Psychopharmacology (2006) 188:498–508
DOI 10.1007/s00213-006-0480-6

ORIGINAL INVESTIGATION

The effect of alcohol and repetition at encoding on implicit and explicit false memories

S. N. Garfinkel · Z. Dienes · T. Duka

Received: 22 February 2006 / Accepted: 14 June 2006 / Published online: 11 August 2006

Abstract
Rationale Alcohol impairs explicit memory, whilst leaving implicit memory relatively intact. Less is known about its effects on false memories.
Aim The present study examines the effects of alcohol on explicit and implicit false memories using study list repetition as a tool for modulating learning at encoding.
Methods Thirty-two participants were given either an alcohol (0.6 g/kg) or placebo beverage before undergoing an encoding phase consisting of 10 lists of nine associated words (veridical items). Each list was associated to a word, which was not presented at encoding (semantically associated non-studied lure; critical item), serving as the measure for false memory. Half of the lists were presented once, and half were repeated three times. The next day, participants underwent an implicit (stem completion and post hoc awareness measurements), and an explicit (free recall) task.
Results Alcohol decreased veridical and false explicit memory for singularly presented lists compared to placebo; no group difference existed for repeated lists. Implicit veridical memory was not affected by alcohol. Awareness memory measures demonstrated in placebo participants an increased ability with repetition in rejecting false memories. The reverse was found in intoxicated participants who with repetition accepted more false memories.
Conclusion Alcohol appears to decrease semantic activation leading to a decline in false memories. Increased learning with repetition, which increases the rejection of false memories under placebo, is reversed under alcohol leading to a decrease in rejection of false memories. The latter effect of alcohol may be due to its ability to impair monitoring processes established at encoding.

Keywords Learning · Free recall · Stem completion · DRM paradigm · Awareness

Introduction

False memories constitute a process involving either 'remembering events that never happened, or remembering them quite differently from the way they happened' (Roediger and McDermott 1995). Deese's (1959) memory paradigm revived and restructured by Roediger and McDermott (1995) has provided the basis for much recent false memory research (DRM paradigm). Its popularity can in part be attributed to the robustness with which it generates false memories, in both recall (e.g. Deese 1959; Roediger and McDermott 1995) and recognition (e.g. Roediger and McDermott 1995). The DRM paradigm is composed of lists of associated ('veridical') words (e.g. hill, valley, climb, summit, etc.), with each list related to a 'semantically associated non-studied lure' (e.g. mountain) termed the 'critical' item. The probability that this non-presented critical item will subsequently be erroneously recalled or recognised as having been previously presented serves as the measure for false memory.

To date, only two published studies have investigated the effect of alcohol on false memories using the DRM paradigm (Milani and Curran 2000; Mintzer and Griffiths 2001), with contrasting findings. Milani and Curran (2000) did not detect an effect of alcohol on false recall rates, though a tendency for alcohol to increase false recognition

S. N. Garfinkel · Z. Dienes · T. Duka (✉)
Department of Psychology School of Life Sciences,
University of Sussex, Falmer,
Brighton BN1 9QG, UK
e-mail: t.duka@sussex.ac.uk

was obtained. In contrast, Mintzer and Griffiths (2001) did not find an increase in false recognition rates under alcohol relative to a placebo. These equivocal findings warrant further investigation into the effect of alcohol on false memories. Research into the effect of alcohol on false memories is not only interesting in its own right, but can also provide an insight into the cognitive processes underlying memory judgements. This follows a tradition where researchers have used pharmacological agents to provide insight into underlying mechanisms (Duka et al. 1996). To date, the effect of alcohol on false memories has not been accounted for using contemporary false memory theories.

The Activation Monitoring Framework (AMF) was proposed by Roediger and colleagues (Gallo and Roediger 2002; Roediger et al. 2001). It postulates dual opponent processes, which determine false memory levels: an activation component and a monitoring component. False memories are thought to be elicited via their activation, and false remembering is thought to subsequently occur when monitoring fails and individuals misattribute the source of this activation to prior presentation.

As the AMF is formed of opponent processes, it can accommodate both an increase and decrease in false memory levels dependent upon the extent to which factors differentially affect activation and monitoring potential. Fundamentally, one can predict that any process which selectively decreases activation should lead to a decrease in false memory, whilst any manipulation that decreases monitoring should cause an increase in false memory. A primary prediction is that alcohol will decrease false memories because it impairs semantic processing (Craik 1977; Birnbaum et al. 1980; Weissenborn and Duka 2000), decreasing activation of false memory items.

The amnesic effects of alcohol consumed before encoding are well documented, with studies consistently demonstrating an impairment of free recall (e.g. Birnbaum et al. 1978) and cued recall (Duka et al. 2001) relative to a placebo control. In 1997, Craik (1977) published an article which accounted for the deleterious effect of alcohol on memory performance using the levels of processing (LOP) framework. According to his argument, alcohol reduces the depth at which items are processed, with depth being defined as 'a continuum of processing running from shallow sensory analyses requiring little attention to deeper semantic processes through which the stimulus is identified, interpreted and enriched by associations with stored knowledge'. Alcohol has been shown to reduce attentional and processing resources (Schweizer et al. 2005), which are deemed necessary for deep encoding (Craik and Lockhart 1972). Because studies which have reduced depth of processing found a decrease in false memory levels (Rhodes and Anastasi 2000; Thapar and McDermott 2001; Toglia et al. 1999), superficial encoding when intoxicated would reduce false memories.

Parker et al. (1974) hypothesised that "the more demanding the task, the greater the impairment from alcohol". Johnson (1977) qualified this claim by defining 'demanding' as being the extent to which a task necessitates the 'finding or generating [of] associations, interrelationships or structures'. Parker et al. (1976) found that a high dose of alcohol (1.0 ml/kg [0.8 g/kg]), impaired paired associative learning. Alcohol was also found to reduce selectively memory for high associations (Weissenborn and Duka 2000) and Birnbaum et al. (1980) have shown an impairment in the semantic processing of incoming stimuli. The impairment of such processes has implications for the forming of conscious associations between veridical items, a process, which is thought to be a potential route to increasing false memories (Libby and Neisser 2001; Roediger et al. 2001). Repetition as a means of increasing learning has been applied with the DRM paradigm as an experimental manipulation to investigate the mechanisms underlying false memories (Benjamin 2001). Repetition at encoding has been shown to increase false memories (Benjamin 2001), decrease false memories (Tussing and Greene 1997), have no effect on false memories (Tussing and Greene 1999) and affect false memories in an inverted U-shaped function (Seamon et al. 2002). As repetition may both increase false memories via increased activation of semantically associated non-studied items, and decrease false memories through increased monitoring, observing the effect of repetition on false memory levels can provide insight into whether activation or monitoring processes are prevailing in memory judgements. We, thus, used in the present study repetition as a tool to gain insight into the effect of alcohol on activation and monitoring processes relevant for false memory endorsement.

