ELSEVIER

Physiology & Behavior 85 (2005) 240 – 245

PHYSIOLOGY & BEHAVIOR

Alcohol-induced impairment and enhancement of memory: A test of the interference theory

Patricia L. Moulton[a,*], Thomas V. Petros[b], Kathyrn J. Apostal[b], Ronald V. Park II[b], Elizabeth A. Ronning[b], Brent M. King[b], James G. Penland[c]

[a]*School of Medicine and Health Sciences, University of North Dakota, 501 North Columbia, P.O. Box 9037, Grand Forks, ND 58202-9037, USA*
[b]*Psychology Department, University of North Dakota, Corwin-Larimore Hall, Box 8380 Grand Forks, ND 85202-8380, USA*
[c]*U.S.D.A. A.R.S. Grand Forks Human Nutrition Research Center, USA*

Received 30 July 2003; received in revised form 3 March 2005; accepted 8 March 2005

Abstract

Many studies have found cognitive deficits related to alcohol consumption. However, few studies have studied cognitive performance when alcohol was administered after the to-be-remembered information was presented with memory testing occurring when participants are once again sober. The present study examined effects of alcohol on cognitive performance using a prose recall task during acute intoxication and a post-trial recall task for prose passages that had been presented before intoxication. Fifty-one men were given either 2.0 g/kg of 100 proof (50% absolute ethanol) vodka or a placebo. In the present study, evidence was found of acute alcohol impairment in prose memory, along with alcohol facilitation of memory on a post-trial task.

Keywords: Alcohol; Prose memory; Memory facilitation; Memory interference

1. Alcohol-induced impairment and enhancement of memory

It has been well established that acute intoxication with ethanol results in memory impairment. Parker et al. [1] found that the number of words recalled and organization of materials decreased with increasing doses of alcohol. Subsequent work reported that intoxication with alcohol disrupted encoding processes [2] but that retrieval of memory is not impaired by intoxication with alcohol [3]. In these studies, subjects encoded new information when they were sober and retrieved the information while they were either sober or intoxicated. The results of both studies indicated that alcohol did not influence retrieval when the information was encoded when the subjects were sober.

Studies using prose passages also indicated that acute intoxication with alcohol impaired memory performance [4]. Forty male participants were administered either 1.0 g/kg of body weight of 80 proof vodka (40% absolute ethanol) or a placebo. Participants were asked to listen to and immediately recall narrative passages presented at 120, 160 or 200 words per minute (wpm). Both sober and intoxicated subjects favored the main ideas of the passage in their recalls relative to the non-essential details. Alcohol-induced memory impairment was similar for text propositions at all levels of importance when the passages were presented at a slow (120 wpm) or medium rate (160 wpm). However, when passages were presented at a fast rate (200 wpm), the largest effect of alcohol was found for the most important ideas in the passages. These results suggested that alcohol-induced deficits in prose memory might result from a general slowing in the rate at which text is encoded into working memory. Additional work has demonstrated that intoxicated participants read slower and recall significantly less from prose passages than sober participants [5,6].

Animal research has found evidence of facilitation of memory when alcohol is administered after to be recalled

* Corresponding author. Tel.: +1 701 777 6781; fax: +1 701 777 6779.
E-mail address: pmoulton@medicine.nodak.edu (P.L. Moulton).

0031-9384/$ - see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.physbeh.2005.03.011

material is presented and then later tested. Alkana and Parker [7] found facilitation for memory of a passive avoidance task in mice administered 0.75–4.5 g/kg of ethanol after training. Ladner et al. [8] found that mice that had previously learned the location of a piece of cheese and then received an intraperitoneal injection of 2.0 g/kg of ethanol had an increased latency to eat the cheese suggesting that the cheese was considered aversive. Prediger and Takahashi [9] found that rats administered a low dose of alcohol (0.5 and 1.0 g/kg) after exposure to juvenile rats showed enhanced social recognition of juvenile rats, a test of short-term memory. These researchers suggest that this facilitation in memory may be due to activation of the opioid system because when rats were administered an opioid antagonist (naloxone) this facilitation was lost.

