warwick.ac.uk/lib-publications

Differential Effects of Alcohol on Associative Versus Item Memory

Elizabeth A. Maylor, Hannah R. Long, and Rhianne A. Newstead

University of Warwick, UK

Author Note

Elizabeth A. Maylor, Hannah R. Long, and Rhianne A. Newstead, Department of Psychology, University of Warwick, United Kingdom.

Correspondence concerning this article should be addressed to Elizabeth A. Maylor, Department of Psychology, University of Warwick, Coventry, CV4 7AL, United Kingdom. Email: e.a.maylor@warwick.ac.uk

Abstract

Alcohol has detrimental effects on a range of cognitive processes, the most prominent being episodic memory. These deficits appear functionally similar to those observed within the normal aging population. We investigated whether an associative memory deficit, as found in older adults, would also be evident in young adults moderately intoxicated by alcohol. Participants were shown unrelated word pairs and then tested on both their item recognition (old/new item?) and associative recognition (intact/recombined pair?). Half the participants were under the influence of alcohol whereas the other half were sober. Alcohol impaired memory performance but significantly more so for associative than for item memory. Moreover, within the alcohol group, the associative memory deficit was significantly related to the amount of alcohol consumed. The findings suggest that not all aspects of episodic memory are equally impaired by alcohol, which may have practical implications for criminal investigations involving eye witnesses who have consumed alcohol.

Keywords: alcohol, recognition, item memory, associative memory

Differential Effects of Alcohol on Associative Versus Item Memory

Learning, retaining and retrieving links between individual elements of an episode is referred to as associative memory (Naveh-Benjamin & Mayr, 2018) and is fundamental to everyday functioning. Associative memory failures vary in severity, from trivial mistakes like forgetting someone's name, to significant errors such as identifying the wrong suspect as the one holding the gun. It is well established that normal aging is detrimental to associative memory due to deficits in binding at encoding, coupled with the inability to retrieve such bound units (Naveh-Benjamin, 2000). Our aim in this study was to investigate – for the first time, as far as we are aware – whether the associative deficit found in aging also occurs after consuming alcohol, as there are many functional similarities between alcohol intoxication and aging (Craik, 1977; Maylor & Rabbitt, 1993; Rabbitt & Maylor, 1991).

Alcohol is one of the world's most popular beverages, especially among University students, who binge-drink regularly as defined by more than five units of alcohol in one session (Chen, Dufour, & Yi, 2004; Wicki, Juntsche, & Gmel, 2010). Unfortunately, there are costs in both the short-term (Acheson, Stein, & Swartzwelder, 1998) as well as the long-term (Jennison, 2004). Studies of the acute effects of alcohol have revealed numerous cognitive deficits including decision making (George, Rogers, & Duka, 2005), executive functioning (Lyvers & Maltzman, 1991), divided attention (Maylor, Rabbitt, James, & Kerr, 1990b), visual search (Maylor & Rabbitt, 1988), and processing speed (Maylor & Rabbitt, 1987b). However, the most well documented alcohol-related impairment is that of memory, including working memory (Finn, Justus, Mazas, & Steinmetz, 1999), prose recall (Maylor, Rabbitt, James, Kerr, 1990a; Petros, Kerbel, Beckwith, Sacks, & Sarafolean, 1985), prospective memory (Leitz, Morgan, Bisby, Rendell, & Curran, 2009), picture recognition (Parker, Birnbaum, & Noble, 1976), and word recognition (Maylor & Rabbitt, 1987a; Maylor, Rabbitt, & Kingstone, 1987).

Craik (1977, 1983) proposed that such memory deficits found in individuals intoxicated via alcohol might be similar in a functional sense to the memory deficits found in aging individuals (e.g., Naveh-Benjamin, Moscovitch, & Roediger, 2001). Both alcohol and aging have general effects of slowed processing/reduced resources (see Maylor & Rabbitt, 1993; Rabbitt & Maylor, 1991), with the consequence that effortful (controlled) processes are substantially impaired whereas automatic processes remain relatively intact (cf. Hasher & Zacks, 1979; Kirchner & Sayette, 2003). Evidence in support of this notion comes from a study by Nilsson, Bäckman, and Karlsson (1989) who investigated cued recall and priming in young, old, and alcohol-intoxicated groups. Cued recall for weakly related word pairs was impaired in the latter two groups whereas cued recall for strongly related word pairs and for priming was unimpaired.