Implicit and explicit measures of memory will be taken—this is because alcohol has been shown to differentially affect these memory measures, by impairing explicit memory, such as free recall, but leaving implicit memory relatively intact (Duka et al. 2001). Priming measures will be taken for both the veridical (studied items) and critical (semantically associated non-studied) items based on previous methodologies used in the alcohol memory literature (e.g. Duka et al. 2001; Weissenborn and Duka 2000). Testing will take place next day without the influence of alcohol so that the effects of alcohol on encoding will be tested independently from its effects on retrieval. Previous studies testing the effects of alcohol on false memories did not fully separate the encoding and retrieval phases of the experiment in terms of alcohol intoxication (Curran and

Hildebrandt 1999; Mintzer and Griffiths 2001). Testing after a 24-h interval will, in addition, provide information about long-term priming for false memories because no study using implicit measures has previously attempted a 24-h retention interval. Finding ways in which veridical memory operates in the same way and differs from false memory provides insight into mechanisms underlying these two types of memory.

Measurements of drinking habits and history were evaluated to make sure that groups did not differ on aspects, which might influence alcohol effects.

Materials and methods

Participants

Thirty-two volunteers (16 men and 16 women) were recruited from the undergraduate and postgraduate population at the University of Sussex. They received either cash or course credits as payment. All participants were native English speakers, not dyslexic and aged 18–34 years. Participants were randomly assigned to either an alcohol or a placebo condition, with the constraint that groups were matched for gender (eight men and eight women). The groups had very similar mean age (mean age 21.30 years in the alcohol group and 20.96 years in the placebo group).

The ethics committee of the University of Sussex approved the experiment. After giving informed consent, participants were screened on the basis of their medical history. Exclusion criteria included: a history of severe mental illness, a history of drug or alcohol misuse, an altered metabolism of alcohol (as determined by impaired liver function or gastroenteritis), anyone displaying current symptoms of mental illness or neurological disease. Participants were asked to abstain from illicit recreational drugs for a minimum of 7 days, from sleeping tablets or hay fever medication for 48 h, and from drinking tea or coffee immediately before the commencement of the experiment and were required to have a low fat breakfast the morning of the experiment. In addition, participants were told to abstain from drinking for 24 h before the beginning of the experimental session, and were breathalysed on entering the lab as a means to determine whether they had complied with this requirement.

Design

The experiment was double blind and took place over two consecutive days. The drink was consumed on day 1, and was followed by the learning phase. Participants returned the next day to undergo two memory tests: an implicit test and free recall.

Memory task

Encoding

Eleven 9-word lists were taken from Stadler et al. (1999) when lists conformed to the specifications made by McEvoy et al. (1999) deemed necessary for stem completion tests. To investigate false memories under implicit stem completion instructions, critical items had to adhere to the specifications outlined by Graf and Schacter (1985), which had been followed by other researchers using the DRM paradigm (McKone and Murphy 2000). These were: critical items were a minimum of five letters long, all 3-letter stems had a minimum of eight different word completions, stems were required to not form words in themselves, all stems used were distinct and not the same as the opening three letters of any veridical words and that baseline stem completion rates were not higher than a 50% probability of completion, as a means to avoid ceiling effects on priming scores (McKone and Murphy 2000).

Only 11 lists with the norms of Stadler et al. (1999) fulfilled the above criteria. Nine new lists were created with the aid of the Edinburgh Associative Thesaurus (EAT: http://www.eat.rl.ac.uk/). The EAT is a set of word association norms, displaying the counts of word association elicited in association to target words, as collected empirically from participants following the methodology employed by Kiss et al. (1973). Following the procedure used by Roediger and McDermott (1995), lists were formed of the top veridical associates of nine new critical words. In a pilot study, the new lists were evaluated and analyses determined that the newly created DRM lists did not significantly differ from those used by Stadler et al. (1999) in terms of backwards associative strength from the veridical items to the critical items [$t(9)=-1.51$, $p=0.166$], baseline stem completion rates for critical items [$t(9)=0.39$, $p=0.708$] and probability that lists would elicit a false memory [$t(15)=1.19$, $p=0.252$].

The 20 9-word lists were separated into two sets consisting of 10 lists each (set A and set B). Each participant viewed either set A or set B in the encoding phase. Each set was also divided into two sections, section 1 and section 2 (resulting in five lists in each section). The difference between the two sections was the frequency of presentation (once or three times). Lists in each section were matched for word frequency (Kucera and Francis 1967) and stem completion rate. List repetition was counterbalanced across sections. The presentation order of lists was fully randomised, with the constraint that repetitions were consecutive.

Testing

The implicit test consisted of 80 stems: two critical items and two veridical items from each list. Twenty studied veridical and 20 non-studied critical items corresponded to lists viewed in encoding. Ten of these veridical items came from lists presented once, whilst the remaining 10 were from repeated lists. Similarly, 10 critical items were from repeated lists, whilst 10 were from singularly presented lists. The remaining 40 stems were from the set not viewed in the encoding session, and served as baseline measures. These baseline stems were comprised of 20 veridical items not viewed in the encoding session, and their associated 20 critical items.

After the implicit test, a free recall test was introduced. Participants were given instructions to write down all the words they remembered from the previous day's encoding phase.

Alcohol administration

Alcohol was administered at the dose of 0.6 g/kg, with 90% *v*/*w* alcohol diluted with tonic water to make up a 300-ml beverage. The drink was divided into 10×30 ml portions and each portion was mixed with four drops of Angostura® bitters. The drink was consumed at a rate of one portion every 3 min over a 30-min period in the presence of the experimenter. The placebo beverage consisted of the respective volume of Angostura® bitters and tonic water only.

Alcohol use questionnaire

Participants' weekly alcohol consumption was assessed using the alcohol use questionnaire (AUQ) (Mehrabian and Russell 1978). The AUQ consists of 12 questions designed to evaluate consumption of beer/cider, wine and spirits in terms of frequency and quantity. Due to the recent changes in drinking trends, an updated version was used which incorporated questions about the consumption of Alco pops (Knowles and Duka 2004). The dependent variables produced by the AUQ are: 8 g UK units of alcohol units drunk each week and an overall AUQ score which represents speed and frequency of drinking and intoxication. In addition, a separate binge drinking score based just on speed of drinking and frequency of intoxication developed by Townshend and Duka (2002) was also determined. The questionnaire was administered to ensure that participants met the specifications for participating—that they were social drinkers who consumed on average between 5 to 50 U a week. In addition, group analyses could ensure that the alcohol and placebo participants were matched in terms of units consumed per week, AUQ score and binge drinking score.