Investigations of the impact of alcohol on memory in humans [10–12] have also found facilitation of memory performance for information learned prior to intoxication if the participants were tested several hours after intoxication when their blood alcohol levels (BAL) was 0. Parker et al. [10] administered 1 g/kg of vodka to 16 male volunteers resulting in a mean BAL of 0.08 g/100 ml. Prior to receiving alcohol, participants had studied 10 scenic slides. Participants were then tested on recognition of the slides 3 h later. Participants correctly recognized significantly more pictures than during a non-alcohol baseline session, which took place the morning of the same day as the alcohol session. In a second experiment, Parker et al. [10] tested 72 male volunteers on incidental memory for a list of 30 words in five categories. Participants were asked to sort the words into the categories and were then administered 1 g/kg of vodka or a placebo. Participants were asked to freely recall the words the next day. Participants who had received alcohol recalled significantly more words and categories than the placebo participants.

Lamberty et al. [11] presented either word lists of 28 unrelated nouns or three narrative prose passages to participants prior to treatment with either 1.0 g/kg of 80 proof (40% absolute ethanol) vodka or a placebo. Participants were tested on recall and recognition of the word lists or recall of the prose passages 24 h after drinking. Lamberty et al. found no effect of treatment on recall or recognition of the word lists. However, for the prose recall task, alcohol participants produced higher recall scores than placebo subjects. The authors hypothesized that the alcohol facilitation of memory may have been due to a disruption of the participant's ability to form new memories, and thus resulting in potential reduction of post-encoding interference.

Mueller et al. [12] presented direct support for an interference account of alcohol facilitation of recall. Mueller et al. tested participants in a 2-day, double-blind experiment. Participants reported to the lab between 3:30 and 6:00 p.m. after a 3-h fast. Each participant was visually presented with two, 24-word lists and recalled each word list immediately after each list was presented. After recall of the first two lists, participants were administered 2.2 g/kg of 90 proof (45% absolute ethanol) vodka mixed with grapefruit juice (3:1 ratio) or a placebo of grapefruit juice. Participants were given 20 min to finish their drink followed by a 20-min absorption period. After the absorption period, participants were presented with two additional 24-word lists and immediate recall was obtained followed by a recall the next day. The average BAL was 0.065% after the absorption period and 0.078% after recall of the fourth list. Recall of lists presented immediately after administration of alcohol was significantly impaired. Recall obtained on the second day of the experiment indicated that recall of words presented prior to the administration of alcohol was significantly higher than recall of words presented after administration of alcohol. The authors argued that alcohol-enhanced memory performance occurs because alcohol impairs the acquisition of new, interfering memories.

The concept of interference as an explanation of retrograde facilitation was originally forwarded by Parker and Weingartner [13]. They argued that facilitation of memory might be the result of a reduction in cognitive processes after alcohol administration: that is alcohol suppresses cognitive activity, which interferes with the formation of new memories. Tyson and Schirmuly [14] tested whether alcohol-induced memory facilitation of a word list was due to consolidation or interference. Participants were given either 0.8 g/kg of 40% alcohol or a placebo immediately after, or 40 min after, presentation of a 25-word list. The peak BAL occurred at 40 min after administration of the alcohol with a mean of 0.055 g/100 ml. In addition, participants were given an incidental learning task at 2 h after drinking consisting of a set of 10 pictures with associated words. Four hours after drinking, subjects were tested on recall and recognition. The BAL at this stage was not reported. The participants who received alcohol recalled significantly more words than the placebo group. Alcohol facilitated memory for both the immediate and 40-min conditions, thus not supporting a consolidation hypothesis. The alcohol-treated participants did, however, have impaired performance on the incidental-learning task, supporting the interference hypothesis.

Parker et al. [15] using the same task as experiments described earlier [10] (recognition of scenic slides) reduced the potential for interference during the 7-h period from administration of alcohol to post-trial testing by limiting cognitive and memory testing. They found memory facilitation at doses of .5 and 1.0 g/kg of alcohol. These researchers cited this as evidence for the consolidation theory of alcohol facilitation of memory.

