two other relatives had a BMAST score above 4. Individuals ever treated for a major psychiatric disorder, having a history of traumatic head injury, or a current major health condition requiring a prescription were also excluded. Each was asked to refrain from alcohol and illicit drugs for at least 24 hours and to refrain from food for at least 4 hours before the first session. They were asked to refrain from alcohol and drugs between sessions. Participants were tested individually. Reimbursement was $5 per hour.

Materials. Statements used in the memory paradigm (Velten 1968) were of two categories, *Elating* (e.g., "I have a sense of power and vigor.") and *Depressing* (e.g., "I've doubted that I'm a worthwhile person."). The emotional tone of these materials has been well validated (e.g., Velten 1968; Whissell and Levesque 1988), and we have shown in a previous experiment (Bruce and Pihl, in press) using a similar paradigm and participants that alcohol enhances memory for these statements relative to placebo: In that study the alcohol group recalled an average of 11.3 percent of the original statements while the placebo group recalled an average of 7.1 percent; for recognition, 14.6 percent of the alcohol group's responses were errors, while 22.8 percent of the responses made by the

placebo group were errors. In the current experiment, fifteen statements from each category were randomly selected, and laser-printed onto index cards. Recognition test distractor cards were taken randomly from the remaining statements. Subjective ratings (negative-positive) of the statements was rated on a 14-point (-7 to -1; 1 to 7) scale. The alcohol was 95% U.S. proof (USP). The dose was 1.0 ml per kg of body weight, mixed with 5 parts orange juice. Alcohol was administered in a large plastic cup. An Alco-Sensor III (Intoximeters, Inc.) was used to determine breath alcohol concentration (BAC).

Demographics. Participants' age, years of education and monthly alcohol consumption were obtained. The WAIS-R Vocabulary scale was used as a demographic measure of verbal IQ; raw scores were used. Verbal intelligence has been shown to mediate verbal learning in intoxicated participants (Maylor et al., 1980). To our knowledge, there is one study (Lamberty et al, 1990) showing vocabulary can mediate alcohol's retrograde effects on consolidation. We were unable to find studies showing vocabulary to mediate subjective or physiological response to alcohol or emotional stimuli. We suspected vocabulary might mediate verbal memory directly (or perhaps mediate alcohol's effects on memory) rather than through mediating emotional or physiological responses.

Verbal Memory. The Logical Memory test (Wechsler Memory Scale; Form I) measures immediate prose recall. Scores for stories A and B were averaged. It was expected that if memory for verbal material mediated our effects, this score should be predictive beyond Vocabulary.

Profile of Mood States (POMS; Lorr and McNair 1982). An extensive literature has shown mood state to influence memory (Blaney 1986; Bower 1981; Fiedler and Stroem

1986; Teasdale and Fogarty 1979). It is important to note that state-dependent changes from acquisition to retrieval were expected to be minimal since participants were sober at both times; mood at acquisition was not measured, as doing so (a verbal-emotional task) may have interfered with acquisition of the emotionally-charged materials even prior to drinking. We expected the POMS would reflect the influence of individual differences in state emotional responsivity (e.g., related to possible alcohol withdrawal or other factors).

Eysenck Personality Questionnaire (EPQ). These scales predict response to alcohol (Brown and Munson 1987; Hammersley et al., 1994; Netter et al., 1994; Ruch, 1994), consumption patterns (Allsopp 1986; Lester and Rassas 1986; Sher and Trull 1994), emotional reactivity (Larsen and Ketelaar 1991), and memory performance for verbal (Bermudez et al., 1988; Gabrys et al., 1987; Gupta and Kumar 1990) and emotional (Bradley and Mogg 1994; Matthews et al., 1995) stimuli. Thus, association between Eysenck's dimensions and memory score could occur for any one or combination of these reasons. Extraversion, neuroticism, and psychoticism scales were used.

Biphasic Alcohol Effects Scale (BAES; Martin et al., 1993). We hypothesized that subjective response to alcohol might predict the interaction between alcohol and memory. The BAES has two empirically-derived subscales: stimulant (ST) effects and sedative (SE) effects. Stimulant effects are more pronounced on the ascending limb (AL) of the blood alcohol curve; sedative effects are more pronounced on the descending limb (DL) of the blood alcohol curve (Martin et al., 1993). Participants fill out the complete scale on both limbs. The four measures are ALST, ALSE, DLST, and DLSE.

Alcohol Expectancy Questionnaire (AEQ; Brown et al., 1987). Alcohol expectancies mediate cognitive and subjective responses to alcohol (e.g., Fillmore and Vogel-Sprott 1995), consumption patterns (Brown et al., 1980), and are built on memory constructs (Brown et al., 1987; Goldman et al., 1991). The adult form of this questionnaire, is widely-used to measure positive outcome expectancies. Each of the six subscales reflects expectations for desirable alcohol-induced outcomes.

Procedure. Participants were contacted and screened by telephone. Testing was divided in two sessions on consecutive days. *Session 1.* Upon arrival, participants read and signed an informed consent. They were weighed, and had an initial breathalyser reading taken to ensure a BAC of zero. We did not tell participants explicitly to learn or remember the material, only that they would be rating the 'emotionality' of some statements, and afterward they would consume alcohol. Rating (and stimulus exposure) occurred in a card sorting task. Statements were presented one at a time, in a standard order, in 20-second intervals. Participants were required to read the statement aloud, imagine themselves in the situation described, and to give their response by sorting the card to one of two piles (*positive* and *negative).* The original Velten category was not disclosed. To ensure compliance, we required that participants sort a minimum of two-thirds of the statements from each of category 'correctly' (i.e., Depressing to the negative pile, and Elating to the positive pile). In addition, participants were asked to subjectively rate each statement. Immediately after all cards had been sorted, participants consumed the alcohol, which they were required to finish within 20 minutes. After card sorting,

participants sat in a minimally-decorated room on a comfortable sofa and listened to soft classical and jazz music. Participants were prevented from smoking, sleeping, reading or working, and were told that the experimenters would have minimal contact with them during the drinking and intoxication phase, except to monitor their progress and take BAC readings. The first BAC reading was taken 10 minutes after the 20-minute drinking period. Readings 2 through 6 were taken every ten minutes thereafter. Readings were then taken every 30 minutes for 3 to 4 additional readings. Participants were then permitted to leave the laboratory. No feedback concerning BAC was given to any participant until the end of the experiment. *Session 2.* Participants first gave a BAC reading to ensure it was zero. They then completed the recall test. They were asked to write down as many of the statements from the previous session as possible within 10 minutes. Participants were encouraged to remember as much as they could word-for-word where possible. In the recognition test which followed, 10 target stimuli from each category were randomly selected from the correctly-sorted statements in Session 1. To these, 10 depressing and 10 elating distractors were added. Participants were presented with the 40 randomly-shuffled cards, one card at a time, at their own rate. They were asked to indicate if they had seen the card in the previous session. Errors were recorded as misses and false positive responses (Bruce and Pihl, in press). Following the recognition test, WMS Logical Memory test was administered. Participants then filled out the packet of remaining questionnaires. At the end of the experiment, they were debriefed, and reimbursed.

Results

Statistical procedures. The relationships among memory scores and individual-difference variables were examined by (a) creating clusters of participants according to performance (better/worse) on memory scores and then (b) comparing the clusters on the mediator variables of interest. In this way, variables associated with better vs. worse memory scores would be identified. However, the data were first inspected and prepared for these analyses (Tabachnick and Fiddel, 1989). The frequency distribution for each variable was examined. Univariate distributions were analyzed for extreme outliers 3.3 SD from the mean (alpha = .001). These were brought in toward the next nearest value (Tabachnick and Fiddel 1989). Inspection of the resulting distributions using skewness and kurtosis criteria (alpha =.001) revealed satisfactory normality for all but two variables: Participants' monthly alcohol consumption was *square-root* transformed, and Vocabulary was *square* transformed (Kleinbaum et al., 1988) to correct for normality. Participants' recall and recognition were scored by a single rater (e.g., Kalin, 1964) blind to other results. Half the recall protocols were scored by a second rater blind to other results. The inter-rater correlation across category of statement was 0.71 ($p<.00005$). For recall, participant responses that accurately resembled the word content of an original stimulus statement were scored. Accurate resemblance was determined as containing at least three of the same key content words (or close derivatives) as the original Velten statement. Such gist criteria are commonly used in the literature (e.g., Goetz et al., 1981). The four measures were free recall (FR) and recognition (RE) of depressing (D) and

elating (E) statements: FR_D, FR_E, RE_D and RE_E.

Cluster analysis. Recall and recognition scores were subjected to a cluster analysis. Results are shown in Table 1. Two significantly different clusters were formed. Cluster 1 had relatively higher scores on FR_D and FR_E, and lower scores on RE_D and RE_E. This cluster had members with higher recall scores, and lower recognition error scores. Cluster 2 had converse results, and is thus comprised of members with lower recall, and higher recognition error scores. The multivariate effect comparing clusters on memory measures was highly significant $F(4,60)=36.73$, $p<.0005$, effect size=.71, power=1.00. As well, univariate F-tests (all 1,63 df) for FR_D=16.65, for FR_E=14.07, for RE_D=86.08, and for RE_E=50.71, were all significant at $p<.0005$. (--------- Table 1 about here ----------)

Measures that potentially mediate memory performance following alcohol consumption. To then examine for variables that might underlie memory performance, the clusters were compared on using MANOVA or ANOVA (1-way or repeated measures) as appropriate.