Visual analogue scales

To assess mood, both in terms of baseline measures and the acute effects of alcohol on mood, participants were asked to complete a set of 100 mm visual analogue scales (VASs) (Duka et al. 1998). These provided measures of how participants were feeling at that particular moment. Dependent variables were 0–100 scores for the following adjectives: contented, lightheaded and relaxed.

Assessment of baseline memory

Baseline memory was assessed to ensure that no differences existed between alcohol and placebo groups. Fifteen-member word lists—lists A and B from the Rey Auditory–Verbal Learning Test Word Lists which were validated for frequency and complexity by Lezak (1983) were administered before drink consumption. The dependent variable was the mean number of words recalled from the two lists (5 min after presentation).

Blood alcohol concentrations

A standard breathalyser with a detection limit equivalent to 0.01 g/l of alcohol in the bloodstream was used to measure blood alcohol concentrations (BAC) levels (Alcolmeter S-D3M, Loborservice GmbH, Bonn, Germany). The dependent variable was BAC measured in gram per liter.

Procedure

Participants were tested individually. Upon entering the lab, participants signed a consent form and read a brief description about the experiment, which stated that the effect of alcohol on learning was to be tested. They were not informed that the experiment was concerned with false memory. Participants underwent a medical interview and filled out a medical questionnaire to ensure they were medically fit to take part, and the AUQ to ensure they were moderate social drinkers.

All participants were breathalysed to ensure their baseline breath alcohol concentrations (BACs) were 0.

Participants height and weight were taken, they consumed a standardised lunch (a small roll and a glass of water) and completed the AUQ and a verbal memory task (Lezak 1983), as a means to obtain baseline memory scores. They were then taken to a medical room to consume their drinks over a 30-min period. Their BAC

level was measured 10 min after the final drink was provided (40 min after the initiation of drinking) when participants completed also a series of visual analogue scales, which took approximately 5 min. Participants were then taken to the experimental rooms where they underwent the encoding phase. The encoding phase lasted for 15 min.

Upon completion of the encoding phase, participants were sent to sit in the waiting room and were subsequently breathalysed at 30-min intervals. Once BACs had fallen to below 0.4 g/l, participants gave consent that they would not drink, ride a bike or operate any kind of machinery for 4 h and were released from the laboratory.

On day 2, participants again completed the VAS and then completed the implicit test following the instructions. Instructions stated that the stems were to be completed with the first word that came to mind. Testing of free recall followed the stem completion. Participants were asked to generate as many words as possible from the ones, which they viewed the day before. No time limit was enforced. Participants then returned to the words they had generated under implicit test instructions; they were instructed to circle all the words they were 'aware' of having viewed in the encoding phase. The words that participants had completed to form words viewed at the encoding phase, which were not circled were classified as 'unaware'.

Statistical analyses

Following a mode of analysis employed by previous researchers using pharmacological manipulations in conjunction with the DRM paradigm (Huron et al. 2001), all analyses were performed separately in veridical and critical items. This allowed an assessment of how repetition and alcohol selectively modulated false and veridical memory levels. All analyses were performed on percentages of words recalled of the words presented at encoding (free recall) or percentages of words completed of the words to be completed (implicit task). The main analysis is divided into three parts. Firstly, the free recall data (number of words recalled) is addressed. The data generated under implicit instructions (number words completed) are analysed with regard to priming (number of words completed studied vs non-studied), followed by an analysis which is concerned with items completed and labelled 'unaware' (rejected as not presented previously at encoding) vs 'aware' (erroneously accepted as being presented previously). Repetition is also included as a within subject factor in each analysis. Details of individual ANOVAs preformed precede each analysis.

Results

Group characteristics

Analyses of the alcohol and placebo groups revealed that participants were matched for units consumed per week [$t(30)=1.60$, $p=0.120$], AUQ score [$t(30)=0.26$, $p=0.799$], binge drinking score [$t(30)=-0.46$, $p=0.625$] and baseline memory measures ($t(30)=0.29$, $p=0.772$) (see Table 1).

VAS self-ratings

The alcohol and placebo groups did not differ on self-ratings of relaxedness on either day 1 [$t(30)=-0.88$, $p=0.383$] or day 2 [$t(30)=-0.83$, $p=0.412$]. Nor did they differ on ratings of contentedness on either day 1 [$t(30)=-1.12$, $p=0.261$] or day 2 [$t(30)=-0.068$, $p=0.946$]. A significant effect of alcohol was displayed in subjective ratings of lightheadedness. On day 1, post consumption of the drink, the alcohol group rated themselves as significantly more lightheaded than the placebo group [$t(30)=5.39$, $p<0.001$]. On day 2, when no alcohol was administered, no significant difference existed in lightheadedness ratings between the two groups [$t(30)=0.00$, $p=1.000$] (see Table 2).

Free recall

To determine whether it was necessary to correct memory scores for the recall of intrusions (recall of non-presented items, which were not critical items), between subjects t tests were administered to determine the extent to which intrusion rates differed between the alcohol and placebo groups. The amount of non-critical intrusions recalled between the two groups approached significance [$t(30)=1.86$, $p=0.072$], the mean value of non-critical intrusions recalled was higher in the placebo group (4.81) than the alcohol group (2.81). As placebo participants recalled more veridical and critical items than the alcohol group (see analyses below), two new variables were formed which looked at the ratio of non-critical intrusions to total veridical memory and total critical memory recalled; these new variables resulted in non-

Table 1 Units per week consumed, AUQ score, binge score and baseline memory measures [mean (SEM)] for the alcohol and placebo groups

	Placebo	Alcohol
Units per week[a]	37.11 (5.40)	26.96 (3.32)
AUQ score	54.09 (5.85)	51.57 (7.92)
Binge score	21.46 (2.91)	24.61 (5.65)
Baseline memory score (number of words)	17.56 (1.08)	17.19 (1.09)

[a]1 U=8 g of alcohol

Table 2 VAS self-ratings [mean (SEM)] for alcohol and placebo groups on day 1 measured after participants completed drinking the beverage (shortly before list presentation), and on day 2 shortly before testing

	Day 1		Day 2	
	Placebo	Alcohol	Placebo	Alcohol
Relaxed	5.51 (.46)	6.1 (.47)	5.32 (.41)	4.77 (.52)
Content	5.65 (.39)	6.27 (.37)	5.65 (.39)	5.69 (.51)
Lightheaded	1.83 (.56)	5.96 (.52)*	0.53 (.09)	0.53 (.09)

*$p<0.05$ vs placebo on the same day of testing

significant differences in intrusions as a function of total veridical memory recalled [$t(30)=.04$, $p=0.91$] and total critical memory recalled [$t(30)=0.95$, $p=0.349$]. Consequently, no corrections were administered before analyses and, thus, results were analysed using raw data only.