Esposito et al. [16] postulated that alcohol enhanced the consolidation of memories encoded prior to consumption of alcohol, specifically that these effects involve activation of dopaminergic–enkephalinergic neuronal pathways thought to be involved in memory consolidation. Esposito et al. theorized that this activation induced a hedonic state and

facilitated memory for events that occurred immediately before the state change.

The purpose of the present study was to expand previous work [11,14] by examining both alcohol-induced impairment and alcohol-induced facilitation of prose memory. The present study examined whether participants would exhibit both, memory impairment during intoxication on an immediate prose recall task, and memory facilitation after intoxication of the prose material presented prior to drinking. Lamberty et al. [11] argued that alcohol facilitation of memory performance resulted from reduction of interference, yet provided no direct test of reduction of post-encoding interference. In the present study, a prose recall task was given after administration of alcohol in order to provide a direct measurement of interference. In fact, Lamberty et al. [11] dismissed their placebo participants once drinking had been completed and no restrictions were placed on the activities of the intoxicated participants other than that they remain in the lab until they were no longer intoxicated. Therefore, no direct measure of post-drinking encoding performance was obtained from the Lamberty et al. study. In the present study, intoxicated and sober participants remained in the lab for the same amount of time. Measures of prose memory were obtained immediately after participants finished drinking to document impaired text memory and demonstrate reduced interference leading to facilitation in memory for passages presented before drinking.

2. Methods

2.1. Participants

Male participants between 21 and 39 years of age (M=22.43 years, S.D.=2.31) were recruited from the University of North Dakota campus, as well as from the surrounding community. A health screening and drinking history were obtained prior to participation. Participants without chronic illness, who were not taking prescription medication, who were moderate social drinkers and who had never been treated for alcoholism, were invited to participate in the study. Moderate social drinkers were defined as those participants who reported that they drank alcoholic beverages at least once per week and consumed at least two drinks each time. The study included 51 male participants with 24 randomly assigned to the alcohol group and 27 randomly assigned to the placebo group. Some attrition resulted from participants who reported to the lab with a BAL above 0 or had a blood pressure that exceeded 140/90. This attrition led to an uneven number of participants in each group. The Institutional Review Board at the University of North Dakota approved this study and informed consent was obtained from each participant. Participants were given either class credit or $25 for their participation.

2.2. Materials

A digital blood pressure cuff was used to assess blood pressure and heart rate. An Intoximeter IV breath alcohol meter (Intoximeter, Inc., Saint Louis, MO) was used to assess BAL.

Participants were given the Wahler Physical Symptom Inventory [17], an inventory of 42 common physical symptoms. Participants were asked to rate how often each symptom bothered them. Participants also completed the Khavari Alcohol Test [18], providing information about the frequency and amount of their consumption of beer, wine and other liquors. The score produced by the Khavari is the annual consumption of absolute alcohol (AAAI).

The vocabulary, digit span forward, digit span backward, digit symbol and letter–number subtests of the WAIS-III [19] were also administered. The vocabulary subtest includes 33 words of increasing difficulty. The digit span forward subtest requires participants to repeat a sequence of orally presented digits in the same order in which they were presented. The digit span backward subtest requires participants to repeat a sequence of digits in reverse order from which they were presented. The digit symbol subtest consists of a list of numbers paired with symbols and requires participants to match as many symbols with numbers as possible in 90 s. The letter–number subtest requires participants to recall a sequence of letters and digits by first stating the numbers in numerical order and then stating the letters in alphabetical order.

The prose passages consisted of three expository and three narrative stories each containing 200–220 words and rated at seventh to eighth grade readability [20]. The passages were previously divided into idea units rated for importance (low, medium or high) [21], with 25–26 idea units for expository passages and 29–34 idea units for narrative passages.