Demographic measures. The clusters were examined for differences in age, years of education, alcohol consumption and Vocabulary using a MANOVA. Means and standard deviations are shown in Table 2. The multivariate effect of cluster was significant ($F(4,60)=6.63$, $p<.0005$, effect size=.31, power=.99, see Table 3). This effect was explained by univariate cluster effects for Vocabulary ($F(1,63)=24.80$, $p<.0005$; power=1.00) and age ($F(1,63)=5.52$, $p<.023$; power=.64) but only the Vocabulary effect was significant after a Boneferroni correction. Cluster 1 had higher Vocabulary scores,

and was younger, than cluster 2. Univariate effects showed the clusters did not differ significantly in years of education or monthly alcohol consumption. (——— Tables 2 and 3 about here ----------)

Verbal memory. The clusters were examined for differences in memory for verbal stimuli using a 1-way ANOVA on Logical Memory. We included the demographic differences in Vocabulary and age as covariates since the clusters differed on these more basic measures. Age did not enter as a covariate, so it was removed, and the analysis was redone. The covariate effect of Vocabulary was significant ($F(1,62)=9.12$, $p<.004$), but the effect of cluster was not (Table 3). Thus, memory for verbal stimuli did not differ between clusters equated for vocabulary.

Subjective ratings. The clusters were examined for differences in initial ratings of the statements using a 1-way ANOVA on ratings. Since the depressing and elating memory was related, and statement subjective ratings were correlated ($r=-0.46$, $p< .0005$), ratings of the two were averaged (absolute values on the respective 1 to 7 scale). The covariate effect of both Vocabulary and age ($F(2,61)=7.71$, $p<.001$) was significant. Beyond this, there was no cluster difference in subjective ratings (Table 3). Average rating for cluster 1 was mean $\pm$ SD 4.42 ± 0.73, and for cluster 2 was 4.54 ± 1.09. In addition, the proportions of incorrect sorts did not differ between clusters for depressing or elating statements as per separate Mann-Whitney tests.

Response to alcohol.

1. BAC. The clusters were examined for differences in BAC over time (i.e., limb, slope, peak, rate, etc.) using an ANOVA with time (at 10, 20, 30, 40, 50, 60, 90, and 120

minutes post-alcohol consumption) as a repeated measures factor. Age and Vocabulary were included as covariates, but their effect was nonsignificant, so the ANOVA was redone without them. Neither the cluster X time interaction, nor the effect of cluster was significant. Statistics are shown in Table 3. Clusters were thus not compared at, or between, individual times.

2. BAES. The clusters were examined for differences in subjective response to alcohol using a MANOVA on the BAES (with ALST, ALSE, DLST, and DLSE subscales as univariate measures). Age and Vocabulary were included as covariates but their effect was nonsignificant, so the MANOVA was redone without them. The cluster effect was not significant indicating the BAES responses did not differ between clusters. Statistics are shown in Table 3.

Mood state on day 2. The clusters were examined for differences with a MANOVA on the POMS -- with the six POMS subscales as univariate measures. Age and Vocabulary were included as covariates, and their effect was significant ($F(12,114)=2.65$, $p<.004$; effect size=.22, power=.97). The cluster effect was *not* significant (Table 3), indicating the POMS did not differentiate clusters equated for age and Vocabulary.

Personality. The clusters were examined for differences in Eysenck's dimensions using a MANOVA (with extraversion, neuroticism and psychoticism as univariate measures). Age and Vocabulary were included as covariates, but their effect was nonsignificant, so the MANOVA was redone without them. The cluster effect was not significant indicating the EPQ did not differ between clusters. Statistics are shown in Table 3. Thus, cluster comparisons on individual EPQ dimensions were not done.

Alcohol expectancies. The clusters were examined by MANOVA on the AEQ -- with the six AEQ subscales as univariate measures. Age and Vocabulary were included as covariates, but the effect was nonsignificant, so the MANOVA was redone without them. The cluster effect was not significant, indicating the AEQ did not differentiate clusters. Statistics are shown in Table 3.

Discussion

Two important points need to be made before the role of individual differences in the memory results can be discussed. First, recall and recognition scores clustered together. This allowed us to streamline the presentation of results by comparing participants who performed well on both tasks to those who performed more poorly on both tasks. And, any limitation due to subjectivity or unreliability in the recall measure is therefore complemented by the more objective recognition results. Second, memory for depressing and elating statements was similarly affected (i.e., clustered together). This suggests that memory for depressing statements was as good as memory for elating statements; there was little indication that participants remembered more from one statement category than the other. This meant that any individual differences in participants that predicted depressing memory would also likely predict elating memory, and vice-versa. These results are highly interesting given our previous work with a similar paradigm and sample (Bruce and Pihl, in press) that showing alcohol enhanced incidental memory for depressing and elating statements relatively equally compared with placebo.

Indeed, the groups in the current experiment recalled and recognized similar percentage amounts of the original stimuli as the alcohol group in the prior experiment. This suggests that the nonspecific enhancement drug actions of alcohol on incidental memory may have been similar in the two experiments.

As for the role of individual differences in the memory results, the principal findings were as follows. First, the primary result was that only participants' age (modestly) and Vocabulary (moderately) significantly predicted memory scores. Memory skills, and the effects of alcohol, are widely known to differ with age, and the modest effect observed here merits further examination; it appears that studies looking at the effects of alcohol on retrograde memory events need to control for it carefully. Vocabulary skills per-se may constrain understanding (it is easier to remember something to the degree that it is understood), as well as alcohol's memory consolidation effects (Lamberty et al., 1990). It would appear likely that prediction of memory scores by verbal skills was probably accounted for by one of these explanations. Also, because the covariate analyses were significant, it is also possible that Vocabulary mediated memory scores via constraining verbal memory, accurate subjective appraisals, or mood state on day 2. However, since the effect of these variables themselves was negligible, this possibility is unlikely.

The second finding was that none of the other variables examined herein seemed to be associated with memory scores. It appears that differences in initial appraisals of the stimuli, response to alcohol, verbal memory while sober, mood state, personality, and alcohol expectancies did *not* predict the memory effects observed. For mood state, for

example, this may mean that we can rule out emotional effects of alcohol withdrawal (e.g., Lemon et al, 1993), or post-intoxication emotional response biases (e.g., Tyson and Schirmuly, 1994), or other emotional factors, in the memory variability. Of the other variables studied herein, personality (Allsopp 1986; Lester and Rassas 1986; Sher and Trull 1994) and alcohol expectancies (Brown et al., 1980) have been associated with many of alcohol's important individual-difference effects. It appears, however, that these do not include alcohol's effects on incidental memory consolidation. This is important given that memory processes are central to some models of consumption (Conger 1956; Wise and Bozarth 1987) and expectancy (Goldman et al., 1991; Stacy et al., 1994).

A parsimonious explanation for the current results appears to be that alcohol interacts with incidental learning in a similar manner across individuals. Alcohol may thus enhance (as the placebo-controlled studies suggest) incidental cues nonspecifically, be they depressing or elating. Individual differences simply do not predict the effects of alcohol on incidental memory. However, *incidental* learning differs markedly from other kinds of learning paradigms. For example, there is the *intentional* learning paradigm, which requires that participants explicitly be asked to remember material. Recent placebo-controlled work from our own laboratory (Bruce et al., submitted) indicates that some individual differences (e.g., in subjective ratings and heart rate response to alcohol) can in fact predict the effects of alcohol on *intentional* memory; alcohol consumption is modestly predicted here as well. These relationships may, in turn, reflect alcohol's putatively *conditioned incentive* properties (Pihl and Peterson, 1995; White 1996). Thus, there is converging preliminary evidence here that suggests while individual differences

in the effects of alcohol on memory may thus be important in mediating the conditioned incentive effects of alcohol on intentional learning, they do not do not mediate the nonspecific enhancing effects of alcohol on incidental learning. The parameters that implicate individual differences have begun to be delineated.

Two aspects of this experiment need brief explanation as they may be perceived as limitations. The first concerns the absence of a placebo group. Without such a group, we cannot *directly* differentiate the components of our task (i.e., *1* - responding emotionally to the material; *2* - verbal ability and *3* - verbal memory; as well as response to the effects of alcohol on *4* - physiology, *5* - emotion, and *6* - incidental memory). However, our external control measures may allow us to infer, albeit indirectly, the separate roles for each individual component. Our controls have shown that components *1* , *3*, *4* and *5* (via initial ratings, Logical Memory, BAC and BAES controls respectively) did not differ between clusters, and thus were not implicated in the memory results. We are left with, among other factors untested here, to be sure, components *2* - individual differences in verbal ability, and *6* - individual differences in the effects of alcohol on memory. Thus, our design controls may make possible the inference that only Vocabulary and age predicted (a) the interaction between alcohol and emotionally salient memory, and possibly, (b) the *effects of alcohol* on incidental memory consolidation. Examination of this possibility with the appropriate control group will undoubtedly answer the question more directly. The second limitation of the study is the constraint imposed by the demographics of the sample: young, non-alcoholic men with above average education. Education was found to not play a significant role, but the findings should be replicated

in other samples.