Veridical items

To explore the effect of alcohol on veridical items, a mixed 2(drink: alcohol vs placebo)×2(repetition: lists presented once vs lists presented three times) ANOVA was performed on the percentage of veridical words recalled, with drink serving as a between subjects variable, and repetition as a within subjects variable.

A main effect of repetition was found [F (1, 30)=67.14, $p<0.001$; see Fig. 1] demonstrating that repetition of lists resulted in a greater probability of later recall. The repetition × drink interaction was not found to be significant [$F(1, 30)=1.19$, $p=0.284$], nor was a main effect of drink obtained [$F(1, 30)=1.77$, $p=0.193$; Fig. 1].

Due to homogeneity of variance problem, separate between subjects t tests between alcohol and placebo groups were run in the single and repeated conditions. It was found that participants in the alcohol group recalled significantly less items than placebo participants for singularly presented lists [$t(30)=2.95$, $p=0.006$]. No difference existed between the two groups for repeated lists [$t(30)=0.42$, $p=0.679$].

Critical items

To explore the effect of alcohol and repetition on critical items a mixed 2(drink: alcohol vs placebo)×2(repetition: lists presented once vs lists presented three times) ANOVA was performed on the percentage of critical words recalled, with repetition as a within subjects factor, and drink as a between subjects factor.

A main effect of repetition [$F(1, 30)=3.93$, $p=0.057$] bordered on significance, with repetition increasing critical recall. Whilst there was no main effect of the drink [$F(1, 30)=1.38$, $p=0.249$], a repetition × drink interaction [$F(1, 30)=3.93$, $p=0.057$], approached significance. Further exploration revealed a non-significant effect of repetition on the percentage of critical items recalled in placebo subjects [$t(1, 15)=0.00$, $p=1.00$]. In contrast, repetition was found to increase significantly the percentage of critical items recalled in participants who had consumed alcohol [$t(15)=2.79$, $p=0.014$; Fig. 2].

Implicit test of memory

Priming

A mixed 2(status: studied vs non-studied)×2(drink: placebo vs alcohol) ANOVA was performed on the percentage of words completed for the veridical and critical items separately. The drink served as the between subjects

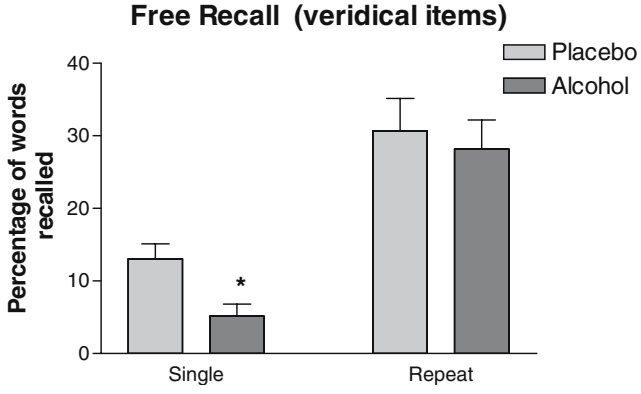

Fig. 1 Percentage of veridical items (mean±SEM) recalled from lists presented singularly (*single*) or three times (*repeat*) after drinking alcohol or placebo beverage. Free recall was tested 24 h after list presentation. *$p<0.05$ compared to placebo in the single presentation condition

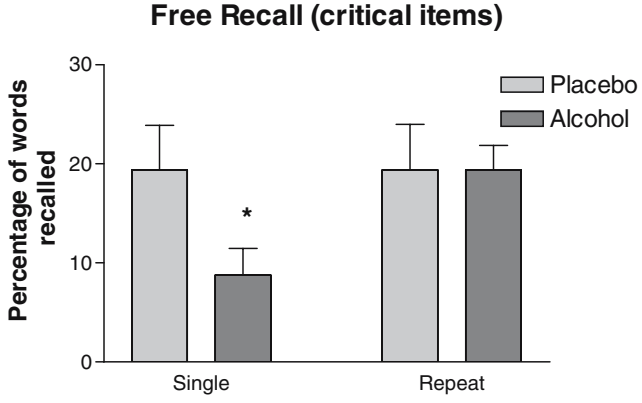

Fig. 2 Percentage of critical items (mean±SEM) recalled associated with lists presented singularly (*single*) or three times (*repeat*) after drinking alcohol or placebo beverage. Free recall was tested 24 h after list presentation. *$p<0.05$ compared to repeated presentation in the alcohol condition

variable, whist word type and status were within subjects factors.

There was an effect of status for veridical [$F(1, 30)=10.17$, $p=0.003$], but not for critical items [$F(1, 30)=1.83$, $p=0.186$] reflecting the finding that previously studying veridical words increased the likelihood of completing a stem to form that word. There was no status × drink interaction found either in veridical [$F(1, 30)=0.91$, $p=0.346$], or in critical [$F(1, 30)=0.36$, $p=0.552$], items (see Fig. 3a,b). A further analysis was also performed on the veridical and critical items from the studied words (excluding baseline scores) to compare stem completion for different degrees of repetition. A mixed 2(repetition: single vs repeated)×2(drink: placebo vs alcohol) ANOVA was performed on the percentage of words completed for the veridical and critical items separately. An effect of repetition was found for the veridical items [$F(1, 30)=5.98$, $p=0.021$] but not for the critical items [$F(1, 30)=0.12$, $p=0.729$] indicating that completion rates increased with repetition for the items studied at encoding (data not shown). There were no interactions found between drink and repetition [Fs(1, 30) <0.66, Ps>0.422].