2.3. Procedure

Participants were instructed to abstain from drinking alcohol and using over-the-counter medication such as

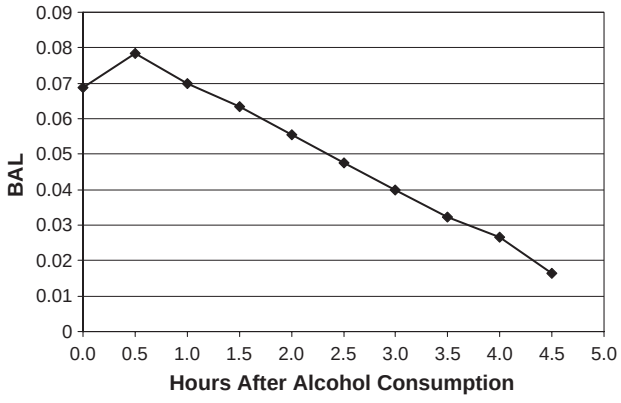

Fig. 1. BAL dose response curve.

Table 1
WAIS sub-tests: scale means and *T*-values as a function of group

Variable	Alcohol	Placebo	*t*	Probabilities
Vocabulary	10.63 (2.28)	11.19 (2.39)	− 0.85	0.40
Digit span	10.71 (2.95)	10.89 (3.13)	− 0.21	0.83
Letter–number	11.13 (3.01)	10.81 (2.68)	0.39	0.70
Digit symbol	7.83 (1.81)	8.70 (2.61)	− 1.37	0.18

Parentheses indicate standard deviation.

aspirin for the 24 h preceding the study. Participants were also asked to eat breakfast before reporting to the lab. All sessions were conducted between 9 a.m. and 5 p.m. Participants were brought into the lab between 9 a.m. and 10 a.m. After giving signed informed consent, each participant's blood pressure, heart rate, height, weight and baseline BAL were measured. Any subject with a baseline BAL other than 0 was dismissed from the study.

Participants were presented with a tape-recorded practice prose passage and asked to listen carefully and remember as much as possible. As soon as the passage was completed, participants were asked to orally recall the passage into a tape recorder. Participants then listened to the two prose passages (one narrative, one expository) that would serve as the stimuli for the post-trial prose recall task, 25 min prior to drinking ethanol. Participants were instructed that they would need to recall these stories at the end of the study. After this, participants were administered the WAIS-III vocabulary subtest, the Wahler Physical Symptom Inventory and the Khavari Alcohol Test.

Participants were then randomly assigned to either the alcohol or placebo condition. Participants in the alcohol condition were given 2.0 g/kg of body weight of 100 proof vodka (50% absolute ethanol) in a solution of one part vodka to two parts orange juice. Participants in the placebo condition were given the same amount of liquid as the alcohol group, consisting only of orange juice. One gram of vodka was used to swab the rims of the glasses in the placebo condition in order to produce an alcohol smell. The beverage was divided into three glasses and the participants were given one glass every 20 min for a total of 1 h of drinking time. The participants then rinsed their mouths out with water. Ten minutes after the drinking period was completed, the first BAL was taken.

Immediately after the first BAL reading, the immediate prose recall task was administered. Participants were asked to read one practice and four experimental passages (two narrative, two expository) at their own pace using a personal computer. The computer presented the passages one idea unit at a time, each time the return key was pressed. Participants controlled the return key and thus the rate of presentation. Immediately after reading each passage, participants orally recalled the passage into a tape recorder.

After all passages were recalled, participants were allowed to read or watch movies, but could not leave the premises, for a 4-h period. A BAL reading was taken every 30 min during this 4-h period. Four hours after the first BAL reading, participants were given the digit span forward, digit span backward, digit symbol and letter–number subtests of the WAIS-III.

After the subtests from the WAIS-III were completed (4.5 h after the first BAL), the post-trial prose recall task was administered. Participants were asked to recall the two prose passages that they had listened to before drinking. Participants were cued with a descriptive title of the story to be remembered.

3. Results

The average age of participants in the alcohol group was 22.50 (S.D.=2.32) years and in the placebo group 22.37 (S.D.=2.31) years. A series of *t*-tests revealed no significant differences between groups on age, height, weight, systolic and diastolic blood pressure, heart rate and overall health as measured by the Wahler Physical Symptoms Inventory. The Khaveri Alcohol Test was used to determine whether there were differences between groups on prior alcohol consumption. No significant differences were found. According to the Khavari Test scores, the alcohol group had an average annual absolute alcohol intake (AAAI) of 380.78 (S.D.=358.84) and the placebo group had an average AAAI of 436.81 (S.D.=390.98). Khavari and Farber [18] found an average AAAI for college students of 312 and an average AAAI for two groups of alcoholics of 2592 and 3163.