In summary, we find that individual differences beyond age and verbal abilities do not mediate the interaction between alcohol and incidental retrograde memory. Alcohol had similar effects on memory for men of similar age and equal verbal skill, regardless of individual differences in emotionality, response to alcohol, personality, alcohol expectancies, or mood state at retrieval. And, because depressing and elating memories were equally recalled, the alcohol-memory interaction is more likely mediated by consolidation or interference processes not selective of statement type or any related incentive properties. However, basic mental abilities in the memory domain under investigation as well as age must be controlled for in future retrograde enhancement studies.

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Table 1. Results of cluster analysis based on memory scores.

Final Cluster Centers

Cluster	n	FR_D	FR_E	RE_D	RE_E
1	37	2.38	2.24	1.19	1.24
2	28	1.18	1.21	3.93	3.39

Note: FR=number of free recall responses; RE=Number of recognition errors; D=Depressing statements; E=Elating statements. Centers also represent cluster means on the measure indicated. Cluster 1 had better performance on all measures relative to cluster 2.

Table 2. Demographic variables by cluster.

	Cluster 1		Cluster 2	
Measure	Mean	SD	Mean	SD
Age	22.41	3.27	24.50	3.91
Years education	14.59	2.19	14.74	3.42
Alcohol consumption (drinks/month)	27.06	21.70	29.11	30.15
Vocabulary	56.70	7.86	42.86	14.32

Note: Clusters differed on MANOVA. Univariate analyses suggested that age and Vocabulary accounted for the multivariate cluster difference.

Table 3. Cluster comparisons on demographic variables, verbal memory, subjective response to stimuli, response to alcohol, mood state on day 2, personality, and alcohol expectancies.

Covar	*F*	*df*	*p<*	*Pwr*	*ES*	*Test*	*Effect*	*F*	*df*	*p<*	*Pwr*	*E*
						Demographic Variables						
						MANOVA	all	4.63	4,60	.0005	.99	13.
						uANOVA	Age	5.52	1,63	.023	.64	
						uANOVA	Edu	.04	1,63	ns	.04	
						uANOVA	Voc	24.80	1,63	.0005	1	
						uANOVA	D/M	.00	1,63	ns	.04	
						Verbal Memory						
Voc	9.12	1,62	.004			uANOVA	LMem	.37	1,62	ns		
						Subjective rating of stimulus materials						
Voc,Age	7.71	2,62	.001			uANOVA	SuR	2.67	1,61	ns		

Table 3. continued

Covar	*F*	*df*	*p<*	*Pwr*	*ES*	*Test*	*Effect*	*F*	*df*	*p<*	*Pwr*	*E*
							Response to Alcohol					
Voc,Age	1.71	2,61	ns	.14	.70	rANOVA	BACxCl	.55	7,441	ns		
							BAC	.19	1,63	ns		
Voc,Age	.72	8,118	ns	.32	.05	MANOVA	BAES	1.52	4,60	ns	.44	90.
							Mood state on day 2					
Voc,Age	2.65	12,114	.004	.97	.22	MANOVA	POMS	.78	6,56	ns	.28	80.
							Personality					
Voc,Age	1.49	6,120	ns	.56	.07	MANOVA	EPQ	1.36	3,61	ns	.34	60.
							Alcohol Expectancies					
Voc,Age	.74	12,114	ns	.41	.07	MANOVA	AEQ	.43	6,58	ns	.16	40.

Table 3. continued

Note 1: Covar=covariate(s); Effect=Clusters compared on the measure indicated. For example BAC represents main effect of cluster on the measure BAC, BACxCl represents the BAC by Cluster interaction, while EPQ represents MANOVA effect of cluster on the EPQ, etc.; ES=Effect size; Pwr=Power rANOVA=repeated measures ANOVA; uANOVA=univariate ANOVA effect

Note 2: AEQ=Alcohol Expectancy Questionnaire; BAC=breath alcohol concentration; BAES=Biphasic Alcohol Effects Scale; Cl=Cluster; D/M=alcohol consumption in drinks per month, square-root transformed; Edu=Years of education; EPQ=Eysenck Personality Questionnaire; LMem=Logical Memory; POMS=Profile of Mood States, administered Day 2; SuR= subjective rating of stimulus materials; Voc=Vocabulary, square-transformed

Postscript to Study 2 and preamble to Study 3

Study 2 demonstrated that individual differences in basic vocabulary skill and participants' age predicted the interaction between alcohol and incidental memory. Response to alcohol, emotional responsiveness, personality, alcohol expectancies and other demographic variables did not mediate the effects of alcohol on incidental memory. These results have implications for Kalin's (1964) conjecture that individual differences might mediate the retrograde effects of alcohol on memory. The conjecture was only minimally supported as far as *incidental* memory was concerned. The possibility of the role of the same variables would mediate the effects of alcohol on *intentional* memory was to be examined in Study 3.

This study replicated the Study 1 finding that the effects of alcohol on the amount of depressing and elating statements remembered was quantitatively similar. Our extrapolation of White and Milner's (1992) model seemed to hold again since the memories were nonselectively affected.

As pointed out by journal reviewers of this manuscript, the lack of a placebo group prevented a "direct" separation of individual differences *in memory* from individual differences *in the effects of alcohol on memory*. The separation was instead inferred by analyses of separate measures of memory, and separate measures of response to alcohol and emotion. An analogy to brain imaging studies is useful here. In positron emission tomography (PET) experiments, so-called "task subtraction" techniques are often used to separate simple components from more complex ones (e.g., separating attention from

memory; Petrides, Alivisatos, Evans, & Meyer, 1993; Petrides, Alivisatos, Meyer, & Evans, 1993). Examining the relationship between external measures of the task components and memory performance, we conclude that only basic verbal skills mediated the effects of alcohol, likely because this constrains initial understanding of the statements, and possibly because it affects alcohol's consolidation of memory.

The finding that the clusters differed on relatively few measures might be construed by some as "negative" findings. Instead, however, they may be at least partly explained in part by the incidental paradigm. Studies 1 and 2 demonstrated that alcohol's effects on incidental memory are nonspecific to stimulus type. It may be that non-contingent presentation of alcohol and stimuli as in the incidental paradigm does not implicate individual participant differences. In this regard, a larger sample size had been used in this study to ensure adequate statistical power so the conclusion could be reasonably drawn. The "lack" of involvement of individual differences makes sense if we think of alcohol having a somewhat uniform effect on incidental memory across people. In this regard, it is not surprising to think that incidental memory processes do not link individual differences to the effects (incentive or memory) of alcohol.

Study 3 replicated many of the procedures of Studies 1 and 2, with the important manipulation of changing the memory paradigm to an intentional one. In so doing, we hoped to create an explicit relationship between alcohol (and/or its effects) and the memory task. A "cognitive" contingency (Norman White, personal communication, April, 1997) was thus established in the sense that participants could easily deduce that the experiment was an examination of what alcohol "might do" to memory. The effect of

alcohol on memory was investigated with the expectation of implicating a contingency with alcohol's well known incentive effects (psychomotor stimulation/heart rate changes, and mood amelioration and enhancement).

Study 3

The more a man can forget,
the greater the number of
metamorphoses which his life
can undergo; the more he
can remember, the more divine
his life becomes

-- Soren Kierkegaard

Study 3

Effects of Alcohol Consumption on Intentional Memory and Subjective Ratings For Emotionally Charged Material

Note: Under review at *Experimental and Clinical Psychopharmacology*

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Abstract

Social drinkers (44 men, 18-30 years old) participated in a study of the effects of alcohol consumption on intentional memory and subjective ratings for emotionally salient verbal stimuli. Participants learned the materials while sober. Five min later they consumed alcohol or active placebo (1.0 or 0.1 ml/kg) in an environment with minimal retrograde interference. When participants were again sober, 24 hr later, the alcohol group had increased recall of positive material, and decreased recall of negative material. Increased recall of positive over negative material was predicted by heart rate response to alcohol and positive changes in subjective ratings. Results suggest that alcohol's incentive effects were implicated in its effects on intentional memory.

Although alcohol impairs new learning, it improves recent memory. The improvement, modest but fairly reliable, has been shown in several experimental paradigms where initial learning is followed by alcohol consumption and memory testing occurs when individuals are once again sober. For example, memory for visual-motor performance (Hewitt, Holder, & Laird, 1996; Mueller, Lisman, & Spear, 1983), and photographic slides (Parker et al., 1980) are improved when alcohol consumption follows learning. Importantly, the visual memory facilitation is dose-dependent (Parker et al., 1981). Verbal memory is also improved when alcohol consumption follows learning (Lamberty, Beckwith, Petros, & Ross, 1990; Mann, Cho-Young, & Vogel-Sprott, 1984; Parker et al., 1981; Tyson & Schirmuly, 1994). Notably, the verbal memory improvement correlates with the amount of alcohol participants have consumed (Kalin, 1964).