The associative deficit hypothesis (ADH) of aging derived from early studies (e.g., Gilbert, 1941) that seemed to show that older adults have an especial difficulty in forming associations between items relative to remembering the items themselves (Naveh-Benjamin, 2000; Naveh-Benjamin, Hussain, Guez, & Bar-On, 2003). This was demonstrated with a simple paradigm in which participants were presented with pairs of unrelated items and were told to memorize both the items themselves and their pairings. Two recognition memory tests followed – in one test, participants responded old/new to previously-presented/new individual items (item memory test); in the other test, participants responded old/new to intact/recombined pairings (associative memory test). A typical pattern of results is that item and associative memory are similar in young adults and also quite similar to item memory in older adults, whereas older adults' associative memory is much poorer, producing an age by memory type interaction and hence support for the ADH (see Old & Naveh-Benjamin, 2008, for a review of studies across a range of stimuli).

The ADH is often interpreted in terms of dual-process models of memory (Yonelinas, 2002). Thus, to complete the item test successfully, automatic familiarity-based processes are sufficient; however, effortful recollection-based processes are necessary to complete the associative test successfully because items making up intact and recombined pairings are matched in terms of familiarity. As reviewed by Light, Prull, La Voie, and Healy (2000; cf. Hasher & Zacks, 1979), effortful processing is more impaired by aging than is automatic processing, and thus an age-related associative deficit occurs (see also Castel & Craik, 2003; Naveh-Benjamin et al., 2009; Shing, Werkle-Bergner, Li, & Lindenberger, 2008).

In the present study, we examined the acute effects of alcohol under naturalistic conditions of social drinking in young adults. We employed the same recognition memory paradigm with unrelated word pairs as Naveh-Benjamin (2000; see also Maylor & Badham, 2018). In view of the supposed parallels between the effects of aging and alcohol, we predicted a similar interaction between alcohol condition and memory type. Thus we expected item and associative memory in sober individuals to be approximately equivalent and also similar to item memory in intoxicated individuals, but we predicted much lower performance for associative memory in intoxicated individuals. Note that although there have been some investigations of the effects on associative memory of acute doses of alcohol (e.g., Duka, Weissenborn, & Dienes, 2001; Nilsson et al., 1989) and of long-term alcohol intake (e.g., Kessler, Irle, & Markowitsch, 1986), there has never been to our knowledge a study undertaking the crucial comparison between effects on item versus associative memory, which is essential in order to test the ADH.

Method

Participants

Sixty-four undergraduate students aged between 19 and 24 years at the University of Warwick took part in the experiment, which was approved by the Psychology Department's Research Ethics Committee at the University of Warwick. One participant did not follow instructions and was therefore replaced. There were 32 participants (16 males, 16 females) in each of the two conditions (no alcohol and alcohol). All participants reported having normal or corrected-to-normal vision and were fluent English speakers.

Testing always took place between 20:00-23:00 and was carried out on multiple occasions over several weeks. Potential participants were approached as they entered the bar in the Student Union on the University of Warwick campus and were asked if they would be willing to take part in a study investigating the effect of alcohol on memory. They were also asked whether they had consumed any alcohol within the last 12 hours (those who said they had were excluded from participation) and whether they intended to consume alcohol that evening (those who said they were not were also excluded). If they agreed to take part, and provided written informed consent, they were then systematically assigned (in a strictly alternating fashion) either to be tested immediately ("no-alcohol" group) or to return later after having consumed a "reasonable amount of alcohol" ("alcohol" group).[1] As already mentioned, based on self-report data, the former group had not consumed any alcohol within the previous 12 hours. All the latter participants self-reported that they had exclusively consumed 'Snakebite', an alcoholic drink consisting of equal amounts of cider and lager with a small amount of blackcurrant cordial, which is approximately 4.5% alcohol by volume. (Snakebite is an extremely popular beverage served in bars on the University campus, traditionally drunk by members of societies and available at a discounted price.) The alcohol group self-reported having consumed between two and eight pints, with a mean of 4.67 pints ($SD = 1.46$), which is equivalent to 12.14 units of alcohol. According to the classifications of alcohol consumption used in a previous field study of self-administered

alcohol by Oorsouw and Merckelbach (2012), the present participants were borderline moderately intoxicated (which they classified as 5.48 pints). Furthermore, they would be considered as binge drinking, having consumed more than five units of alcohol in one session (Chen et al., 2004).

Materials

The experimental stimuli were 81 high-frequency bisyllabic nouns of 4-8 letters in length. The study list comprised 30 word pairs in which the two words in each pair were unrelated semantically, acoustically and visually (e.g., *ruler–chicken*, *sulphur–cowboy*). Presentation order of the word pairs was randomized for each participant. In the item recognition memory test, there were 20 old words from the study list and 21 new words, presented in a different random order for each participant except that the final word (a new item) was always the word "purple" (see below). In the associative recognition memory test, there were 10 intact word pairs from the study list and 10 "recombined" word pairs with the left-hand word from one pair presented together with the right-hand word from another pair, again presented in a different random order for each participant. Each word from the study list appeared once only in the test phase, either in the item test or in the associative test.

