

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

185,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Binge Drinking and Memory in Adolescents and Young Adults

Concepción Vinader-Caerols and Santiago Monleón

Abstract

The binge drinking (BD) pattern of alcohol consumption, characterized by intermittent consumption of large quantities of alcohol in short periods, is currently prevalent during adolescence and early youth. This period is characterized by critical changes to the structural and functional development of brain areas related with memory, as well as other executive functions. As a result, BD has been associated with undermined learning and memory ability in adolescents and youths of both sexes. One distinctive contribution of this chapter is to evaluate, together, the impact of an acute BD episode, the sample's history of consumption, and its effect on learning and memory performance and as potential gender differences. The main findings of the published research show that BD has differential effects on several types of memory and confirm that women are more vulnerable to these detrimental effects of alcohol than are men. These cognitive differences between men and women seem to be overridden as the blood alcohol concentration progressively increases. As BD pattern of consumption has been associated with inhibitory control deficits, future research also should investigate long-term implementation of inhibitory control training, emphasizing the importance of this training as part of the intervention strategies focused on this at-risk group.

Keywords: binge drinking, memory, adolescence, youth, gender

1. Introduction

Alcohol is one of the most widely consumed psychoactive substances in the world, especially among adolescents and young adults [1, 2]. Many of these develop a pattern of alcohol consumption known as binge drinking (BD). BD has been defined by The National Institute on Alcohol Abuse and Alcoholism (NIAAA) as a pattern of drinking that elevates a person's blood alcohol concentration (BAC) to 0.8 g/L or above [3]. This pattern involves the intake of large quantities of alcohol in a short period (about 2 h), followed by a period of abstinence, with a variability between 1 week and 1 month (see **Figure 1**). BD is the most common pattern of alcohol use among adolescents and young adults in Western countries. In Spain, the prevalence of BD pattern is similar in both sexes among 14–16 year-old adolescents and is more widespread among men than women in the age range of 17–18 years [4].

Individuals engaging in frequent BD have an increased risk to develop an alcohol use disorder (AUD) later in life. This risk has been suggested to be linked to