In these experiments, the improvement occurred in most cases whether memory was tested by recall or by recognition. Further, the improvement occurred when learning occurred explicitly, (i.e. when the experimenter asked participants to remember). Improvement also occurred when learning occurred incidentally, (i.e., when the experimenter did not explicitly ask participants to remember and subsequent memory testing was thus a surprise for them). Also, memory improvement occurred despite the fact that the material participants had learned had no obvious emotional meaning for them.

Thus, it is of interest to note that postlearning alcohol consumption also improves subsequent memory for material judged subjectively by experimental participants to have

an emotional "charge". Improvement of emotionally charged, i.e., negative, positive, etc., material has been demonstrated in an incidental learning paradigm (Bruce & Pihl, in press). In that study, alcohol improved memory for both negatively- and positively-charged verbal materials. To our knowledge, the effects of alcohol consumed after exposure to emotionally charged memory have yet to be investigated in an intentional learning paradigm.

Further, the mechanisms responsible for these intriguing postlearning effects of alcohol are not yet clear. Four primary hypotheses have been suggested. First, reduction of retrograde interference by alcohol may be implicated (Lamberty et al., 1990; Mann et al., 1984; Mueller et al., 1983; Parker et al., 1980; Tyson & Schirmuly, 1994). Second, the effect may instead reflect individual differences in experimental participants, or their interaction with alcohol (e.g., Kalin, 1964). Our own studies (Bruce & Pihl, in press: Bruce, Mayerovitch, Shestowsky, & Pihl, submitted) have suggested a possible role for individual differences in age, vocabulary skill and a self-report drinking measure; the three measures predicted alcohol's effects on incidental memory. The retrograde effects of alcohol on intentional memory for emotionally charged material has not yet been investigated.

Third, a nonselective enhancement of memory trace consolidation by alcohol may be implicated (Hewitt et al., 1996; Kalin, 1964; Lamberty et al., 1990; Mann et al., 1984; Parker et al., 1980, 1981). In this case memory for all types of stimuli would be improved by alcohol (e.g., Bruce & Pihl, in press). Fourth, postlearning effects might reflect the so-called incentive effects of alcohol (Esposito, Parker, & Weingartner 1984; Lamberty

et al., 1990; Mann et al., 1984: Parker & Weingartner, 1984). The so-called incentive effects of alcohol include a subjectively positive psychomotor stimulation on the ascending limb of the blood-alcohol curve (Connors & Maisto, 1979; Martin, Earleywine, Musty, Perrine, & Swift, 1993; Sano et al., 1993; Wise & Bozarth, 1987) and relief from anxious and depressed moods (Conger, 1956; Freed, 1978; Mayfield, 1968; Mayfield & Allen, 1967; Pihl & Peterson, 1992; Sayette, 1993; Williams, 1966). Thus, the incentive effects of alcohol would be reflected to the degree that postlearning alcohol consumption produced desirable memory outcomes.

The experiment described in this article was an examination of the effects of alcohol on the intentional memory and subjective ratings for emotionally charged verbal materials. While attempting to control for retrograde interference and individual differences, it was our hypothesis that if nonselective enhancement processes mediated, alcohol would increase both negative and positive memory. These memories would be (positively) correlated, and scores would not be predicted by alcohol's incentive effects. The effect would not be associated with changes in subjective ratings.

By contrast, if alcohol's incentive properties mediated, our hypothesis was that alcohol would produce memory outcomes with incentive value. This might occur if (a) alcohol facilitated desirable (i.e., positive) memories and/or inhibited undesirable (i.e., negative) ones, and (b) if this effect was associated with positive increases in subjective ratings. In addition, one or both of these effects might be related to previously established indices of alcohol's incentive properties.

Method

Participants

Forty-four nonalcoholic men (aged 18-30 years) were recruited through newspaper advertisements. Nonalcoholic status was defined as (a) never having met the criteria of the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*; American Psychiatric Association, 1994) for alcohol dependence or abuse and (b) a score of less than 5 on a shortened version of the Michigan Alcoholism Screening Test (MAST; Pokorny, Miller, & Kaplan, 1972). Participants were also assessed for family history of alcoholism using MAST criteria adapted for family members. Individuals with a diagnosis of alcoholism in their mother, or father, or paternal grandfather were excluded, as were those with a history of alcoholism in any two other relatives. Individuals who had ever been treated for a major psychiatric disorder, including substance abuse or dependence, schizophrenia, bipolar disorder, depression, and anxiety disorders were excluded. Participants also were excluded if they had a history of traumatic head injury or cardiac condition, or were currently taking prescription medication that contraindicated alcohol consumption.

Participants were asked to refrain from drinking alcohol and taking illicit drugs for at least 24 hr and from eating for at least 4 hr before Session 1. They also were asked to refrain from drinking alcohol and taking illicit drugs between sessions. All participants were tested individually and did not interact with one another. They were paid $5 per hour.

Apparatus

Materials used in the verbal learning paradigm were taken from Velten (1968). Two types of self-referring statements were used: depressing and elating. The valence, (i.e., subjective positivity-negativity), of these statements has been well validated (Velten, 1968; Whissell & Levesque, 1988). We printed the 120 (2 x 60) statements onto 7.72 cm x 12.70 cm index cards, one statement per card. The statements used in Session 1 (15 from each type) were randomly selected. The same 30 statements were used for all participants. Distractor items used in the recognition test were randomly selected from the remaining statements. Valence was rated on a 10-point Likert-type scale where 1=negative, 10=positive.

The alcohol used was 95% U.S. proof. In the alcohol condition, the dosage was 1.0 ml/kg, mixed with 5 parts orange juice. In the placebo condition, an "active" placebo (e.g., as recommended by Stewart, Finn, & Pihl, 1992) was used to disguise flavor; the dosage was 0.1 ml/kg mixed with 0.9 ml/kg Vichy brand mineral water; the alcohol-mineral water mixture was mixed with 5 parts orange juice. Drinks were served in a large plastic cup. A spray bottle was used to add trace amounts (maximum of 2 ml) of alcohol to the top of drinks. An Alco-Sensor III (Intoximeters, Inc., St. Louis, MO) was used to determine the breath alcohol concentration (BAC).

A Polar Electro (Kempele, Finland) Sport Tester PE300 portable heart rate monitor system was used to measure heart rate before and after drinking. The system has been shown to be reliable in recording resting heart rate when compared to a polygraph (r = .97, Bruce & Pihl, in press). Mean heart rate response to alcohol reflects a psychomotor

stimulant response to alcohol (Pihl & Peterson, 1995), and is thus a candidate biological marker for alcohol's effects on intentional memory.

The Biphasic Alcohol Effects Scale (BAES; Martin, Earleywine, Musty, Perrine, & Swift, 1993) measures subjective response to alcohol. We hypothesized that subjective response to alcohol might predict the interaction between alcohol and memory. The BAES has two empirically-derived subscales: stimulant (ST) effects and sedative (SE) effects. Stimulant effects are more pronounced on the ascending limb (AL) of the blood alcohol curve; sedative effects are more pronounced on the descending limb (DL) of the blood alcohol curve (Martin et al., 1993). Participants fill out the complete scale on both limbs. The four measures are ALST, ALSE, DLST, and DLSE.

Variables used for group matching

Thirteen participant measures were used to match the 2 groups and limit potential confounds (e.g., Bruce et al., submitted; Kalin, 1964) to the interpretation of the memory results. Age, years of formal education, and quantity of drinking (average weekly consumption over the past year) were obtained. The Vocabulary subtest of the Wechsler Adult Intelligence Scale-Revised (Wechsler, 1974) measures word knowledge and general verbal capacity. Raw scores were used. The Logical Memory subtest of the Wechsler Memory Scale, Form I (Wechsler, 1945) is a measure of immediate prose recall. Scores for stories A and B were averaged.

The Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975) contains three well-known basic personality dimensions: extraversion, neuroticism, and psychoticism

scales were used.

The Alcohol Expectancy Questionnaire (AEQ; Brown, Christiansen, & Goldman, 1987), adult form, is a well validated measure of positive alcohol-outcome expectancies. Total questionnaire scores were used.

The Inventory of Drinking Situations (IDS; Annis, 1982) is a self-report measure of drinking. The IDS contains subscales of drinking frequency associated with a variety of situations, including drinking in response to pleasant (IDS-P subscale) and unpleasant (IDS-U subscale) emotional states. The IDS, normed with clinical populations, was adapted for use in our nonalcoholic participants (who were expected to drink considerably less) by changing the heading at the top of the form to "I had at least one drink" from "I drank heavily". The initial subjective response to the statements, as defined by the number of incorrectly sorted cards, was also assessed.

Procedure

Participants were contacted by telephone and the MAST and *DSM-IV* alcoholism questions were administered in a brief interview. The actual experiment was divided into two testing sessions that occurred at the same time on consecutive days. Session 1 lasted 4-6 hr, and Session 2 lasted 2-3 hr. Participants were treated in accordance with the American Psychological Association's (1992) Ethical Principles of Psychologists.

Session 1

On arrival, participants read and signed an informed consent, were weighed and

had an initial breathalyzer reading taken to ensure a BAC of zero.