Stimuli appeared in black on a white background and were presented in lower case in a large font size at the center of an 11-inch laptop computer screen. A long hyphen was used to separate the items in word pairs during study and in the associative recognition test.

Procedure

Participants were tested individually in a relatively quiet secluded area away from the bar. The room was large, with high ceilings, such that background noise from the bar at the other end of the room (mostly indistinct chatter) was not too distracting. Moreover, participants were seated at tables and chairs facing away from the bar and towards a wall.

After recruitment, allocation to conditions, and drink consumption as appropriate (all as described in the Participants section), participants were instructed that they would see a list of 30 word pairs presented sequentially on a computer screen at a rate of 4 s per pair and were explicitly asked to remember the words and the associations between them for later memory tests. Following the study phase, there was a distractor task of counting backwards in threes from 300 for 60 s before the item and associative recognition tests were administered, test order being fully counterbalanced across participants in both no-alcohol and alcohol conditions. In the item memory test, 40 words (20 old; 21 new) were presented sequentially on the screen and participants were required to respond verbally (yes/no) according to whether or not they had seen each word in the study phase. In the associative memory test, 20 word pairs (10 intact; 10 recombined) were presented sequentially and participants were required to respond yes/no according to whether or not they had seen the words paired together in the study phase. There were no time limits imposed on responding in the test phase – once a verbal response was made, the experimenter initiated the next trial. Before starting the experiment, participants received practice at the task with a study list of five word pairs, followed by item and associative memory tests (test order being the same as that assigned for the experimental trials), each with four trials. None of the practice words was included in the main experiment.

Following the main instructions, participants were additionally told that if at any point during the memory tests they saw the name of a color on the screen, they should not answer 'yes' or 'no' but should tell the experimenter the name of the color instead. This was included as a test of event-based prospective memory (cf. Maylor, Darby, Logie, Della Sala, & Smith, 2002); the prospective memory target ('purple') was always the final (new) word on the item recognition memory test. All participants apparently understood the prospective memory requirements at the time of encoding, as indicated by their ability to correctly repeat

them back to the experimenter. Moreover, almost all of them were able to recall the task correctly at the end of the experiment when specifically prompted by the experimenter asking if there had been anything else they were expected to do. As the results of this additional task were as expected from the alcohol literature, we only briefly summarize them here before focusing on our main (novel) findings. Thus, in line with at least two previous studies (Leitz, Morgan, Bisby, Rendell, & Curran, 2009; Paraskevaides et al., 2010), significantly fewer participants in the alcohol condition were successful in actually carrying out the prospective memory task unprompted in comparison with those in the no-alcohol condition (2/32 vs. 16/32, respectively, $\chi^2(1) = 15.15$, $p < .001$).

Results

Age

In a 2 (condition: no alcohol, alcohol) $\times$ 2 (gender) factorial ANOVA on age, there was no effect of gender, $F(1, 60) = 1.36$, $MSE = 0.74$, $p = .248$, $\eta_p^2 = .022$, and no interaction between condition and gender, $F < 1$. However, there was a significant effect of condition, $F(1, 60) = 8.50$, $MSE = 0.74$, $p = .005$, $\eta_p^2 = .124$, such that those in the no-alcohol condition ($M = 20.91$, 95% CI: 20.60–21.21) were slightly older than those in the alcohol condition ($M = 20.28$, 95% CI: 19.98–20.58). In view of this age difference, the following analyses included age as a covariate and the means in tables and figures are correspondingly age-adjusted.[2]

Recognition Memory

For item recognition memory, performance was scored in terms of hit rates (i.e., yes responses to old items, as a percentage) and false alarm rates (i.e., yes responses to new items, as a percentage, excluding responses to the prospective memory target). Similarly, for associative recognition memory, hit and false alarm rates were calculated on the basis of yes responses to intact and recombined pairs, respectively, as percentages. Table 1 summarizes

the hit and false alarm rates. Corrected recognition measures of performance were calculated as percentage hits minus percentage false alarms (see Figure 1), but also signal detection measures of sensitivity, d', and response bias, c, were derived (following Stanislaw & Todorov, 1999) from participants' hit and false alarm rates (see Table 2). Analyses based on corrected recognition and d' were qualitatively identical and so we focus here on the former.

Two initial ANCOVAs (age as the covariate) were performed on corrected recognition measures. The first included condition (no alcohol vs. alcohol) and gender (male vs. female) as between-subjects factors, and memory type (item vs. associative) as a within-subjects factor. This revealed no effect of gender and no interactions involving gender, all Fs < 1. The second replaced gender with task order (item-associative vs. associative-item) as the other between-subjects factor; again, there was no effect of order and no interactions involving order, all Fs < 1. Thus both gender and task order were dropped as factors to be considered.