For participants receiving alcohol, the average BAL for the first reading (immediately after participants finished drinking) was 0.07 (S.D.=0.02) and the average BAL 30 min later was 0.08 (S.D.=0.02). The average BAL 4 h later was 0.03 (S.D.=0.01) and at the completion of all testing 0.02 (S.D.=0.01) (see Fig. 1).

A *t*-test determined that there were no significant differences between groups on the vocabulary subtest of the WAIS-III. The alcohol group had an average scale score of 10.63 (S.D.=2.28) and the placebo group had an average

Table 2
Immediate prose recall task: mean proportion of idea units recalled as a function of treatment group, importance level and passage type

Type	Narrative			Expository		
Importance	High	Medium	Low	High	Medium	Low
Alcohol	0.84 (0.13)	0.63 (0.17)	0.48 (0.17)	0.57 (0.16)	0.46 (0.15)	0.29 (0.16)
Placebo	0.89 (0.08)	0.77 (0.11)	0.56 (0.15)	0.67 (0.13)	0.60 (0.15)	0.42 (0.13)

Parentheses indicate standard deviations.

Table 3
Post-trial prose recall task: mean proportion of idea units recalled as a function of treatment group, importance level and passage type

Type	Narrative			Expository		
Importance	High	Medium	Low	High	Medium	Low
Alcohol	0.93 (0.08)	0.64 (0.15)	0.36 (0.14)	0.51 (0.23)	0.57 (0.27)	0.44 (0.29)
Placebo	0.90 (0.21)	0.51 (0.17)	0.27 (0.16)	0.55 (0.23)	0.50 (0.20)	0.37 (0.20)

Parentheses indicate standard deviations.

scale score of 11.19 (S.D.=2.39). Several cognitive tests were given 4 h after drinking ended. No significant differences were found on t-tests for the digit span, letter–number or digit symbol subtests of the WAIS-III. Mean scaled scores and independent *t*-test scores for these variables are presented in Table 1.

A transcript of each prose passage recall was scored by a rater who was blind to treatment group. A subset (20%) of the recall transcripts was independently scored by a second rater who was also blind to treatment group. The percent agreement for the two raters ranged from 80% to 91% with a mean inter-rater reliability of 87%. Memory for each passage was expressed as the proportion of idea units recalled at each of three levels of importance (low, medium and high).

The mean recall as a function of treatment group, importance level and passage type for the prose recall task given during intoxication (immediate prose recall task) is presented in Table 2. A 2 (group)×2 (passage type)×3 (importance level) mixed ANOVA revealed significant main effects of group, $F(1,42)=12.08$, $p<0.001$, and passage type, $F(1.42)=112.94$, $p<0.001$, which indicated that alcohol participants recalled fewer idea units than placebo participants ($M=0.55$ vs. $M=0.65$) and recall of narrative passages ($M=0.70$) was significantly higher than recall of expository passages ($M=0.50$). A significant main effect of importance level was observed, $F(2,84)=253.13$, $p<0.001$, indicating that recall of idea units increased as a function of their importance to the overall story. A significant interaction of passage type×importance level was also observed, $F(2,84)=4.48$, $p<0.014$. A subsequent Tukey analysis of the interaction indicated that the superior recall of narrative passages compared to expository passages was greatest for the idea units of low importance.

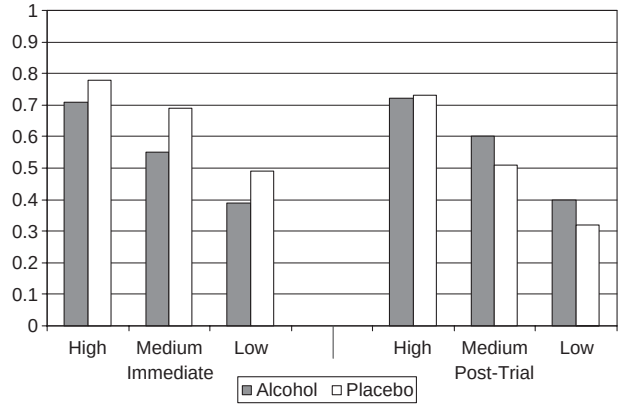

Fig. 2. Immediate and post-trial prose recall: treatment group×importance level×task.