Learning paradigm

Participants were asked to try to remember as much of the materials as they could, and that their memory for the material would be tested the following day. In addition, they were told the experimenters were interested in studying potential effects of alcohol on the memory, but were not told that the experimenters expected any particular result. Learning of the statements also involved a card sorting task. Statements were presented in 20-s intervals. Within an interval, participants were required to read the statement aloud, refer it to themselves and to rate its valence. Valence rating involved (a) sorting the statement to one of two piles respectively labeled *negative* and *positive* and (b) rating on the Likert-type scale. The original type of statement as per Velten, (1968) was not disclosed. As a data check, we required that participants sort a minimum of 10 of 15 (67%) of the statements correctly (i.e., depressing to the negative pile and elating to the positive pile).

Immediately after participants had completed the card sorting task they took a seat on a comfortable sofa in a small minimally-decorated room. They remained on the sofa for the duration of Session 1. The Sport Tester monitor was attached and a 5-min predrink heart rate was recorded while participants were at rest. Participants then were randomised to receive alcohol or placebo. They were required to finish the drink within 20 minutes. To further enhance placebo effectiveness, the experimenter told all participants that they were receiving "1.0 ml/kg alcohol, the equivalent of three to five drinks" and that

"because of the purity of the alcohol, the taste and subjective effects may be different (either stronger or weaker)" than they might expect.

After the 20 minute drinking period, and ten additional minutes for further absorption, a 5-min postdrink resting heart rate was recorded. After the recording participants were taken off the Sport Tester. They filled out the BAES (ascending limb), and then listened to soft classical and jazz music. This was to prevent boredom and to minimize the potential confound of retrograde interference. On the descending limb, when BAC had peaked and dropped back to below 0.045, participants again filled out the BAES.

After drinking, participants were prevented from smoking, sleeping, reading or working, and were told that the experimenters would have minimal contact with them during the drinking and intoxication phase, except to monitor their progress and take BAC readings. For participants in the alcohol condition, BAC was measured every 10 min for 6 readings, then every 30 min thereafter, until it dropped below 0.04 ng/dl; participants were then permitted to leave the laboratory. For participants in the placebo condition, BAC was measured every 10 min for 6 readings, then every 30 min for 2 readings; participants then were permitted to leave the laboratory. No feedback about BAC was given to any participant until the end of the experiment.

Session 2

On arrival for Session 2, participants were given a breathalyzer reading to ensure that it was zero and were asked about alcohol or illicit drugs since Session 1. Memory

testing involved recall and recognition tests.

Participants first completed the recall test. They were asked to write down as many of the statements from Session 1 as possible within 10 minutes. Participants were encouraged to remember as much as they could word-for-word where possible. In the recognition test which followed, 10 target stimuli from each type were randomly selected from the correctly-sorted statements in Session 1. To these, 10 distracter cards were added. Participants were presented with the 40 randomly-shuffled cards, one card at a time, at the participant's own rate. They were asked to indicate if they had seen the card in Session 1. Errors were recorded as combined misses and false positive responses (Bruce & Pihl, in press). In addition, participants gave a subjective valence rating of the statement.

Following the memory tests, the Vocabulary test and the Logical Memory test were administered. Participants then filled out the packet of remaining questionnaires. At the end of the experiment, they were debriefed, and reimbursed.

Results

Data analysis

The data were first inspected and prepared for the analyses (Tabachnick & Fiddel, 1989). No multivariate outliers were found. Next, the frequency distribution for each variable was examined. Univariate distributions were analyzed for extreme outliers 3.3 SD from the mean (alpha = .001). These were rounded off to the next nearest value by

0.1 SD (Tabachnick & Fiddel 1989). Inspection of the resulting distributions using skewness and kurtosis criteria (alpha =.001) revealed satisfactory normality for all but 1 variable: IDS-U was square-root transformed to correct for normality (Tabachnick & Fiddell, 1989).

Participants

Age, years of education, weekly alcohol consumption, Eysenck personality, the AEQ, the IDS and sorting errors were used to match the groups and thereby limit confounds. Nonparametric Mann-Whitney tests were used for the 2 card sorting variables. No significant group differences were found. The groups were compared on the other variables using a multivariate analysis of variance (MANOVA). No significant group differences were found. Means and standard deviations for the variables used to match the groups are shown in Table 1.

----- Insert Table 1 about here -----

BAC

Average BAC levels were 0.0633 ng/dl (*SD* = 0.0002) for the alcohol group, and 0.0007 ng/dl (*SD* = 0.0002) for the placebo group. BAC was analyzed with a group (alcohol, placebo) x Time (10, 20, 30, 40, 50, 60, 90, and 120 min postdrink) as a repeated measures variable. The two-way interaction, F (7, 294) = 9.64, and the main effects for Group, F (1, 42) = 1024, and Time, F (7,294) = 10.94, were all highly

significant, *p*s < .0005. As anticipated, alcohol produced much higher BACs than placebo, which itself produced negligible concentrations.

Heart rate

Pre-drinking baselines were determined by averaging readings for the 5-minute period immediately before the drinking period. Postdrinking baselines were determined by averaging readings for the 5 minute period immediately following the first BAC sample. Changes in heart rate were analyses by t-test: mean change differed significantly between the alcohol (M=9.66 $\pm$ SD= 8.31) and placebo (M=3.61 $\pm$ SD=5.65) groups (t(42df)=2.95, p<.005).

BAES

A group (alcohol, placebo) x limb (ascending, descending) x effect (stimulant, sedative) ANOVA was used. The 3-way interaction was significant, F(1, 42) = 6.26, p <.02, as was the main effect of group, $F(1,42) = 12.96$, $p < .001$. Post-hoc analysis with the Newman-Keuls method revealed that the alcohol group had higher stimulant scores on the ascending limb, and higher sedative scores on the descending limb. No other effects were significant.

Memory Measures

For recognition, errors were totaled for each statement type. Recognition was analyzed using a group (alcohol, placebo) x type (depressing, elating) ANOVA, with

statement type as a repeated measures variable. The two-way interaction and the main effect for group were nonsignificant. The mean number of errors committed was 2.20, *SD* = 1.81.

Recall was scored by a single rater (e.g., Bruce & Pihl, in press; Kalin, 1964) *who did not know the participants' group membership*. As determined by a one-way ANOVA, the total number of responses to the recall task did not differ between the alcohol and placebo groups. Responses that accurately resembled the word content original stimulus statements (e.g., Bruce & Pihl, in press) were then tabulated for the recall task; confabulated responses were omitted. Accurate resemblance was determined when the response contained at least three of the same key content words as one of the original Velten (1968) statements. Such gist criteria are commonly used in research as measures of accuracy for the entire stimulus phrase (e.g., Goetz, Anderson, & Schallert, 1981). The number of recalled statements was tabulated for both types. A second experimenter, who also did not know participants' group membership, scored a random selection (25%) of the protocols. Inter-rater agreement was highly significant, $r = .70$, $p < .00005$.

Recall was analyzed using a group (alcohol, placebo) x type (depressing, elating) ANOVA, with statement type as a repeated measures variable. The two-way interaction was significant, $F(1, 42) = 4.65$, $p = .037$. The interaction is shown in Figure 1: The alcohol group recalled an increased number of elating statements and fewer depressing statements than the placebo group. The main effects of group and statement type were nonsignificant.

----- Insert Figure 1 about here -----

Regression Analyses

To further examine the experimental hypotheses, we used stepwise regression analysis to determine if we could predict memory scores with other variables. We wanted to limit the number of dependent (memory) variables for the regression, due to sample size constraints. Thus, only memory variables showing an alcohol-placebo difference were used: From the recall interaction, we found significant differences in type of recall as a function of group. The group difference in the interaction was thus reflected in the difference-score between depressing and elating recall. Scores were calculated by subtracting depressing recall scores from elating recall scores for each participant. Univariate outliers at the .001 level were rounded to the next nearest value (Tabachnick & Fiddel, 1989), leaving the variable normally distributed.

Again due to sample size constraints, we also wanted to limit the number of independent variables. Four predictor variables were selected. First, we used drinking-induced changes in resting heart rate. Change scores for resting heart rate were calculated by subtracting predrink from postdrink heart rate. Second, we used changes in subjective ratings for the statements. Change scores for subjective ratings of the statements were calculated by subtracting Session 1 ratings from Session 2 ratings. (Participants rated 15 depressing and 15 elating statements in Session 1. Of these, 10 depressing and 10 elating were also rated again in Session 2. Change scores were calculated with these 20 statements).

We combined change scores for depressing and elating statements into one variable (the 2 scores were significantly correlated, $r = -.40$, $p = .007$) in order to simplify our presentation of results. The combined rating change score thus reflected the total of the "desirable" changes in subjective ratings from Session 1 to Session 2, i.e., positive changes in the rating of depressing and elating statements. The third predictor variable was the BAES ALST score. We speculated that subjective stimulant response might be related to memory, much as physiological stimulant response would be. The fourth predictor variable was self-reported alcohol consumption. We selected this variable despite the fact that the groups were equated on this measure because we were interested in determining whether our memory effects were associated with consumption.