Corrected recognition measures were next analyzed by a 2×2 (alcohol condition $\times$ memory type) between-within ANCOVA (covarying age). There was a significant main effect of condition, $F(1, 61) = 11.57$, $MSE = 1012.59$, $p = .001$, $\eta_p^2 = .159$, and a significant interaction between condition and memory type, $F(1, 61) = 6.15$, $MSE = 446.76$, $p = .016$, $\eta_p^2 = .092$. Separate one-way ANCOVAs on item and associative memory revealed significant effects of alcohol for both item memory, $F(1, 61) = 4.07$, $MSE = 383.10$, $p = .048$, $\eta_p^2 = .063$, and associative memory, $F(1, 61) = 11.99$, $MSE = 1076.25$, $p < .001$, $\eta_p^2 = .164$, though clearly the influence of alcohol was stronger for the latter. As can be seen in Figure 1, whereas recognition memory was similar for item and associative memory with no alcohol, $t(31) = 1.47$, $p = .153$, item memory was significantly higher than associative memory with alcohol, $t(31) = 4.18$, $p < .001$, again demonstrating a clear differential effect of alcohol on associative relative to item memory.

Further ANCOVAs were conducted to investigate the source of this effect by separately analyzing hit and false alarm rates (see Table 1 for means). For both hit and false alarm rates, there were significant effects of condition, $F(1, 61) = 6.89$, $MSE = 412.75$, $p = .011$, $\eta_p^2 = .102$, and $F(1, 61) = 6.27$, $MSE = 481.36$, $p = .015$, $\eta_p^2 = .093$, respectively, indicating fewer hits and more false alarms in the alcohol than in the no-alcohol condition. Crucially, whereas the interaction between condition and memory type was not significant for hits, $F(1, 61) = 1.91$, $MSE = 197.88$, $p = .172$, $\eta_p^2 = .030$, it was significant for false alarms, $F(1, 61) = 5.47$, $MSE = 198.70$, $p = .023$, $\eta_p^2 = .082$. In other words, false alarm rates were higher with alcohol than without, but reliably more so for associative than for item memory. Thus although there were significant deficits due to alcohol for both hits and false alarms, the especially damaging effect of alcohol on associative memory (in comparison with item memory) was attributable more to false alarm rates than to hit rates.

The response bias measure, c (see Table 2), was slightly but significantly more positive for item than for associative memory, $p = .008$, suggesting a greater conservative bias toward responding no/not seen before for item memory. Such a difference has been noted before (Bender, Naveh-Benjamin, & Raz, 2010; Maylor & Badham, 2018). Importantly, there was no effect of condition and no interaction between condition and memory type (both Fs < 1), indicating no influence of alcohol on response bias.

Correlations with Alcohol Consumption

Within the alcohol condition ($n = 32$), we examined the relationship between the self-reported number of pints consumed and item and associative memory (see Figure 2 for scatterplots). For item memory, the correlation between corrected recognition and alcohol consumption was not significant, $r = -.076$, $p = .681$.[3] In contrast, the correlation for associative memory did reach significance, $r = -.429$, $p = .014$. Additionally, alcohol consumption correlated with the difference between item and associative memory, $r = .388$,

p = .028, again indicative of a specific impairment to associative memory due to alcohol consumption, in a dose-dependent manner.

Discussion

On the basis of analogies drawn in the literature between the cognitive effects of aging and of alcohol (e.g., Craik, 1977, 1983; Nilsson et al., 1989), our prediction was that the age-related ADH would apply equally to individuals under the influence of alcohol. We compared sober students with moderately intoxicated students in a social drinking context and found that, as predicted, there was a significantly greater influence of alcohol on associative recognition memory than on item recognition memory. Furthermore, this alcohol-related associative deficit was dose-dependent such that it increased significantly with self-reported alcohol consumption among the intoxicated group.

This differential impact of alcohol on associative memory (relative to item memory) is consistent with alcohol's known disruption of activity in the hippocampus (White, 2003; White, Matthews, & Best, 2000), a brain structure crucial to the formation of episodic memories in binding together different elements of an event (Henke, Weber, Kneifel, Wieser, & Buck, 1999; Sperling et al., 2003). An associative deficit with alcohol could also be attributable to executive functioning impairments (e.g., Lyvers & Maltzman, 1991; Weissenborn & Duka, 2003), possibly leading to failures to employ effective strategies at encoding to form lasting bonds between individual items in a word pair (cf. Naveh-Benjamin, 2000, on aging).