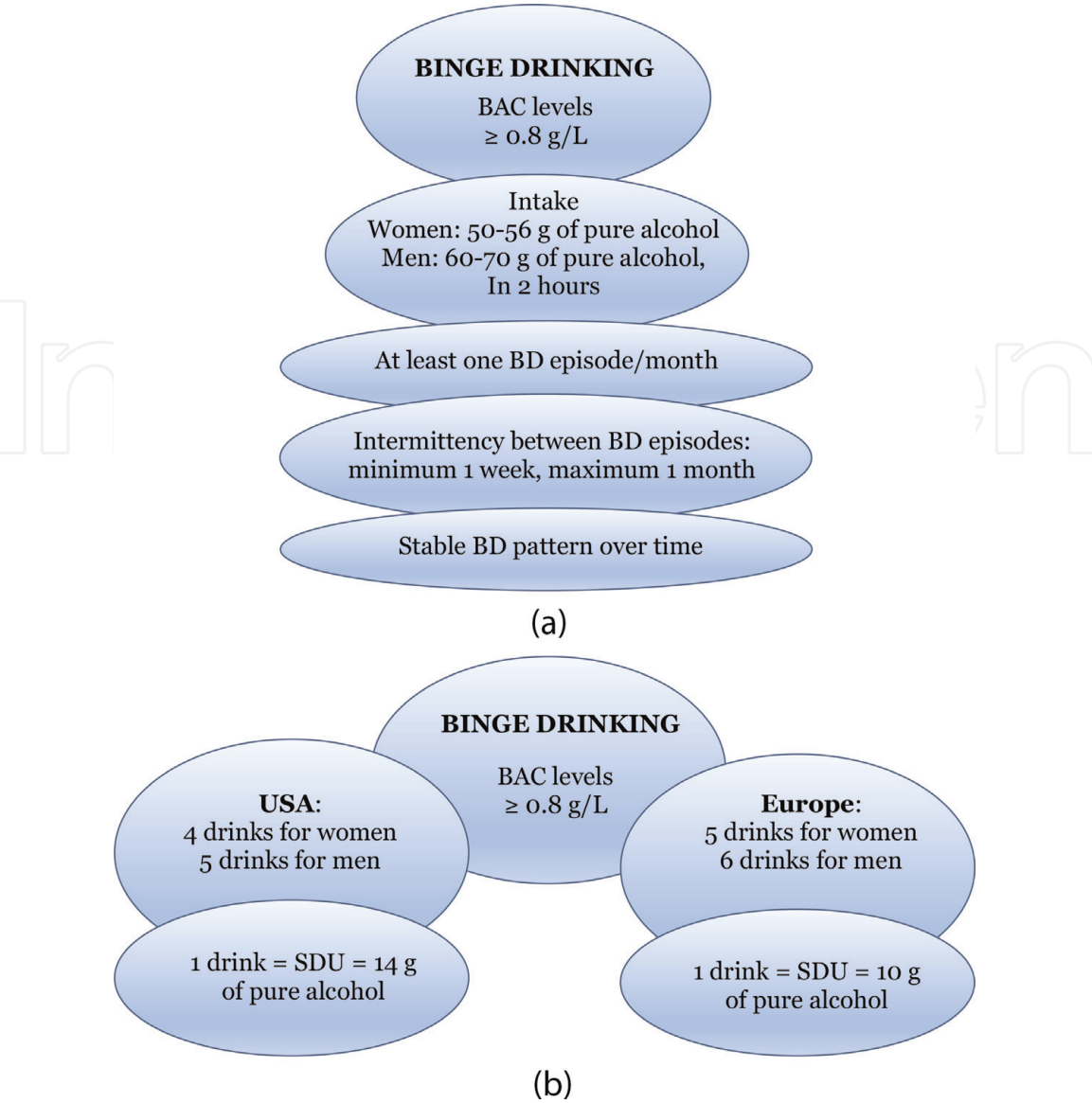


Figure 1.
(a) Binge drinking pattern criteria. Quantity: intake of 50–56 g of pure alcohol in women and 60–70 g in men, in 2 h. Frequency: at least one BD episode per month. Intermittency: abstinence between BD episodes over time (minimum 1 week, maximum 1 month). (b) Number of drinks (1 drink = SDU, standard drink unit) in the USA and Europe for binge drinking’s BAC levels.

executive deficits (e.g., [5]). The BD pattern of consumption seems to be especially associated with increased impulsivity and inhibitory control deficits (e.g., [6–8]). At the same time, this seems to be due to an attenuated frontal activation (e.g., [8, 9]). Thus, a higher incidence of BD has been related to decreased activation of dorsolateral prefrontal cortex, dorsomedial prefrontal cortex, and anterior cingulate cortex, brain regions strongly implicated in executive functioning [9]. The neurotoxic effects of BD on these regions can be less evident throughout adolescence, but if this alcohol consumption pattern persists, the executive dysfunction could be exacerbated. While individuals with AUD typically exhibit inhibitory control dysfunction, evidence of impaired inhibitory control among binge drinkers, who are at increased risk of developing an AUD, is mixed. Despite the variability in the literature, some findings point to mechanisms that may confer vulnerability for transition from binge drinking to AUD [6]. Therefore, inhibitory control deficits must be considered as an important factor that contributes to alcohol abuse.

On the other hand, important physical, social, and cognitive skills are acquired during adolescence and early youth. This period is also characterized by critical changes to the structural and functional development of brain areas related with

these skills [10]. For example, the superior associative cortex (e.g., prefrontal cortex) undergoes myelination, pruning, and synaptic reorganization [11, 12], among other alterations. Significant changes in the volume and shape of the hippocampal complex, a brain region that plays an important role in memory functions, are also observed during this developmental period [13–15].

Due to this plasticity, the adolescent brain seems to be especially vulnerable to the neurotoxic effects of alcohol. In fact, alcohol-related performance deficits in tasks assessing cognitive processes, such as attention, memory, and executive functions, in the not-yet-adult brain are more evident during adolescence [16, 17] and become more pronounced with a BD pattern of consumption [12, 18].

The intermittence between BD episodes seems to be the most important factor involved, as the repeated alternation between intoxication and withdrawal is particularly deleterious for the brain, due to the excitotoxic cell death it provokes [19, 20]. Thus, it has been demonstrated that BD episodes can be more harmful for the brain than an equivalent amount of alcohol without withdrawal episodes [20, 21].