The proportion of idea units recalled as a function of group and level of importance for the prose recall test given on material learned prior to drinking is presented in Table 3. A 2 (group)×2 (passage type)×3 (importance level) mixed ANOVA revealed significant main effects of passage type, $F(1,41)=12.21$, $p<0.001$, and importance level, $F(2,41)=183.63$, $p<0.000$. Recall of narrative passages ($M=0.60$) was significantly higher than recall of expository passages ($M=0.49$) and recall of high importance idea units ($M=0.72$) was significantly better than recall of medium importance ($M=0.55$) and low importance ($M=0.36$) idea units. A significant interaction of importance level×group was observed, $F(2,82)=3.70$, $p=0.03$. Subsequent Tukey analysis indicated that the participants who received alcohol had significantly greater recall of idea units of low and medium importance relative to placebo participants, with no group difference in recall observed for high importance idea units. A passage type×importance level interaction was also found $F(2,82)=55.20$, $p<0.000$, with high importance idea units in the narrative passage ($M=0.92$) having the greatest recall.

Fig. 2 displays the proportion of idea units recalled as a function of importance level and condition. As is apparent in the graph, alcohol resulted in impaired performance on all types of idea units (high, medium and low importance levels) during the prose recall task given after drinking.

For the prose passages learned prior to intoxication and recalled 4 h after intoxication (post-trial), the alcohol group recalled approximately the same proportion of idea units as the placebo group on the high importance level idea units. However, the alcohol group exhibited greater recall of medium and low importance level idea units than did the placebo group.

4. Discussion

The findings of the present study demonstrated that acute intoxication with alcohol resulted in impaired prose recall. This finding is consistent with previous work in which participants read passages at their own rate [5,6] or listened to recorded passages at several rates of presentation [4]. In addition, the present study found facilitation of recall for passages that had been presented immediately prior to intoxication with alcohol. This finding is consistent with Lamberty et al. [11]. The present study demonstrated both

impairment under acute intoxication and post-intoxication facilitation of memory. In particular, for the low importance idea-units in the narrative passages, alcohol participants had reduced recall during the immediate prose recall task and had significantly increased recall on the post-trial task. The recall facilitation was observed even though average BAL was above 0 ($M=0.03$), a limitation of the present study. Future research may find a greater facilitation of memory on a prose memory task if participants are tested when BAL has reached 0.

Improved recall was observed for the idea units of low and medium importance, presumably the most difficult information to remember. Perhaps future work in this area should explore whether post-trial alcohol facilitation of memory varies as a function of task-difficulty. This may help delineate the degree to which post-trial facilitation is a general effect or unique to the information-processing demands of the task. In addition, manipulation of the time interval would serve as a test of the consolidation theory. Previous researchers [12,14] have suggested that this facilitation is the result of reduced interference. Mueller et al. [12] explained that reduced interference results in a decrease in the number of memories stored during intoxication, which in turn results in fewer memories to potentially interfere with recall when sober. The recall decrement we observed along with the recall facilitation is consistent with the reduced interference explanation and adds to the evidence for the reduced interference hypothesis.

Post-trial facilitation has also been found with nicotine [22] and caffeine [23]. Alcohol, nicotine and caffeine all result either directly or indirectly in an increase in dopamine [24,25]. This increase in dopamine may lead to a hedonic state supporting a physiological explanation of post-trial facilitation [16]. Other studies have found post-trial facilitation with benzodiazepines [26], which along with alcohol augment GABA-mediated synaptic transmission [25]. Future research is needed to more clearly determine the physiological mechanism of action for post-trial prose recall facilitation.

References

[1] Parker E, Alkana R, Birnbaum I, Hartley J, Noble E. Alcohol and the disruption of cognitive processes. Arch Gen Psychiatry 1974;31:824–8.

[2] Parker E, Birnbaum I, Noble E. Alcohol and memory: storage and state-dependency. J Verbal Learn Verbal Behav 1976;15:691–702.