Notably, the pattern of associative deficit observed in the alcohol condition resembled that seen in older adults (e.g., Old & Naveh-Benjamin, 2008) such that it was false alarm rates for associative memory that were particularly increased (relative to item memory) by alcohol. In terms of dual-process models of memory (Yonelinas, 2002), this would be explained as an over-reliance on automatic familiarity-based processes rather than

engaging in effortful recollective processing or strategic retrieval (e.g., Rotello & Heit, 2000; cf. Cohn, Emrich, & Moscovitch, 2008, on aging). It should be emphasized that this is not the same as simply applying a more liberal response criterion – recall that there was no influence of alcohol whatsoever on the response bias measure, c.

Our results mesh well with eyewitness identification research as exemplified by Yuille and Tollestrup (1990) who had sober and intoxicated participants watch a staged theft and later attempt to identify the "thief" in photospreads that either contained or did not contain a picture of the thief. Whereas there was no effect of alcohol on participants' recognition of the thief in target-present lineups, in target-absent lineups, alcohol increased the number of false identifications. It is not uncommon for an alcohol-intoxicated individual to be a key witness to a violent crime (Haggård-Grann, Hallqvist, Långström, & Möller, 2006; Yuille & Tollestrup, 1990), and thus it may be important to consider alcohol-related associative deficits when reviewing eyewitness identifications. Our results suggest that even though individual items may be recognized relatively well under the influence of alcohol, their combination (e.g., whether Person A did Action X and Person B did Action Y, or vice versa) may lead to inaccurate responses.

Some alternative explanations of our data can be readily discounted. One is that participants in the alcohol condition were simply less motivated to perform well than those in the no-alcohol condition. However, the key result was the interaction between alcohol condition and memory type, with intoxicated participants performing much closer to sober participants on the item memory test than on the recognition memory test. It is unclear why intoxicated participants would be differentially motivated across the two tests. (Note that at least for sober participants, the associative memory test was apparently not much more difficult than the item memory test.)

The same counterargument applies to a second possibility, namely, intoxicated participants may have focused more of their resources on the prospective memory task, thereby damaging their performance on the ongoing word-pair memory task (see, e.g., Smith, 2003, on the costs of remembering to remember in event-based prospective memory tasks). Again, it is difficult to see why ongoing task costs would be different across the item and associative tests. Moreover, participants in the alcohol condition performed abysmally on the prospective memory task (see Method section) and therefore showed little evidence of favoring it at the expense of the ongoing task.

A third possibility relates to the absence of a placebo condition (e.g., non-alcoholic Snakebite) to assess the behavioral effects of alcohol expectations. Our participants knew that the study's aim was to measure the effect of alcohol on memory and they were obviously fully aware of the condition to which they had been assigned. Thus the results could be attributable to alcohol expectancy effects rather than to pharmacological effects of alcohol. However, whereas alcohol expectancy effects have been identified in some social domains, there is no evidence of such effects in nonsocial domains including memory (see Hull & Bond, 1986, for a meta-analysis), and therefore in these cases a placebo condition can be omitted (Testa et al., 2006).

Finally, some limitations of the present study need to be acknowledged. First, it was conducted in a naturalistic social setting rather than under distraction-free laboratory conditions. This may partly account for the slightly lower levels of performance in the no-alcohol condition in comparison with data from students tested in the evening using a similar paradigm (Maylor & Badham, 2018), though as already mentioned, the additional prospective memory requirement here may also have contributed to this small difference. (Of course, it could be argued that the present context is more appropriate for applied research.) Second, we relied on self-reported alcohol consumption rather than measuring

blood alcohol concentrations. Nevertheless, there was a significant relationship between pints consumed and the associative memory deficit in the alcohol group, suggesting that self-reports were not entirely uninformative. Third, our participants were all University students and thus in view of evidence that adverse effects of alcohol are a function of baseline levels of performance (Maylor & Rabbitt, 1993), it would seem important to extend this study to investigate a wider range of both ages and abilities.

References

Acheson, S. K., Stein, R. M., & Swartzwelder, H. S. (1998). Impairment of semantic and

figural memory by acute ethanol: Age-dependent effects. *Alcoholism: Clinical and*

Experimental Research, *22*(7), 1437-1442.

Bender, A. R., Naveh-Benjamin, M., & Raz, N. (2010). Associative deficit in recognition

memory in a lifespan sample of healthy adults. *Psychology and Aging, 25,* 940-948.

Castel, A. D., & Craik, F. I. M. (2003). The effects of aging and divided attention on

memory for item and associative information. *Psychology and Aging, 18,* 873-885.

Chen, C. M., Dufour, M. C., & Yi, H.-y. (2004). Alcohol consumption among young adults

ages 18-24 in the United States: Results from the 2001-2002 NESARC

survey. *Alcohol Research & Health, 28*, 269-280.