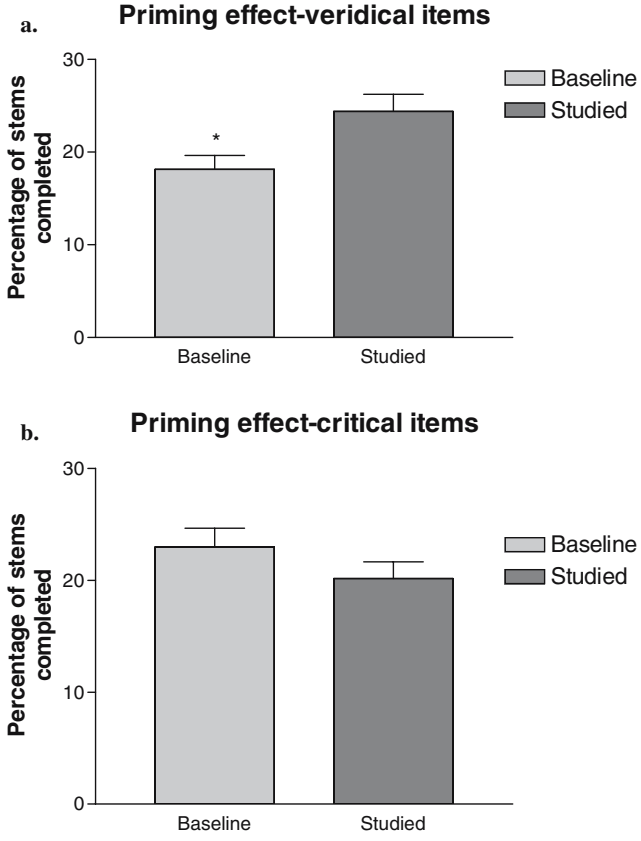

Fig. 3 Percentage of stems completed (mean±SEM) to form veridical (**a**) or critical (**b**) items corresponding to those viewed in the encoding phase (*studied*) vs the percentages of stems completed to form words not encountered in the encoding phase (*baseline*) for the alcohol and placebo groups. *$p<0.05$ compared to studied words

As a consequence of the non-significant priming effect in critical items, conclusions regarding implicit memory can only relate to veridical items, but the critical priming data can still be used to generate measures of aware and unaware items.

Awareness measures

Analyses were performed to compare the relative rates of aware and unaware items, and how they differed as a function of alcohol and repetition.

Veridical items A mixed 2(drink: placebo vs alcohol)×2 (awareness: aware vs unaware)×2(repetition: presented once vs repeated three times) ANOVA was performed on the percentages of stems completed of the veridical items or of their semantic associates (critical items) encountered in the encoding phase that were presented during the implicit task. The drink served as a between subjects variable, whilst repetition and awareness were within subjects variables.

A main effect of awareness was found, signifying more aware than unaware items [$F(1, 30)=8.82$, $p=0.006$]; thus once participants had completed a stem to correspond to an item viewed at encoding, they were more likely to endorse it as an item they were aware of, than fail to do so. A main effect of repetition was also found [$F(1, 30)=5.98$, $p=0.021$], demonstrating that repetition served to increase the probability that a stem would be completed to form veridical items viewed in the encoding phase. An awareness × repetition interaction [$F(1, 30)=14.26$, $p=0.001$], signified that repetition significantly increased the amount of aware items [$t(31)=-3.54$, $p=0.001$], whilst had no effect on the amount of unaware items [$t(31)=1.22$, $p=0.234$]. An awareness × repetition × drink interaction [$F(1, 30)=4.71$, $p=0.038$], was further explored using two-way ANOVAs separately for the alcohol and placebo groups. In the placebo group, the repetition × awareness interaction was non-significant [$F(1, 15)=1.24$, $p=0.283$; Fig. 4a]. In contrast, analysis in the alcohol group found a significant repetition × awareness interaction [$F(1, 15)=18.48$, $p=0.001$]. This interaction was further explored to reveal that repetition significantly decreased the amount of unaware veridical items [$t(15)=2.18$, $p=0.046$] but increased the amount of aware veridical items [$t(15)=-3.39$, $p=0.004$] within the alcohol group (see Fig. 4b).

Critical items A mixed 2(drink: placebo vs alcohol)×2 (awareness: aware vs unaware)×2(repetition: presented once vs repeated three times) ANOVA was performed on the amount of stems completed to correspond to critical items semantically related to veridical items encountered in the encoding phase. The drink was a between subjects variable, whilst awareness and repetition were both within subjects variables.

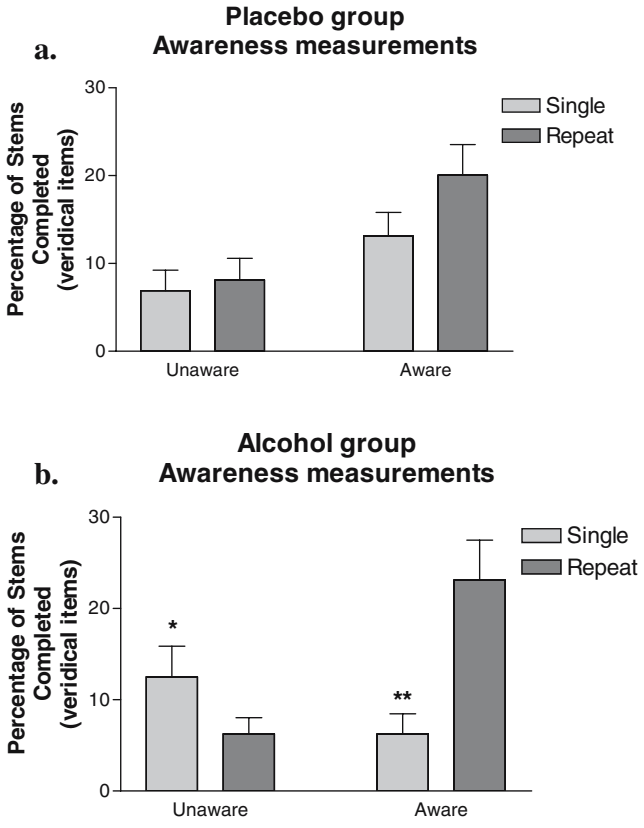

Fig. 4 Number of veridical stems completed (mean±SEM) and rejected (*unaware*) or accepted (*aware*) as previously seen in placebo (**a**) and in alcohol (**b**) participants for both the singularly (*single*) and three times (*repeat*) presented lists. *$p<0.05$, **$p<0.01$ compared to single presentation in aware and unaware judgement