Therefore, the BD adolescent population constitutes a cohort at risk of brain damage, and any disruptive effects of alcohol on learning and memory abilities in

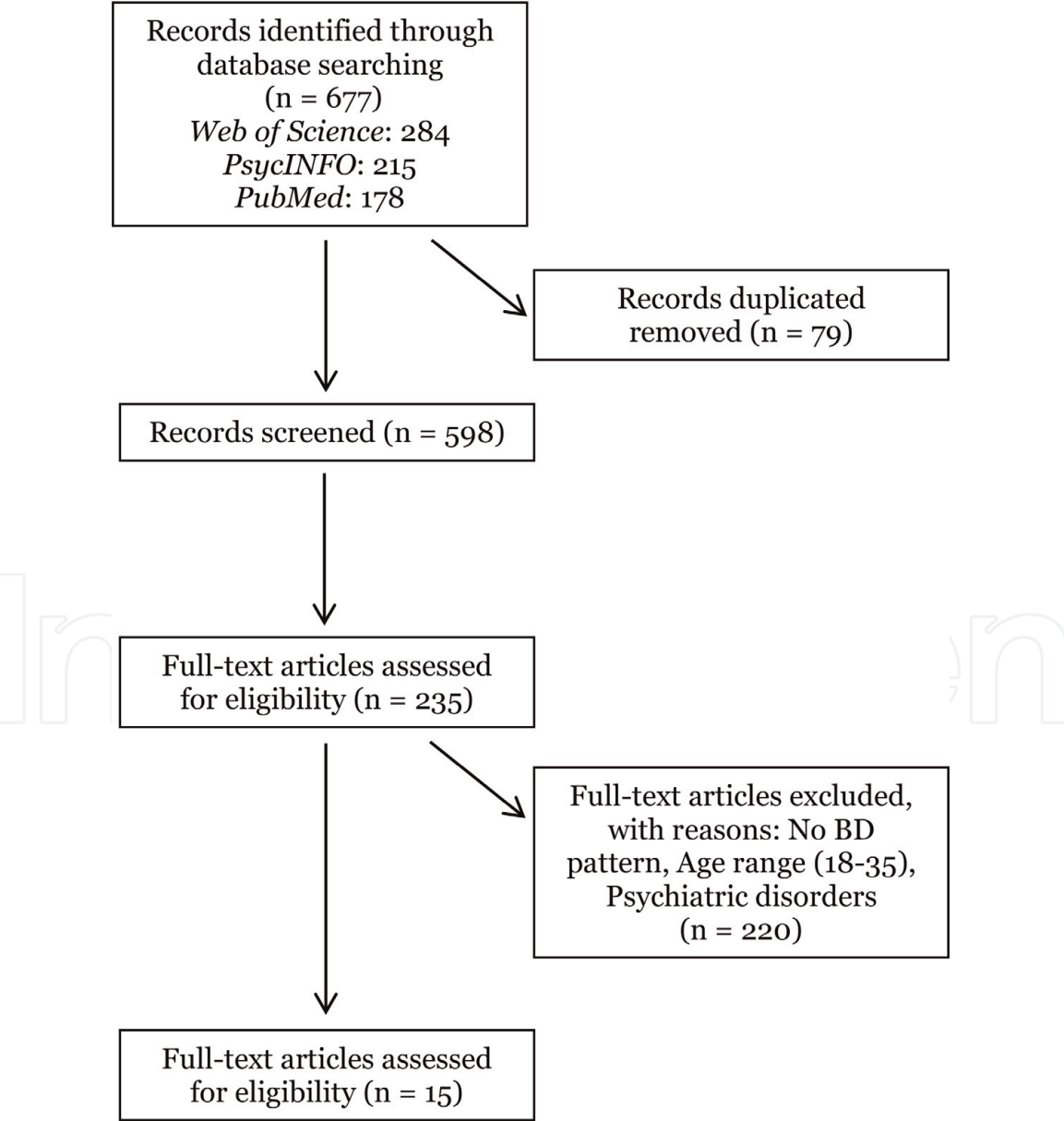


Figure 2.
Preferred reporting items for systematic reviews and meta-analyses (PRISMA) flow diagram showing how articles were selected for review.

this age group could have a particularly deep impact and last through to adulthood. Moreover, females would seem to be more vulnerable to these detrimental effects of alcohol [22].