Cohn, M., Emrich, S. M., & Moscovitch, M. (2008). Age-related deficits in associative

memory: The influence of impaired strategic retrieval. *Psychology and Aging, 23,* 93-

103.

Craik, F. I. M. (1977). Similarities between the effects of aging and alcoholic intoxication on

memory performance, construed with a "Levels of Processing" framework. In I. M.

Birnbaum & E. S. Parker (Eds.), *Alcohol and memory* (pp. 9-22). Hillsdale, N.J.:

Erlbaum.

Craik, F. I. M. (1983). On the transfer of information from temporary to permanent

memory. *Philosophical Transactions of the Royal Society of London, 302,* 341-359.

Duka, T., Weissenborn, R., & Dienes, Z. (2001). State-dependent effects of alcohol on

recollective experience, familiarity and awareness of memories.

Psychopharmacology, 153, 295-306.

Finn, P. R., Justus, A., Mazas, C., & Stienmetz, J. E. (1999). Working memory, executive processes and the effects of alcohol on Go/No-Go learning: Testing a model of behavioural regulation and impulsivity. *Psychopharmacology, 146,* 465-472.

George, S., Rogers, R. D., & Duka, T. (2005). The acute effect of alcohol on decision making in social drinkers. *Psychopharmacology*, *182*, 160-169.

Gilbert, J. G. (1941). Memory loss in senescence. *The Journal of Abnormal and Social Psychology, 36*, 73-86.

Haggård-Grann, U., Hallqvist, J., Långström, N., & Möller, J. (2006). The role of alcohol and drugs in triggering criminal violence: A case-crossover study. *Addiction, 101,* 100-108.

Hasher, L., & Zacks, R. T. (1979). Automatic and effortful processes in memory. *Journal of Experimental Psychology: General, 108*, 356-388.

Henke, K., Weber, B., Kneifel, S., Wieser, H. G., & Buck, A. (1999). Human hippocampus associates information in memory. *Proceedings of the National Academy of Sciences*, *96*, 5884-5889.

Hull, J. G., & Bond, C. F. Jr. (1986). Social and behavioral consequences of alcohol consumption and expectancy: A meta-analysis. *Psychological Bulletin, 99,* 347-360.

Jennison, K. M. (2004). The short-term effects and unintended long-term consequences of binge drinking in college: A 10-year follow-up study. *The American Journal of Drug and Alcohol Abuse*, *30*, 659-684.

Kessler, J., Irle, E., & Markowitsch, H. J. (1986). Korsakoff and alcoholic subjects are severely impaired in animal tasks of associative memory. *Neuropsychologia, 25*, 671-680.

Kirchner, T. R., & Sayette, M. A. (2003). Effects of alcohol on controlled and automatic memory processes. *Experimental and Clinical Psychopharmacology, 11,* 167-175.

Leitz, J. R., Morgan, C. J. A., Bisby, J. A., Rendell, P. G., & Curran, H. V. (2009). Global impairment of prospective memory following acute alcohol. *Psychopharmacology, 205,* 379-387.

Light, L. L., Prull, M. W., La Voie, D. J., & Healy, M. R. (2000). Dual-process theories of memory in old age. In T. J. Perfect & E. A. Maylor (Eds.), *Models of cognitive aging* (pp. 238-300). Oxford: Oxford University Press.

Lyvers, M. F., & Maltzman, I. (1991). Selective effects of alcohol on Wisconsin Card Sorting Test performance. *British Journal of Addiction*, *86*, 399-407.

Maylor, E. A., & Badham, S. P. (2018). Effects of time of day on age-related associative deficits. *Psychology and Aging, 33,* 7-16.

Maylor, E. A., Darby, R. J., Logie, R. H., Della Sala, S., & Smith, G. (2002). Prospective memory across the lifespan. In P. Graf & N. Ohta (Eds.), *Lifespan development of human memory* (pp. 235-256). Cambridge, Massachusetts: The MIT Press.

Maylor, E. A., & Rabbitt, P. M. A. (1987a). Effect of alcohol on rate of forgetting. *Psychopharmacology, 91,* 230-235.

Maylor, E. A., & Rabbitt, P. M. A. (1987b). Effects of alcohol and practice on choice reaction time. *Perception & Psychophysics, 42,* 465-475.

Maylor, E. A., & Rabbitt, P. M. A. (1988). Amount of practice and degree of attentional control have no influence on the adverse effect of alcohol in word categorization and visual search tasks. *Perception & Psychophysics, 44,* 117-126.

Maylor, E. A., & Rabbitt, P. M. A. (1993). Alcohol, reaction time and memory: A meta-analysis. *British Journal of Psychology, 84,* 301-317.