[3] Birnbaum I, Parker E, Hartley J, Noble E. Alcohol and memory: retrieval processes. Verbal Learn Verbal Behav 1978;17:325–35.

[4] Petros T, Kerbel N, Beckwith B, Sacks G, Sarafolean M. The effects of alcohol on prose memory. Physiol Behav 1985;35:43–6.

[5] Haut J, Beckwith B, Petros T. Acute ethanol intoxication, gender differences and prose processingPaper presented at the American Psychological Association Annual Meeting, Boston, MA.

[6] Sharp R, Beckwith B, Petros T. Gender differences in the influence of ethanol on prose memoryPaper presented at the Society for Neuroscience, New Orleans, LA.

[7] Alkana R, Parker E. Memory facilitation by post-training injection of ethanol. Psychopharmacology 1979;66:117–9.

[8] Ladner C, Babbini M, Davies D, Parker E, Alkana R. Effects of posttraining ethanol on an appetitive task. Neurobiol Learn Mem 2001;75:111–20.

[9] Prediger R, Takahashi R. Ethanol improves short-term social memory in rats. Involvement of opioid and muscarinic receptors. Eur J Pharmacol 2003;462:115–23.

[10] Parker E, Birnbaum H, Weingartner H, Hartley J, Stillman R, Wyatt R. Retrograde enhancement of human memory with alcohol. Psychopharmacology 1980;69:219–22.

[11] Lamberty G, Beckwith B, Petros T. Post-trial treatment with alcohol enhances recall of prose narratives. Physiol Behav 1990;48:653–8.

[12] Mueller C, Lisman S, Spear N. Alcohol enhancement of human memory: tests of consolidation and interference hypotheses. Psychopharmacology 1983;80:226–30.

[13] Parker E, Weingartner H. Retrograde facilitation of human memory by drugs. In: Weingartner H, Parker E, editors. Memory consolidation: psychobiology of cognition. Hillsdale, NJ: Lawrence Erlbaum Associates; 1984.

[14] Tyson P, Schirmuly M. Memory enhancement after drinking ethanol: consolidation, interference or response bias? Physiol Behav 1994;56:933–7.

[15] Parker E, Morihisa J, Wyatt R, Schwartz B, Weingartner H, Stillman R. The alcohol facilitation effect on memory: a dose–response study. Psychopharmacology 1981;74:88–92.

[16] Esposito R, Parker E, Weingartner H. Enkephalinergic–dopaminergic "reward" pathways: a critical substrate for the stimulatory, euphoric and memory-enhancing actions of alcohol. A hypothesis. Subst Alcohol Actions/Misuse 1984;5:111–9.

[17] Wahler H, Wahler J. Physical symptoms inventory. Los Angeles, CA: Western Psychological Services; 1983.

[18] Khaveri KA, Farber PD. A profile instrument for the quantification and assessment of alcohol consumption: the Khaveri alcohol test. J Stud Alcohol 1978;9:1525–38.

[19] Wechsler D. Wechsler adult intelligence, scale III. New York: The Psychological Corporation; 1997.

[20] Dale E, Chall JS. A formula for predicting readability. Educ Res Bull 1948;27:37–54.

[21] Petros T, Bentz B, Hammes K, Zehr H. The components of text that influence reading times and recall in skilled and less skilled college readers. Discourse Process 1990;13:387–400.

[22] Rusted J, Warburton D. Facilitation of memory by post-trial administration of nicotine: evidence for an attentional explanation. Psychopharmacology 1992;108:452–5.

[23] Brouse J, Petros T, Beckwith B, Water W. Pre-trial and post-trial effects of caffeine on memory. Poster presented at the Midwestern Psychological Association Annual Meeting, Chicago, IL.

[24] Carlson N. Physiology of behavior. Boston, MA: Allyn & Bacon; 1998.

[25] Julien R. A primer of drug action. New York: W.H. Freeman and Company; 1998.

[26] Fillmore M, Kelly T, Rush C, Hays L. Retrograde facilitation of memory by triazolam: effects on automatic processes. Psychopharmacology 2001;158:314–21.