A main effect of awareness was found [$F(1, 30)=4.53$, $p=0.042$], reflecting the finding that participants were more aware than unaware of critical items. There was no significant main effect of repetition [$F(1, 30)=0.12$, $p=0.729$], and neither the awareness × repetition [$F(1, 30)=0.35$, $p=0.558$], nor the drink × repetition [$F(1, 30)=0.66$, $p=0.442$] interactions were found to be significant. A significant awareness × repetition × drink interaction [$F(1, 30)=8.75$, $p=0.006$] was further explored using separate two-way ANOVAs for the alcohol and placebo groups. In the placebo group, a significant repetition × awareness interaction $F(1, 15)=5.44$, $p=0.034$], was further explored to reveal that repetition increased the amount of unaware critical words [$t(15)=-2.24$, $p=0.041$], but did not affect critical aware words [$t(30)=1.31$, $p=0.211$]. As being unaware of critical items constitutes accurate memory, being unaware of them, thus, amounts to being able to correctly reject them, and this was enhanced in the placebo group as a function of repetition. In the alcohol group, a repetition × awareness interaction approached significance [$F(1, 15)=4.24$, $p=0.057$]. This was further explored to reveal that repetition had a borderline significant tendency to decrease the amount of unaware critical items [$t(15)=2.09$, $p=0.055$]. In contrast, repetition did not affect the amount of aware items [$t(15)=-1.37$, $p=0.191$]. As depicted in Fig. 5a, b, a double dissociation was obtained, as repetition was found to have opposite effects on levels of aware and unaware critical items, dependent on the drink consumed. Repetition was thus found to increase the accuracy of memory for participants in the placebo group, and decrease memory accuracy for participants in the alcohol group.

Discussion

The present study investigated how alcohol and repetition given at encoding modulated veridical and false memory levels tested 24 h later, and whether these two distinct ways of manipulating encoding interacted. Repetition was found to effectively modulate the degree of information encoding for both alcohol and placebo participants, as a main effect of repetition signified that viewing lists multiple times increased the probability of veridical recall. In addition, and in accordance with the established anterograde impairments of alcohol on explicit memory (e.g. Birnbaum and Parker 1977; Birnbaum et al. 1978; Duka et al. 2001; Weissenborn and Duka 2000), participants in the alcohol group recalled significantly less singularly presented veridical items than placebo participants. This deleterious effect of alcohol was rendered non-significant through increased learning, as recall levels for repeated veridical items did not differ as a function of the drink.

Participants in the alcohol group also recalled significantly less false memory items from singularly presented lists than the placebo group. The drink was not found to affect false recall for items from repeated lists. Levels of false recall were thus found to mirror patterns of veridical memory in the alcohol and placebo groups. A meta-analysis conducted by Roediger et al. (2001) demonstrated that levels of veridical recall significantly predicted the degree of false memory levels. Consequently, the decrease in false memories in the alcohol group relative to the placebo group for false memory items from singularly presented lists could be the direct result of decreased veridical memory for singularly presented lists. Craik (1977) hypothesised that alcohol impaired veridical memory via a reduction of encoding depth, leading to superficial encoding and a reduction in semantic processing. Depth of processing has been shown to predict the degree of semantic activation, affecting levels of false memory (Rhodes and Anastasi 2000; Thapar and McDermott 2001; Toglia et al. 1999). Superficial encoding under an alcohol challenge could account for the effects of alcohol to decrease both veridical and false memory. The effect of alcohol to decrease false memory recall differs from previous findings (Mintzer and Griffiths 2001; Milani and Curran 2000) showing no effect

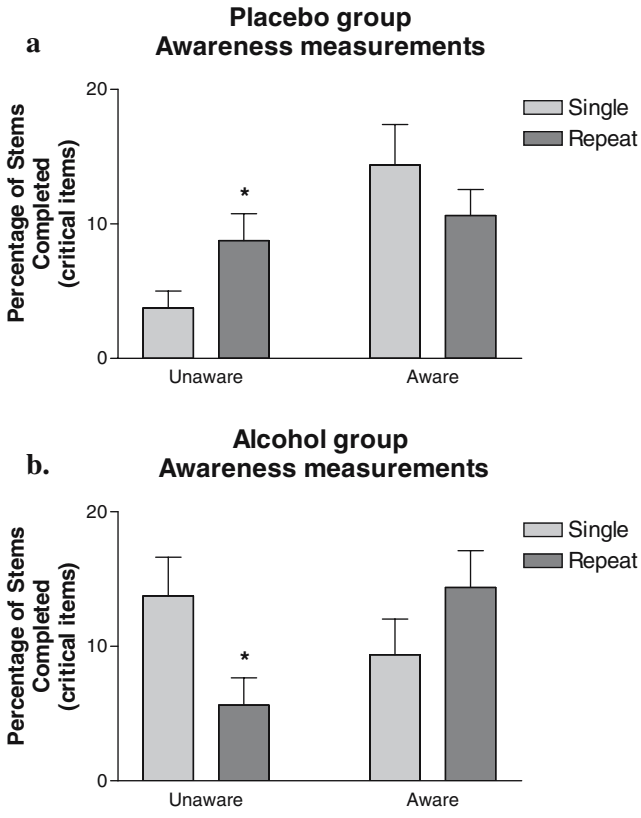

Fig. 5 Number of critical stems completed (mean±SEM) and rejected (*unaware*) or erroneously accepted (*aware*) as previously seen in placebo (**a**) and in alcohol (**b**) participants for both the singularly (*single*) and three times (*repeat*) presented lists. *$p<0.05$ compared to single presentation in unaware judgement

or a tendency for alcohol to increase the false recognition rates, respectively. The previous studies, however, differed from the present study in that they did not test recall but recognition and testing took place under the influence of alcohol.

The implicit instructions for the stem completion task ('complete the stems to form the first word to come to mind') resulted in a priming effect for veridical items, but not critical items. The priming found with veridical items did not differ as a function of the drink in accordance with previous data showing that priming (measured also 24 h after encoding) was unaffected by the drink consumed (Duka et al. 2001). It is also in accordance with empirical research that has demonstrated the preservation of automatic components of memory under alcohol (Kirchner and Sayette 2003; Tracy and Bates 1999). In contrast, no priming effect was obtained in critical items. This appears contrary to researchers who demonstrated a significant effect of priming in critical items using stem completion (Hicks and Starns 2005; McKone and Murphy 2000). These researchers, however, used immediate stem completion, as opposed to the 24-h retention interval employed in the current experiment. This differential effect of priming for critical and veridical items over an extended retention interval thus provides an interesting insight into the relative durability of traces underlying true and false memory items. Repetition was found to increase rates of completion in the veridical items, whilst left the rates of completion with critical items unaffected. This finding suggests that durability of traces underlying true memory can be further supported by repetition.