Maylor, E. A., Rabbitt, P. M. A., James, G. H., & Kerr, S. A. (1990a). Comparing the effects of alcohol and intelligence on text recall and recognition. *British Journal of Psychology, 81,* 299-313.

Maylor, E. A., Rabbitt, P. M. A., James, G. H., & Kerr, S. A. (1990b). Effects of alcohol and

extended practice on divided-attention performance. *Perception & Psychophysics, 48,* 445-452.

Maylor, E. A., Rabbitt, P. M. A., & Kingstone, A. F. (1987). Effects of alcohol on word

categorisation and recognition memory. *British Journal of Psychology, 78,* 233-239.

Naveh-Benjamin, M. (2000). Adult age differences in memory performance: Tests of an

associative deficit hypothesis. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 26*, 1170-1187.

Naveh-Benjamin, M., Hussain, Z., Guez, J., & Bar-On, M. (2003). Adult age differences in

episodic memory: Further support for an associative-deficit hypothesis. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 29*, 826-837.

Naveh-Benjamin, M., & Mayr, U. (2018). Age-related differences in associative memory:

Empirical evidence and theoretical perspectives. *Psychology and Aging, 33,* 1-6.

Naveh-Benjamin, M., Moscovitch, M., & Roediger III, H. L. (Eds.) (2001). *Perspectives on

human memory and cognitive aging: Essays in honor of Fergus Craik.* New York: Psychology Press.

Naveh-Benjamin, M., Shing, Y. L., Kilb, A., Werkle-Bergner, M., Lindenberger, U., & Li,

S-C. (2009). Adult age differences in memory for name-face associations: The effects of intentional and incidental learning. *Memory, 17*, 220-232.

Nilsson, L.-G., Bäckman, L., Karlsson, T. (1989). Priming and cued recall in elderly, alcohol

intoxicated and sleep deprived subjects: A case of functionally similar memory deficits. *Psychological Medicine, 19*, 423-433.

Old, S. R., & Naveh-Benjamin, M. (2008). Differential effects of age on item and associative

measures of memory: A meta-analysis. *Psychology and Aging, 23*, 104-118.

Oorsouw, K. V., & Merckelbach, H. (2012). The effects of alcohol on crime-related memories: A field study. *Applied Cognitive Psychology, 26,* 82-90.

Paraskevaides, T., Morgan, C. J. A., Leitz, J. R., Bisby, J. A., Rendell, P. G., & Curran, H. V. (2010). Drinking and future thinking: Acute effects of alcohol on prospective memory and future simulation. *Psychopharmacology*, *208*, 301-308.

Parker, E. S., Birnbaum, I. M., & Noble, E. P. (1976). Alcohol and memory: Storage and state dependency. *Journal of Verbal Learning and Verbal Behavior*, *15*, 691-702.

Petros, T. V., Kerbel, N., Beckwith, B. E., Sacks, G., & Sarafolean, M. (1985). The effects of alcohol on prose memory. *Physiology & Behavior*, *35*, 43-46.

Rabbitt, P. M. A., & Maylor, E. A. (1991). Investigating models of human performance. *British Journal of Psychology, 82,* 259-290.

Rotello, C. M., & Heit, E. (2000). Associative recognition: A case of recall-to-reject processing. *Memory & Cognition*, *28*, 907-922.

Shing, Y. L., Werkle-Bergner, M., Li, S-C., & Lindenberger, U. (2008). Associative and strategic components of episodic memory: A life-span dissociation. *Journal of Experimental Psychology: General, 137,* 495-513.

Smith, R. E. (2003). The cost of remembering to remember in event-based prospective memory: Investigating the capacity demands of delayed intention performance. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 29,* 347-361.

Sperling, R., Chua, E., Cocchiarella, A., Rand-Giovannetti, E., Poldrack, R., Schacter, D. L., & Albert, M. (2003). Putting names to faces: Successful encoding of associative memories activates the anterior hippocampal formation. *Neuroimage*, *20*, 1400-1410.

Stanislaw, H., & Todorov, N. (1999). Calculation of signal detection theory measures. *Behavior Research Methods, Instruments, & Computers*, *31*, 137-149.

Testa, M., Fillmore, M. T., Norris, J., Abbey, A., Curtin, J. J., Leonard, K. E., … Hayman,
L. W. Jr. (2006). Understanding alcohol expectancy effects: Revisiting the placebo
condition. *Alcoholism: Clinical and Experimental Research, 30,* 339-348.

Weissenborn, R., & Duka, T. (2003). Acute alcohol effects on cognitive function in social
drinkers: Their relationship to drinking habits. *Psychopharmacology*, *165*, 306-312.