Final results of the regression analysis indicated that the memory score was significantly predicted by change scores in heart rate and subjective ratings, adjusted $R^2 = .26$, $F\ (2, 41) = 8.53$, $p = .0008$. There was also a trend ($p < .10$) for alcohol consumption to enter the equation. Thus, a desirable memory outcome (increased elating over depressing recall) was associated with alcohol's putative incentive reward effect (larger positive drink-induced changes heart rate) as well as desirable changes in statement ratings. The more a participant recalled elating over depressing statements, the more he experienced alcohol's stimulant effects on heart rate. Further, the more he recalled elating statements over depressing, the more likely he was to change his ratings of the statements in a positive direction. Analysis for bivariate outliers at the alpha = .001 level was satisfactory.

Discussion

Our principal findings were as follows. First, there was a statistically significant interaction for recall between group and type of statement such that recall of elating material was relatively enhanced by alcohol and recall of depressing material was relatively inhibited by alcohol. This finding seems to reflect a highly desirable outcome, and therefore best fits the incentive explanation. Further to this, the second major finding was that the relative enhancement of elating memories over depressing memories was associated with changes in heart rate on the ascending limb of the blood-alcohol curve. The ascending limb of the blood-alcohol curve has been implicated in alcohol's enhancement of memory (Parker et al., 1981). It now appears that ascending limb heart rate responses may be a biological marker for the desirable effects of alcohol on intentional memory.

Alcohol-induced heart rate change on the ascending limb of the blood-alcohol curve is a purported measure of alcohol's desirable psychomotor stimulant effects (Pihl & Peterson, 1995). This effect of alcohol on heart rate is a strong predictor of alcohol consumption in nonalcoholic and alcoholic individuals (Peterson et al., 1996; Peterson, Pihl, Séguin, Finn, & Stewart, 1993). In addition, the degree of alcohol-induced heart rate change varies with risk for development of alcoholism (Conrod, Peterson, Pihl, & Mankowski, 1997). Unfortunately, the subjective stimulant response did not predict the memory effect, but it should be noted that the alcohol group had an increased memory effect, heart rate response *and* subjective stimulant response compared with placebo. A

larger sample size will enable the relationships among these variables to be more thoroughly explored.

The third main finding was that the desirable memory outcome was accompanied by desirable changes in the subjective ratings of the statements. It may be that drink-induced positive changes in the ratings affected how memorable statements were, or that changes in the memory for statements affected their ratings. In either case, it appears that the incentive effects of alcohol may have desirable influences on both quantity of what is remembered, as well as the subjective importance of the memory.

A fourth finding was a trend for alcohol self-reported alcohol consumption to predict the desirable memory effect. Thus, we find an association between desirable memory and subjective outcomes and psychomotor stimulation, and a trend for an association between desirable memory outcomes and alcohol consumption. We raise the possibility that consumption is related in part to the conditioned incentive effects of alcohol reflected in these relationships. Unfortunately, the stimulant scale of the BAES did not enter the equation; perhaps a larger sample size will be needed to find an effect.

Our previous work (Bruce & Pihl, in press) compliments the present findings. That study showed alcohol's effects on *incidental* memory for emotionally salient material may reflect *nonselective enhancement.* In addition, incidental memory effects of alcohol were not associated with alcohol's effects on heart rate, or with alcohol consumption levels. By contrast, the current results demonstrate that alcohol's effects on *intentional* memory for emotionally salient material instead reflect alcohol's *conditioned incentive* effects. Alcohol's nonselective enhancement vs. conditioned incentive effects on memory has thus

been somewhat elucidated.

The results also differ somewhat from findings of other memory experiments in which neutral materials were used (Hewitt et al., 1996; Lamberty et al., 1990; Mann et al., 1984; Mueller et al., 1983; Parker et al., 1980, 1981). Those experiments found enhancement of emotionally-neutral memory by alcohol in both intentional and incidental learning paradigms. By contrast, we found intentional *enhancement* of elating, but *inhibition* of depressing memory. Alcohol's incentive effects on intentional memory thus imply enhanced elating and neutral memory, and impairment of depressing memory.

As with many of the other studies in this area, the strength of our effects was modest, and significant results were obtained for a portion of the measures. In this regard, clarification of the results for recall vs. recognition might be possible in a larger sample. To this end, the recall results here were scored with explicit, reliable, and objective criteria (e.g., Bruce & Pihl., in press) that can easily be replicated by other researchers. The findings for recognition may relate to paradigmatic conditions (e.g., Parker et al., 1980). For example, the number of errors in the recognition task was quite low and may indicate a floor effect.

The current findings also appear to rule out a role for many individual differences in participants (Kalin, 1964). The desirable memory effects occurred despite the fact that the groups were matched in terms of demographics, personality, drinking behavior and initial responses to the stimuli. The findings were also observed despite the minimizing of retrograde interference. Any mediating involvement of these factors now has little empirical support, and their importance to the effects of alcohol on incidental and

intentional memory must be revised.

The current and our previous (Bruce et al., submitted) studies suggest that alcohol expectancies (as reflected in the AEQ) were unassociated with the retrograde effects of alcohol on memory. Current models of expectancy (e.g., Brown, Goldman, Inn, & Anderson, 1980; Goldman, 1994; Goldman, Brown, Christiansen, & Smith, 1991; Oei & Baldwin, 1994; Rather & Goldman, 1994; Stacy, Leigh, & Weingardt, 1994; Stacy, Widaman, & Marlatt, 1990) have proposed that expectancies reflect learning and memory. Any memory effects seem therefore unrelated to alcohol's acute, direct effects on intentional and incidental cues.

The current findings also have implications for a memory-based theory of alcohol "addiction" advanced by White (1996). It appears that alcohol meets two of White's criteria for the mnemonic-addictive effects of drugs, namely the capacity to affect memory by enhancing memory associations *contiguously* (or incidentally), and the capacity to affect memory *contingently*. According to White's model, a drug's ability to form contingent associations may reflect its conditioned incentive properties. If so, it would appear that there are now two separate lines of memory-based evidence for the so-called "reinforcing" properties of alcohol (see Peterson et al., 1996; Pihl & Peterson, 1995). Extension of the current paradigm to clinical and "at-risk" populations may prove illuminating in the context of a "memory" model of alcohol consumption.

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This research was supported by grants from the Medical Research Council of Canada, and by a personal fellowship from the Fonds pour la Formation de Chercheurs et l'Aide de la Recherche (Quebec).

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Table 1. Demographic, personality, drinking behavior and errors in card sorting.

	Placebo		*Alcohol*	
Variable	*Mean*	*SD*	*Mean*	*SD*
A. Demographics				
Age	21.05	3.20	22.18	3.46
Years of education	13.68	1.65	14.64	2.17
Consumption (drinks per week)	8.31	8.04	7.09	6.57
Vocabulary	52.32	9.43	52.77	12.91
Logical Memory	8.59	2.71	9.05	3.01
B. Personality				
Extraversion	12.00	1.00	15.32	0.66
Neuroticism	12.73	1.04	11.32	1.12
Psychoticism	5.23	0.57	5.00	0.51
C. Drinking				
AEQ	34.02	2.63	31.39	2.49
IDS-P	15.45	1.34	15.45	1.14
IDS-U	13.03	2.17	7.82	2.23
D. Sorting Errors				
Sort-D	0.36	0.19	0.50	0.27
Sort-E	0.55	0.21	0.55	0.26