Participants were required to make awareness judgements for the words they had completed under implicit instructions, following a procedure used by Duka et al. (2001). By circling a word, participants were endorsing it as having been presented at encoding, thus proclaiming 'awareness' for its prior presentation. Awareness of veridical words, thus, constituted accurate memory. The reverse was true, however, for false memory items. For, as they were never presented, proclaiming 'awareness' of their presentation was thus incorrect. As a priming effect was only achieved in veridical items, a conclusion regarding the automatic influences of memory as indicated by levels of unaware items is restricted to veridical items (Duka et al. 2001). Whilst aware and unaware measures can still be obtained for critical items, conclusions cannot be derived from these measures in terms of automatic and controlled memory processes. This is because stem completion for critical items was at chance and consequently one cannot infer that automatic memory processes aided their completion.

For the veridical items, repetition was found to increase stem completion under implicit instructions. It is interesting to note that research into the effect of multiple repetitions on implicit memory is unclear (Jacoby and Dallas 1991; Roediger and Challis 1992). Whilst research has demonstrated no effect of study list repetition on implicit memory (Challis and Sidhu 1993; Parkin et al. 1990), the present finding is consistent with more recent research that has found a significant priming effect for multiply presented words over singly presented words (Erickson and Reder, unpublished manuscript). This finding can be accounted for using the Source of Activation Confusion theory (e.g. Reder and Schunn 1996), which implies that multiple repetitions of study items should enhance priming relative to single presentation of items due to an increase in activation of corresponding *concept* (word) nodes. Multiple presentations, hence, increase the potential for super threshold activation of the word in question, which leads into an enhanced priming effect. Such a finding substantiates the use of repetition as a modulator to enhance encoding and demonstrates that this effect of repetition to enhance encoding can be obtained using implicit instructions.

Awareness status for veridical words differed as a function of repetition in alcohol participants. This was

not true for placebo participants who were more aware of presented words than unaware of them irrespective of how many times the words were presented (Fig. 4a). Specifically, in the alcohol group, increased repetitions served to bring veridical items into conscious awareness; for words viewed just once, participants were significantly more likely to be unaware of their presentation relative to words viewed repeated times (Fig. 4b). This finding supports empirical research which documents the differential impairment of alcohol on automatic and controlled memory processes (Duka et al. 2001; Kirchner and Sayette 2003; Lister et al. 1991; Tracy and Bates 1999). As priming was not affected by the drink, automatic memory influences, as quantified using degree of priming for veridical items, were not found to be affected by the drink consumed. Explicit awareness of previous presentation of words generated under implicit instructions, however, was found to be affected by the drink. In addition, explicit awareness of previous presentation of veridical items was mediated by repetition in the alcohol group only, as increased learning brought these words into conscious awareness counteracting the deleterious effect of alcohol on encoding.

Awareness measures taken for critical items initially completed under implicit instructions demonstrated that repetition had opposite effects in placebo and alcohol participants. Awareness, and thus endorsement, of critical items increased with repetition in alcohol participants, whilst no such effect was present in the placebo group. In addition, placebo participants were significantly better at rejecting false memory items from repeated lists than from lists presented once, whilst the reverse was true for alcohol participants. Thus, regarding memory accuracy as indexed by false memory endorsement, placebo participants' memory got more accurate with repetition, whilst accuracy for participants in the alcohol group declined.

This finding can be accounted for using the AMF (Roediger et al. 2001). Under the AMF, activation of the critical item can occur via direct activation of semantic networks as a result of the formation of explicit associations between veridical items during list presentation (Underwood 1965). Critical items are also thought to be activated by the automatic spread of activation within semantic networks. Under this latter route, conscious thought of the critical item is not deemed necessary, as the mere processing of veridical items is considered sufficient for activation to spread to the critical item. The effect of alcohol to increase false memories during free recall (as discussed above) can be explained by its ability to block activation of explicit associations during encoding. Alcohol should leave unaffected the automatic spread of activation within semantic networks as previous work would suggest (Kirchner and Sayette 2003).

The monitoring component within the AMF functions to ascertain the source of the semantic activation with erroneous attribution of familiarity thought to derive from prior presentation leading to false memory. Correctly identifying the source of the activation reflects a successful 'reality monitoring process' (Johnson et al. 1993), as it supposedly demonstrates the ability of individuals to successfully attribute the activation of the critical items to internal (e.g. thinking of the critical items) as opposed to external (e.g. prior presentation) factors. It has been argued that such a process is dependent upon intact recollective memory (Benjamin 2001).

It is possible that due to impaired learning under alcohol, activation levels are initially (under single presentation) reduced. Repeated presentation, however, may lead to increased activation of semantic networks and, hence, to an increase in false memories. In contrast, placebo participants are able to use repetition to increase the richness of their memory traces. Such a richness would mean when making awareness judgments for false memory items, they are better able to differentiate between which items were presented and which were not—resulting in an enhanced ability to reject critical false memory items with repetition, something that alcohol participants are unable to do because of their shallow processing. This differential effect of repetition on false memory endorsement is consistent with experiments that have shown increased false memories as a function of repetition in populations with impaired recollective processes such as the elderly (Benjamin 2001).

Thus, in summary, the present study has shown a differential effect of alcohol and repetition on the generation of false memories, and on accuracy judgements in recognition of false memories in comparison to placebo. Alcohol relative to placebo decreased the generation of false memories for items presented once; repeated presentations of items counteracted this effect of alcohol. On the other hand, alcohol decreased accuracy judgements for repeated items whilst an increase in accuracy judgement was seen with repeated presentation of items in the placebo participants. The activation and monitoring framework is used to explain these effects of repetition and alcohol.

References

Benjamin AS (2001) On the dual effects of repetition on false recognition. J Exp Psychol Learn Mem Cogn 27:941–947

Birnbaum IM, Parker ES (1977) Alcohol and human memory. Erlbaum, New Jersey

Birnbaum IM, Parker ES, Hartley JT, Noble EP (1978) Alcohol and memory—retrieval processes. J Verbal Learn Verbal Behav 17:325–335

Birnbaum IM, Johnson MK, Hartley JT, Taylor TH (1980) Alcohol and elaborative schemas for sentences. J Exp Psychol [Hum Learn] 6:293–300

Challis BH, Sidhu R (1993) Dissociative effect of massed repetition on implicit and explicit measures of memory. J Exp Psychol Learn Mem Cogn 19:115–127

Craik F (1977) Similarities between the effects of ageing and alcoholic intoxication on memory performance, construed within a 'levels of processing' framework. In: Birnbaum IM, Parker ES (eds) Alcohol and human memory. Erlbaum, Hillsdale, NJ, pp 9–21

Craik FIM, Lockhart RS (1972) Levels of processing—framework for memory research. J Verbal Learn Verbal Behav 11:671–684