White, A. M. (2003). What happened? Alcohol, memory blackouts, and the brain. *Alcohol
Research and Health*, *27*, 186-196.

White, A. M., Matthews, D. B., & Best, P. J. (2000). Ethanol, memory, and hippocampal
function: A review of recent findings. *Hippocampus, 10,* 88-93.

Wicki, M., Kuntsche, E., & Gmel, G. (2010). Drinking at European universities? A review
of students' alcohol use. *Addictive Behaviors*, *35*, 913-924.

Yonelinas, A. P. (2002). The nature of recollection and familiarity: A review of 30 years of
research. *Journal of Memory and Language, 46*, 441-517.

Yuille, J. C., & Tollestrup, P. A. (1990). Some effects of alcohol on eyewitness memory.
Journal of Applied Psychology, 75, 268-273.

Footnotes

[1]Although exact time of testing was not recorded, those in the alcohol group were probably tested slightly later in the evening than those in the no-alcohol group. However, this was not considered to be a significant potential confound because effects of time of testing on memory in young adults between much more extreme comparisons (morning vs. afternoon/evening) are largely absent in studies that do not also take individual differences in morningness-eveningness preferences into account (see Maylor & Badham, 2018, for discussion).

[2]All the data patterns were qualitatively similar with and without this adjustment for age.

[3]There was no correlation between age and the number of pints consumed, $r = .059$, $p = .747$, and therefore age was not partialled out.

Table 1

Means (and 95% Confidence Intervals) for Hit and False Alarm Rates (%) as a Function of

Type of Memory (Item Vs. Associative) and Condition (No Alcohol Vs. Alcohol)

	Condition			
	No Alcohol		Alcohol	
Memory	Hits	False Alarms	Hits	False Alarms
Item				
	73.1 (67.8–78.4)	17.0 (11.3–22.7)	66.7 (61.4–72.0)	21.1 (15.4–26.8)
Associative				
	72.5 (65.2–79.8)	21.9 (14.2–29.5)	58.8 (51.4–66.1)	38.4 (30.8–46.1)

Table 2

Means (and 95% Confidence Intervals) for Signal Detection Measures (D-Prime and C) as a

Function of Type of Memory (Item Vs. Associative) and Condition (No Alcohol Vs. Alcohol)

	Condition			
	No Alcohol		Alcohol	
Memory	*d'*	*c*	*d'*	*c*
Item				
	1.81 (1.53–2.08)	0.21 (0.06–0.36)	1.39 (1.12–1.67)	0.24 (0.09–0.39)
Associative				
	1.57 (1.19–1.94)	0.10 (-0.04–0.23)	0.58 (0.21–0.96)	0.05 (-0.08–0.19)

Figure 1. Recognition performance as percentages of hits minus false alarms for item and

associative memory as a function of condition (no alcohol and alcohol). Error bars represent
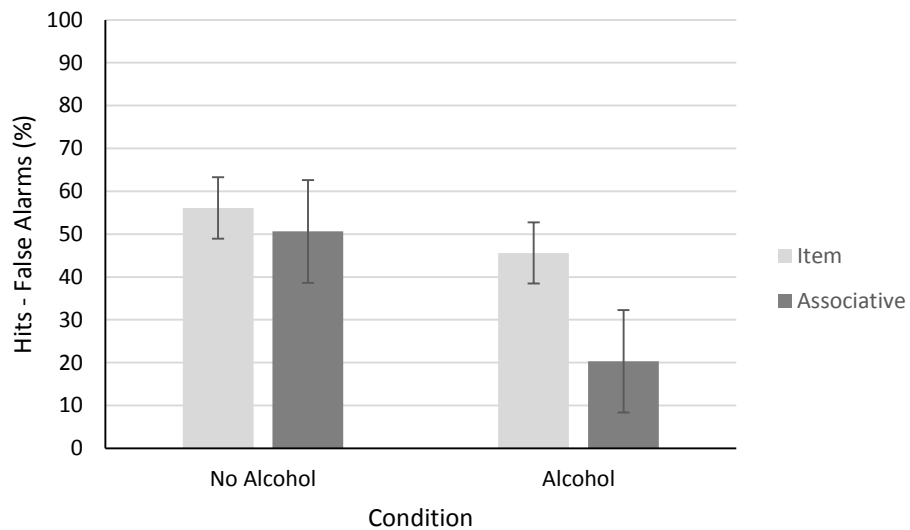
95% confidence intervals.

Figure 2. Scatterplots (with best-fitting linear functions) of percentages of hits minus false alarms for item memory (top panel), associative memory (middle panel) and item minus associative memory (bottom panel) plotted against the number of pints of alcohol consumed (based on self-report) by those in the alcohol condition (*n* = 32).