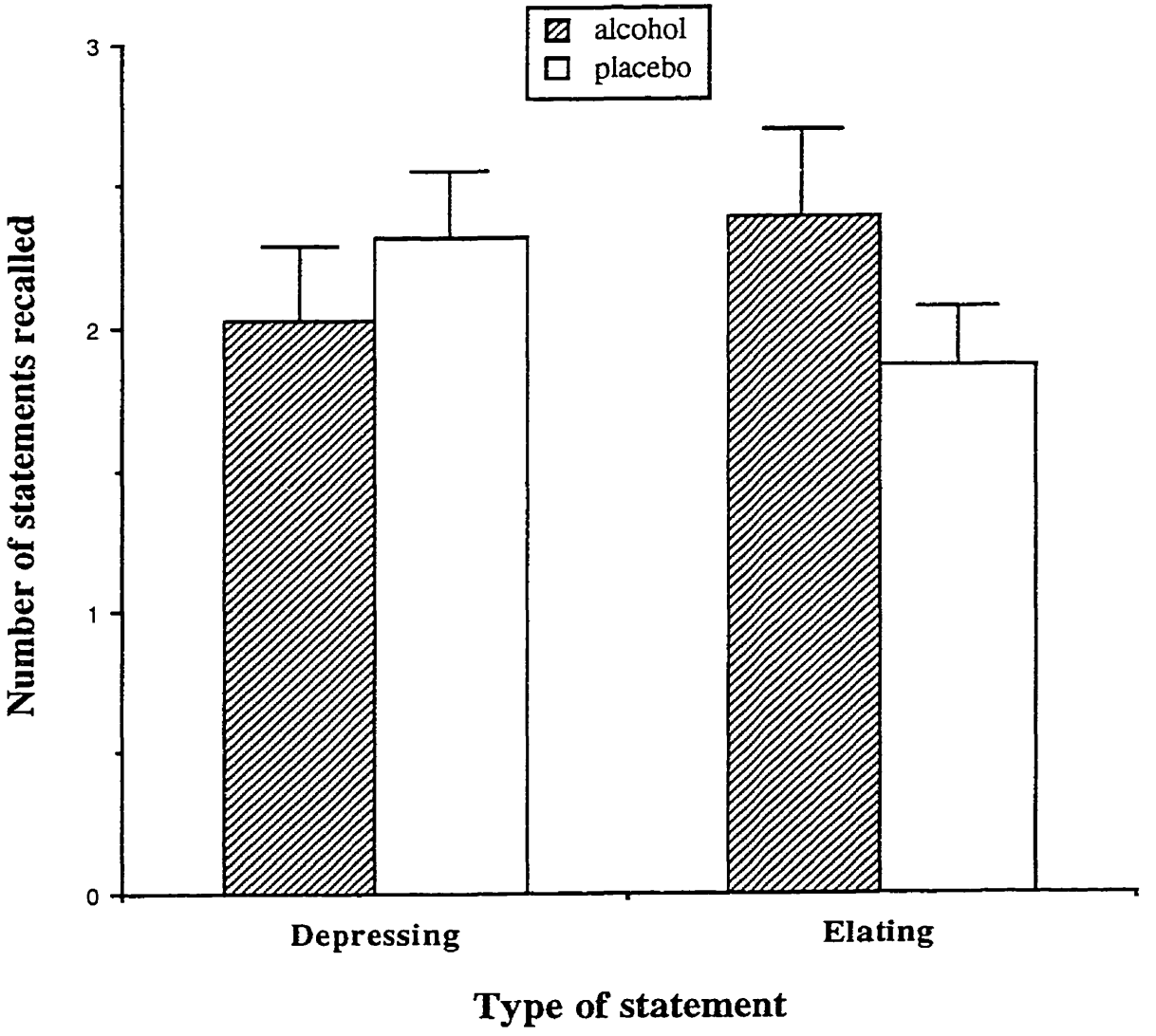
Note. AEQ=Alcohol Expectancy Questionnaire, total score; Consumption=self-reported average alcohol consumption in drinks per week, over the past year; IDS-P=Inventory of

Table 1. continued:

Drinking Situations, pleasant; IDS-U=Inventory of Drinking Situations, unpleasant, prior to square-root transformation; Logical Memory=Wechsler Memory Scale, Logical Memory subtest, Form I, Average of Stories A and B; Sort-D=average number of incorrectly sorted depressing statements; Sort-E=average number of incorrectly sorted elating statements. Vocabulary=Wechsler Adult Intelligence Scale-Revised, Vocabulary subtest, raw score. Group comparisons for variables in Table sections A through C were nonsignificant as per a MANOVA. Group comparisons for Sort-D and Sort-E nonsignificant as per separate Mann-Whitney tests.

Figure Caption:

Figure 1. Recall of statements; a minimum of three of the same content words as the original stimulus (Velten, 1968) statement was required. A significant two-way interaction reflected enhancement by alcohol of elating recall, and inhibition of depressing recall, relative to placebo.


alcohol
placebo
3
2
1
0
Number of statements recalled
Depressing
Elating
Type of statement

Postscript to Study 3

This study showed two primary effects. First, alcohol enhanced the intentional learning of elating material relative to depressing material. Thus, alcohol may have mediated relative remembering of positive memory (conditioned reward) and/or relative forgetting of negative memory (conditioned relief). In any case, we may infer "desirable" memory outcomes, and if so, implicate an association with alcohol's incentive effects. Involvement of reward was speculated (by Esposito et al., 1984: Lamberty et al., 1990; and Parker et al., 1980, 1981) to be involved in posttraining memory effects of alcohol. However, this study is the first to show that alcohol's incentive effects are indeed associated with memory, specifically intentional memory. Note that alcohol's incentive properties are not thought to directly affect memory, i.e., incentive and memory modulation are not thought to be isomorphic processes (cf. Esposito et al., 1984). Instead, incentive effects must be conditioned or associated (likely using separated brain structures) with the statements in order for the memory outcome to occur. Alcohol appeared to affect intentional memory in social drinkers much as posttraining saccharin or sucrose consumption in rats, as described at the outset. Thus, combined with Study 1, we show that alcohol's rewarding effects are implicated in alcohol's effects on intentional, but not incidental, memory. Further, although alcohol's incentive effects can be conditioned in memory (given the appropriate contingency), they are in fact separate events from alcohol's memory-modulating properties.

Second, the degree to which the desired memory outcome occurred was

significantly predicted by two important individual participant variables, and a trend emerged for an association with a third. This appeared to implicate an association between the desirable memory effects, and alcohol's putatively psychomotor stimulant effects on heart rate. If so, this supports the Peterson et al., (1996) notion that alcohol-induced heart rate change does reflect/predict other of alcohol's desirable effects.

Whether the conditioned motivating effects described in Study 3 reflect activity in White's (1996) amygdala system (responsible for classical conditioning of drug effects to stimuli) remains to be seen. The incentive reward (VTA, nucleus accumbens) system may also interact with the hippocampal learning system (Olds, 1969), as may the incentive relief system (reviewed in Pihl & Peterson, 1995). In this regard, the hippocampal system is responsible for conditioning of complex associations between drug and internal affective states (White, 1996). These possibilities certainly deserve further investigation since PET studies (eg, Sano et al., 1993) have shown metabolic changes in the temporal lobe (where both amygdala and hippocampus are located) to be associated with alcohol's acute subjective effects.

Of course, White's theories are not the only ones supported by the findings of study 3. The Pihl and Peterson (1995) theory of conditioned incentive was also supported in that alcohol's effect on heart rate did indeed predict alcohol's desirable effects on memory. Interestingly, this was true for the intentional paradigm, but, perhaps not surprisingly, not true in the incidental paradigm used in Study 1. The result that desirable changes in participants' subjective ratings of the stimuli were implicated as well here makes the heart rate findings all the more important.

Other theories were supported as well. Semantic network model of emotions and memory (Bower, Gilligan, & Monteiro, 1981) hypothesize that information congruent with the ongoing mood state is more likely to be remembered. If the BAES findings reflect that alcohol produced desirable changes in affect (e.g. Martin et al., 1993) then it may be more likely that our elating statements would be recalled than the depressing ones.

There may be some implication for alcohol expectancy theories as well. These theories postulate that memory is important in determining drinking decisions. However, the relationship between expectancies and the effects of alcohol on memory are not well understood. For example, the recall effects showed an alcohol-placebo group difference. This occurred despite the fact that the groups were match on AEQ scores. Thus, the effects of alcohol on memory were observed despite similar expectancies; this may mean alcohol expectancies do not influence what alcohol does to emotional memory (or vice versa). For example some participants may have had beliefs about what alcohol would do to memory (there was a contingency between alcohol and memory). Some participants may have believed that alcohol would have impaired or enhanced some or all types of memories. If so, this tendency may have been reflected in their recall. Thus, it is important to not that the group recall difference was observed in groups matched for AEQ scores.

As with other studies examining the effects of alcohol on memory, significant results were found for some of the dependent variables, but not others. In particular, significant results were found for recall. Objectivity of the recall results was bolstered in two ways. First, clear and simple criteria were used in scoring. Second, inter-rater

reliability was established. Results for recognition may represent a "floor" effect where very few errors were made by either the alcohol or the placebo group. Perhaps if a larger sample of statements and participants were used, the results may have been more clear. Nonetheless, it may instead reflect a subtle drug effect of alcohol. As with study 1, other limitations of this study include a restriction to verbal stimuli (i.e., omission of visual stimuli), the unknown generalizability of the results given the demographically homogeneous sample, and the modest sample size.

General discussion

Does alcohol enhance memory for recent experiences? Does it do so independent from, or as a result of, its desired subjective-physiological (incentive) effects? Do the memory effects depend on other individual differences of interest to alcohol usage? Is the effect on memory one of facilitation, or is it instead a secondary effect due to alcohol's well known capacity to reduce new learning? What is the relationship between alcohol's effects on emotional experience, and the later memory and impact of this effect? What does "reinforcement" mean as applied to alcohol and alcohol consumption?

These are some of the questions this thesis has tried to answer. It appears the distinction between incidental and intentional learning is a crucial one in explaining how alcohol affects memory, and why there are some discrepancies in the posttraining human literature that has thus far been restricted to paradigms that examine emotionally neutral memory. The incidental-intentional distinction has also proven useful in testing, refining

and supporting theories of alcohol (Pihl and Peterson, 1995) and drug self-administration (White, 1996).

The studies in this thesis represented an attempt at delineating the effects of alcohol on emotionally charged memory, and the underlying cognitive-motivational mechanisms. Four mechanisms (interference reduction, individual differences, nonselective enhancement, and enhancement specifically associated with alcohol's incentive effects) were studied under two different experimental learning paradigms. The main finding was that alcohol appeared to enhance incidental memory by non-contingent, nonspecific pharmacological enhancement (much as the sucrose injections presented at the outset) and to affect intentional memory by its contingent, conditioned incentive or motivating psychomotor effects (much as the consumption of saccharin and sucrose presented at the outset). Individual differences in response to alcohol were important in the intentional paradigm, and individual differences in basic cognitive abilities were important in the incidental paradigm. Finally, alcohol's effects on memory were observed despite minimal retrograde interference, suggesting alcohol affected memory even when there was little interference to be reduced.