In the following sections, the main insights provided by studies performed by our group and other researchers about the effects of BD on learning and memory performance will be discussed. We focus on the types of memory that are most damaged by alcohol: immediate visual memory (IVM) and working memory (WM). One distinctive contribution of this chapter is to evaluate, together, the impact of an acute BD episode and the sample's history of consumption on learning and memory performance, as well as the possible gender differences at play.

For this review, we conducted a literature search of three databases: *Web of Science*, *PsycINFO*, and *PubMed*. The following combination of key terms was used: binge/heavy/social OR adolescent/young OR blood alcohol OR immediate/working/memory OR alcohol/ethanol OR cognitive AND acute alcohol. These keywords were examined in the "title" section for *Web of Science* and *PsycINFO* and "title/abstract" sections for *PubMed*. We considered studies published in English since 2000 (1 January 2000–30 November 2018) in humans. The total number of studies identified through the initial database searching was 677 (*Web of Science*, 284 records; *PsycINFO*, 215 records; *PubMed*, 178 records). Duplicated records were removed, and other articles were excluded using strict exclusion criteria: no BD pattern, out of age range (18–35 years old), psychiatric disorders, and other criteria described in the "methods" section. Eventually, 15 full-text articles were included in this review (see **Figure 2** and **Table 1**). This review is limited by the publication bias (databases not included), procedure of selection bias, and unavailable data.

2. Methods

2.1 Subjects

The experimental subjects in our studies (e.g., [23, 24]) were adolescent university students, who filled in a self-report questionnaire about consumption of drugs, frequency and level of alcohol consumption, hours and quality of sleep, and physical and psychological health. They were recruited based on strict inclusion and exclusion criteria. The inclusion criteria used were 18–19 years old, a healthy body mass index (between 20 and 25), and good health (without major medical problems). The subjects had to be refrainers (or very occasional alcohol consumers) or binge drinkers. The exclusion criteria were as follows: on medication; a history of mental disorders (diagnosed by a health professional according to DSM criteria); an irregular sleep pattern (non-restorative sleep and/or irregular schedule); having consumed, albeit sporadically, any drug (apart from alcohol) or having a history of substance abuse, including caffeine (our criterion: ≤ 2 stimulant drinks/day), tobacco (our criterion: ≤ 10 cigarettes/day), and alcohol (except for the BD consumption pattern); and having first-degree relatives with a history of alcoholism.

Other studies reviewed in this chapter included adolescents and young adults (18–35 years old) selected by similar or less restrictive inclusion/exclusion criteria, considering the alcohol use of subjects (history of problems due to alcohol use) and a history of mental health treatment (e.g., [25]).

2.2 Gender

Gender differences in the effects of alcohol have been reported, supporting the view that the brains of male and female adolescents are differentially affected by

alcohol use [22]. There is evidence suggesting that female adolescents are more vulnerable to the neurotoxic effects of alcohol on cognition [22, 26, 27], since the cognitive tolerance effect of alcohol on IVM develops in BD women but not in BD men [24]. Other authors have found that men generally report lower sensitivity to alcohol (individuals need more alcohol to experience the same sensations or impairments) than women, and reactivity to alcohol-related cues is more pronounced in male than in female binge drinkers (e.g., [11]). These results might at least partially explain why men typically show a higher prevalence of alcohol consumption than women. However, in Spain at least, the incidence of alcohol consumption in 14–18-year-old adolescents is higher among females than males [4], while the BD pattern during adolescence is similar in 14–16-year-old adolescents and is more common among men than women in the age range of 17–18 years [4].

Gender differences in WM have also been reported in healthy young subjects, showing an advantage in this memory among males, with females exhibiting disadvantages manifested by a small effect size in both verbal and visuospatial WM [28]. This male advantage could be due to the activating effects of testosterone [29], though age and specific task modulate the magnitude and direction of the effects (e.g., [28, 30]). However, there are reviews in literature that explore the history of BD consumption but not the acute effects it exerts and which does not support the existence of gender differences in the effects of alcohol on this type of memory (e.g., [31]).

In the light of these data, it would seem crucial to consider (a) including both sexes, men and women, in any studies carried out and (b) evaluating potential gender differences in the relationship between BD and memory in adolescents and young adults.