Curran HV, Hildebrandt M (1999) Dissociative effects of alcohol on recollective experience. Conscious Cogn 8:497–509

Deese J (1959) On the prediction of occurrence of particular verbal intrusions in immediate recall. J Exp Psychol 58:17–22

Duka T, Curran HV, Rusted JM, Weingartner HJ (1996) Perspectives on cognitive psychopharmacology research. Behav Pharmacol 7:401–410

Duka T, Stephens DN, Russell C, Tasker R (1998) Discriminative stimulus properties of low doses of ethanol in humans. Psychopharmacology (Berl) 136:379–389

Duka T, Weissenborn R, Dienes Z (2001) State-dependent effects of alcohol on recollective experience, familiarity and awareness of memories. Psychopharmacology (Berl) 153:295–306

Gallo DA, Roediger HL 3rd (2002) Variability among word lists in evoking memory illusions: evidence for associative activation and monitoring. J Mem Lang 47:469–497

Graf P, Schacter DL (1985) Implicit and explicit memory for new associations in normal and amnesic subjects. J Exp Psychol Learn Mem Cogn 11:501–518

Hicks JL, Starns JJ (2005) False memories lack perceptual detail: evidence from implicit word-stem completion and perceptual identification tests. J Mem Lang 52:309–321

Huron C, Servais C, Danion JM (2001) Lorazepam and diazepam impair true, but not false, recognition in healthy volunteers. Psychopharmacology (Berl) 155:204–209

Jacoby LL, Dallas M (1991) On the relationshop between autobiographical memory and perceptual learning. J Exp Psychol Learn Mem Cogn 110:306–340

Johnson MK (1977) What is bring counted none the less? Erlbaum, New Jersey

Johnson MK, Hashtroudi S, Lindsay DS (1993) Source monitoring. Psychol Bull 114:3–28

Kirchner TR, Sayette MA (2003) Effects of alcohol on controlled and automatic memory processes. Exp Clin Psychopharmacol 11:167–175

Kiss GR, Armstrong C, Milroy R, Piper J (1973) An associative thesaurus of English and its computer analysis. University Press, Edinburgh

Knowles SK, Duka T (2004) Does alcohol affect memory for emotional and non-emotional experiences in different ways? Behav Pharmacol 15:111–121

Kucera H, Francis WN (1967) Computational analysis of present-day American English. Brown University Press, Rhode Island

Lezak MD (1983) Neuropsychological assessment. Oxford University Press, New York

Libby LK, Neisser U (2001) Structure and strategy in the associative false memory paradigm. Memory 9:145–163

Lister RG, Gorenstein C, Fisher-Flowers D, Weingartner HJ, Eckardt MJ (1991) Dissociation of the acute effects of alcohol on implicit and explicit memory processes. Neuropsychologia 29:1205–1212

McEvoy CL, Nelson DL, Komatsu T (1999) What is the connection between true and false memories? The differential roles of interitem associations in recall and recognition. J Exp Psychol Learn Mem Cogn 25:1177–1194

McKone E, Murphy B (2000) Implicit false memory: effects of modality and multiple study presentations on long-lived semantic priming. J Mem Lang 43:89–109

Mehrabian A, Russell JA (1978) A questionnaire measure of habitual alcohol use. Psychol Rep 43:803–806

Milani R, Curran HV (2000) Effects of a low dose of alcohol on recollective experience of illusory memory. Psychopharmacology (Berl) 147:397–402

Mintzer MZ, Griffiths RR (2001) Alcohol and false recognition: a dose–effect study. Psychopharmacology (Berl) 159:51–57

Parker ES, Alkana RL, Birnbaum IM, Hartley JT, Noble EP (1974) Alcohol and the disruption of cognitive processes. Arch Gen Psychiatry 31:824–828

Parker ES, Birnbaum IM, Noble EP (1976) Alcohol and memory—storage and state dependency. J Verbal Learn Verbal Behav 15:691–702

Parkin AJ, Reid TK, Russo R (1990) On the differential nature of implicit and explicit memory. Mem Cognit 18:514

Reder LM, Schunn CD (1996) Metacognition does not imply awareness: strategy choice is governed by implicit learning and memory. In: Reder LM (ed) Implicit memory and metacognition. Erlbaum, Hillsdale, pp 45–77

Rhodes MG, Anastasi JS (2000) The effects of a levels-of-processing manipulation on false recall. Psychon Bull Rev 7:158–162

Roediger HL 3rd, McDermott KB (1995) Creating false memories: remember words not presented in list. J Exp Psychol Learn Mem Cogn 21:803–814

Roediger HL 3rd, Watson JM, McDermott KB, Gallo DA (2001) Factors that determine false recall: a multiple regression analysis. Psychon Bull Rev 8:385–407

Roediger HL, Challis BH (1992) Effects of exact repetition and conceptual repetition on free recall and primed word-fragment completion. J Exp Psychol Learn Mem Cogn 18:3–14

Schweizer TA, Vogel-Sprott M, Dixon MJ, Jolicoeur P (2005) The stage-specific effect of alcohol on human information processing. Psychopharmacology (Berl) 178:52–57

Seamon JG, Luo CR, Schwartz MA, Jones LJ, Lee DM, Jones SJ (2002) Repetition can have similar or different effects on accurate and false recognition. J Mem Lang 46:323–340

Stadler MA, Roediger HL 3rd, McDermott KB (1999) Norms for word lists that create false memories. Mem Cognit 27:494–500

Thapar A, McDermott KB (2001) False recall and false recognition induced by presentation of associated words: effects of retention interval and level of processing. Mem Cognit 29:424–432

Toglia MP, Neuschatz JS, Goodwin KA (1999) Recall accuracy and illusory memories: when more is less. Memory 7:233–256

Townshend JM, Duka T (2002) Patterns of alcohol drinking in a population of young social drinkers: a comparison of questionnaire and diary measures. Alcohol Alcohol 37:187–192

Tracy JI, Bates ME (1999) The selective effects of alcohol on automatic and effortful memory processes. Neuropsychology 13:282–290

Tussing AA, Greene RL (1997) False recognition of associates: how robust is the effect? Psychon Bull Rev 4:572–576

Tussing AA, Greene RL (1999) Differential effects of repetition on true and false recognition. J Mem Lang 40:520–533

Underwood BJ (1965) False recognition produced by implicit verbal responses. J Exp Psychol 70:122–129

Weissenborn R, Duka T (2000) State-dependent effects of alcohol on explicit memory: the role of semantic associations. Psychopharmacology (Berl) 149:98–106