Memory for emotionally charged stimuli is affected by alcohol in a different manner depending on whether the initial learning is incidental or intended. It appears that two distinct mechanisms (nonselective enhancement of incidental learning, independent of most individual difference variables) and conditioned motivation (of intentional learning, dependent on individual responses to alcohol) have been described.

Further, alcohol's effects on memory are separable from its incentive effects.

Depending on the circumstances, alcohol's effects on memory reflect (a) a direct action of alcohol on memory processes, or (b) an influencing of memory by alcohol whereby association(s) are made between the memory processes and alcohol's incentive effects. The critical differentiation (at least in humans) appears to depend on whether there is a contingency between alcohol and memory. If there is no contingency, alcohol affects memory by nonselective enhancement. If there is a contingency, alcohol affects memory by the conditioning or associating of its motivating properties to concurrent or recent cognitive activity.

Thus, our extrapolation of White and Milner's (1992) ideas concerning "reinforcers" to the acute effects of alcohol on memory in social drinkers has been pleasantly successful. Speculating, we might hypothesize that alcohol enhances incidental learning by promoting or enhancing ongoing memory activity in the caudate nucleus, hippocampus and related structures. Further, alcohol may activate the incentive reward (VTA/nucleus accumbens) and relief (limbic) systems, and these effects may condition intentional learning in the amygdala and/or hippocampus. Formal testing of these possibilities awaits.

Individuals may thus be assessed in terms of "risk" for drinking (their drinking behaviours, motives, expectancies, etc.) in terms of both alcohol's memory-modulating and incentive effects. The term "alcohol reinforcement" may be better described in terms of these two separate processes. It is needlessly confusing to say that individuals drink because they find alcohol to be "reinforcing." Instead, it may be better to describe the *relationship* between alcohol's incentive effects (such as reducing pain, hurt, anger,

depression and anxiety; as well as increasing satisfaction, contentment, calm, excitement, curiosity, pleasure and hope) *and* alcohol's effects on learning or conditioning (such as modulating memory for recent neutral and emotional experiences). Thus, for example, does the alcohol-induced heart rate change (or stress-response dampening) observed in a given individual predict/produce alcohol's memory outcomes?

If an individual has a strong susceptibility to alcohol's incentive effects (strong heart rate change or stress-response dampening), but has poor memory for them or does not associate them with other cues, the motivation to drink again in the future may not be altered by his acute drinking experience. By contrast, for individuals who, despite a low susceptibility to alcohol's incentive effects, may have "memories" for incentives, or may have associated them with many available internal and external cues, could have quite strong motivations to drink in the future. There may be two populations of drinkers here: those who experience both incentive and memory effects, and those who have a kind of "false memory" or belief that alcohol has incentive for them, and proceed to drink nonetheless.

The studies presented herein have accomplished some important goals. The mechanisms behind the effects of alcohol on memory consolidation for incidental and intentional memory have been clarified. The parameters defining the influence of individual participant differences has been somewhat delineated. Finally, the applicability of a memory-based animal model of reinforcers (White & Milner, 1992) to the effects of alcohol on incidental and intentional memory in male social drinkers was established, and the animal model (White, 1996) for drug addiction has been clarified for alcohol.

Further, the link between these two models and an exciting motivational theory of alcoholism (Pihl & Peterson, 1995) was established. Extension of this established link to studies of women and of "at-risk" and clinical populations may prove useful in addressing the incentive and mnemonic aspects of alcohol use and misuse. What are some of the next steps this research could take? Perhaps a next study might investigate the effect of alcohol on intentional memory in a sample of females and males. The sample could draw on individuals known (based on previous studies) or demonstrated to be sensitive to alcohol-induced heart rate change; matched controls with little alcohol-induced heart rate response would also be included. The hypothesis might be that these individuals would show preferential associated increases in memory for positive statements. In this regard, it would be highly interesting to examine individuals at high genetic risk for alcoholism since they tend to experience large heart rate responses to alcohol. Further, the study could also include individuals who show increased susceptibility to alcohol's stress dampening effects (and matched controls). That is, persons sensitive to alcohol-induced relief of negative mood states (anxiety and depression sensitive) could be implicated here. The hypothesis here might be that these individuals would show an associated impairment in memory for negative material following alcohol consumption. This study would be one possible next step in determining "why" some individuals are at heightened risk for alcohol consumption and potential misuse. Other studies could examine the effects of alcohol on memory in clinical populations, particularly those with alcohol dependence, or mood- or anxiety disorders.

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IMAGE EVALUATION
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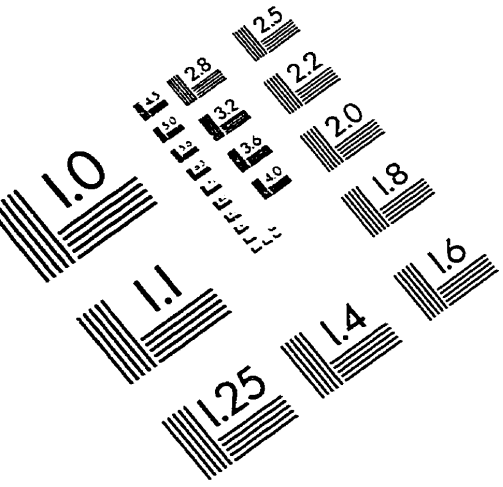

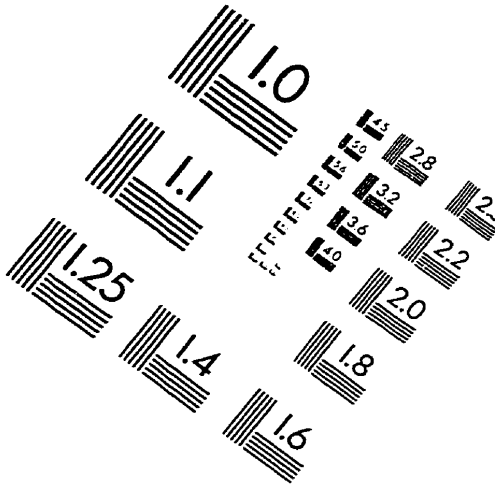

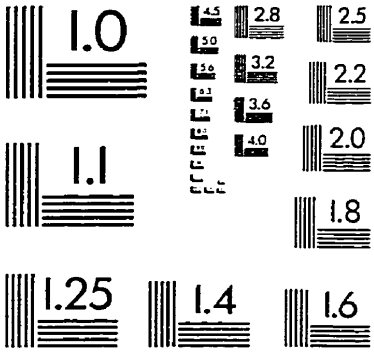

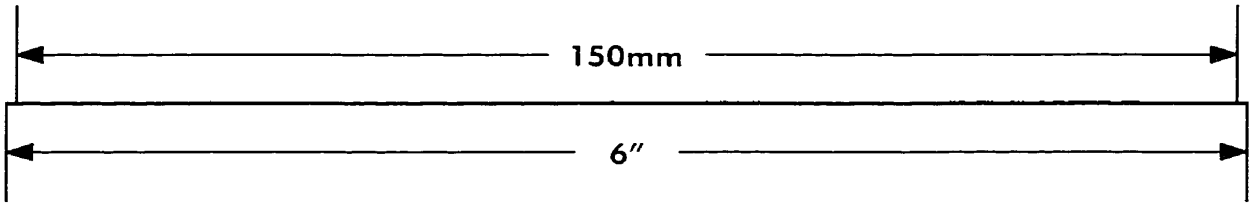

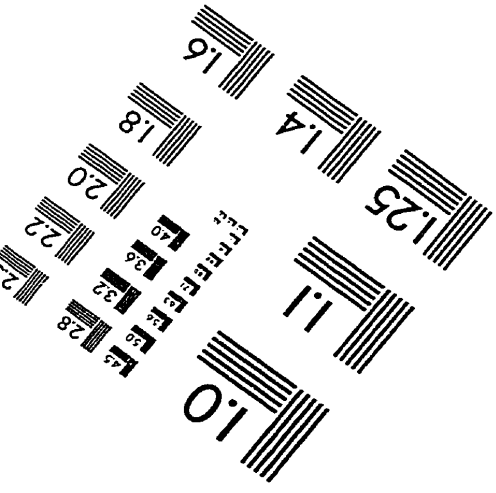

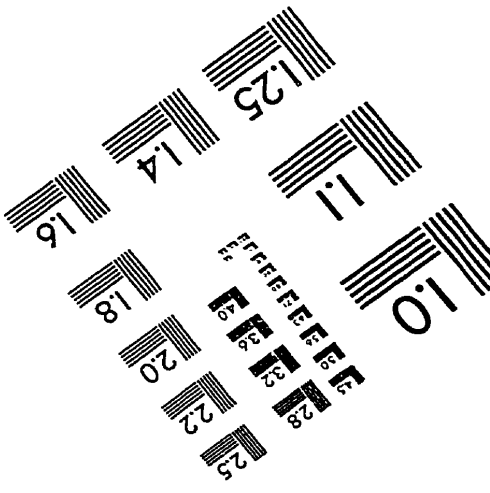

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