2.3 Pattern alcohol consumption

Selected subjects were invited to participate in our studies if they reported refraining from alcohol consumption (or having indulged in very sporadic consumption) or a history of alcohol use classified as a BD pattern according to the NIAAA criteria for Spain (see [12]). Subsequently, the participants were classified as fulfilling a BD pattern if they had drunk six or more standard drink units (SDU) in the case of men or five or more SDU in the case of women on a minimum of two or three occasions per month throughout the 12 months prior to the survey. In Spain a SDU = 10 g of alcohol of distilled spirits (alcohol content $\geq 40\%$ vol.). It is important to clarify that a stable BD pattern maintained over the time (12 months in the case of our studies) is a crucial criterion, because repeated alternation between intoxication and abstinence has been shown to be particularly harmful to the developing brain [19, 20]. Participants were classified as refrainers if they had never consumed alcoholic beverages or had drunk very sporadically (<1 SDU on <3 occasions per year, for example, 250 ml of beer, per occasion) since the onset of their alcohol use.

Therefore, in the studies reviewed in this chapter, including ours:

- A. The experimental subjects were nondependent individuals indulging in alcohol use, usually evaluated by the Alcohol Use Disorders Identification Test (AUDIT) or others, such as the brief Michigan Alcoholism Screening test (e.g., [25]).
- B. A very noticeable factor is the variability both in the samples' history (refrainers, habitual consumers, binge drinkers, light binge drinkers, etc.) and in the acute administration of alcohol that leads to a BAC of 0.8 g/L (see

Table 1 “sample’s history of consumption” and “cognitive performance—with (BAC)—” entries for details).

- C. Depending on the study, performance in the memory task was registered as either rising or declining BACs.
- D. Taking into account the scarcity of studies evaluating acute alcohol consumption in adolescent and young adult refrainers or occasional consumers (e.g., [23]), the present chapter provides unique insights into this field of research.

2.4 Memory tests

In our studies, the third edition of the Wechsler Memory Scale (WMS-III; version adapted for the Spanish population) [32] was used to assess IVM and WM. The IVM subscales require the respondent to recognize faces and remember scenes, while the WM subscales require the respondent to put letter-number sets in order and to reproduce visual-spatial sequences. The literature reports a poorer performance in these types of memory under the acute effects of alcohol (e.g., [24, 33]) and especially in WM associated with a stable BD maintained in time (e.g., [34]).

Other scales used for the evaluation of these or similar types of memory are:

- The Cambridge Neuropsychological Test Automated Battery (CANTAB) for evaluating spatial recognition memory. The CANTAB is a computer-based cognitive assessment system consisting of a battery of neuropsychological tests, administered to subjects using a touch screen computer. This battery evaluates several areas of cognitive function using nonverbal stimuli in the majority of its tests, including the pattern recognition memory, a test of visual recognition memory in a two-choice forced discrimination paradigm.
- The Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) for evaluating long- and short-term memory, working memory, and declarative memory. The ImPACT is a computer-based program for assessing neurocognitive function and concussion symptoms. This neurocognitive test battery consists of several modules for evaluating attentional processes, verbal recognition memory, visual working memory, visual processing speed, reaction time, and numerical sequencing ability.
- The Wechsler Adult Intelligence Scale (WAIS-R) with the digit symbol substitution test (DSST) for evaluating short-term memory. The WAIS-R (revised form of the WAIS, a test designed to measure intelligence and cognitive ability in adults and older adolescents) consisted of six verbal and five performance subtests, including the DSST. This subtest (DSST-WAIS-R) consists of digit-symbol pairs followed by a list of digits; under each digit the subject must write down the corresponding symbol as fast as possible within the allowed time.

Obviously, the use of different tests/batteries for evaluating memory contributes to the heterogeneity of results in this field of research.

2.5 Procedure

In our procedure, all participants signed an informed consent and a data confidentiality agreement on arrival at the laboratory. BAC was measured in all subjects

with an alcoholmeter to ensure that they had not previously drunk alcohol on the day in question, and the alcohol use of the BD adolescent subjects was assessed using the AUDIT test (none of the subjects was assessed as alcohol-dependent). Next, refrainers and binge drinkers drank 330 ml of lime- or orange-flavored refreshment (control groups), and binge drinkers' drank a high dose of alcohol. Alcohol was administered in a fixed dose of 120 ml (38.4 g) consisting of vodka mixed with the abovementioned refreshment for both genders or in function of their body weight (0.9 g alcohol/kg body weight in men and 0.8 g alcohol/kg body weight in women). The subjects were instructed to consume their drink within a period of 20 min. After finishing the drink, all subjects rinsed their mouths with water, and BAC was repeatedly measured every 5 min throughout the waiting period, until it reached a peak (approximately 20 min after consuming the drink). This peak of BAC was considered the value with which to classify the participants into the different experimental groups. The subjects performed the IVM and WM tests, while BAC was descendent. BAC was measured once again at the beginning of the tests, between the tests and at the end of the experiment. The BACs registered for the male and female subjects (separately or together) in the different experimental groups were:

- A. 0.0 g/L, in refrainers men ($n = 17$) and women ($n = 24$) or BD men ($n = 23$) and women ($n = 27$). These are control groups receiving a nonalcoholic drink.
- B. 0.33 g/L, in refrainers men ($n = 17$) or BD men ($n = 22$).
- C. 0.38 g/L, in refrainers men ($n = 11$) and women ($n = 11$) or BD men ($n = 11$) and women ($n = 11$).
- D. 0.5 g/L, in refrainers ($n = 18$) or BD women ($n = 24$).
- E. 0.3–0.5 g/L (mean = 0.4 g/L), in BD men ($n = 12$) and women ($n = 12$).
- F. 0.54–1.1 g/L (mean = 0.8 g/L), in BD men ($n = 14$) and women ($n = 24$).

(Note: The A, B, C, and D experimental groups belong to Ref. 23; and the A, E, and F experimental groups belong to Ref. 24).

All tests were performed between 4:00 pm and 8:00 pm, and the subjects that received alcohol remained on the premises until their alcohol concentration dropped to legal limits for driving (<0.3 g/L).

Similar procedures were applied in the other reviewed studies, where cognitive performance—with (BAC)—was evaluated after alcohol intake administered in fixed doses or according to body weight. Participants also abstained from alcohol for at least 12 h prior to the experiment, as well as drinking coffee or tea on the mornings prior to the experiment, and were instructed to eat a low-fat breakfast and lunch on the day on which tests were performed (e.g., [35]).

3. Results

The main findings obtained in our experimental investigations and those of other groups are summarized in **Table 1**. The effects of acute alcohol consumption—one BD episode with different BACs—on different types of memory are reviewed.

A total number of 15 studies are summarized. Only three of them included adolescent male and females (18–20 years old) [23, 24, 33]; the participants in the

rest of the studies were in the 18–35-year-old group, without studies comparing adolescents and young adults.

The sample’s history of consumption encompasses a range from refrainers to heavy binge drinkers, including habitual consumers/moderate drinkers and light binge drinkers. This variability in the samples of the reviewed studies gives us a more specific view of the acute effects of alcohol in different types of consumers and not only in binge drinkers.

In general, the results obtained in the evaluated memory tasks confirm the deleterious effects of alcohol use. Significant impairments were observed in spatial recognition memory, WM, associative learning, word fragment completion, free recall, long-term memory, short-term memory, and IVM. However, an absence of effects has also been observed with respect to some of these memories, such as visual memory, short-term memory, WM, and IVM. It is possible that the impairing effects observed are conditioned by BAC (ascendant BAC, BAC peak, descendant BAC) in the case of some types of memory. Thus, in studies in which there were

<i>Sample</i>	<i>Sample’s history of consumption</i>	<i>Memory/Test</i>	<i>Cognitive performance -with (BAC)-</i>	<i>Ref.</i>
N = 50 (18-34 years old) Men/Women	BD (12-45 SDU/week)	↓ Spatial recognition memory (CANTAB)	BD (0.59-0.52 g/L) < Placebo	[35]
N = 41 (≥ 18 years old) Men	BD (≥ 8 SDU /week) / Light BD (< 8 SDU /week)	↓ WM (Random Object Span Task)	BD and Light BD (0.8 g/L) < Placebo	[36]
		↓ Associative learning	Light BD (0.8 g/L) < Placebo	
N = 72 (19-25 years old) Men/Women	Habitual consumers (1.11 ml ethanol/kg weight and 1.2 occasions/week)	↓ WM (Sternberg Memory Scanning task)	Habitual consumers (ascendant BAC 0.68-0.86 g/L) < Placebo	[37]
		= WM (Sternberg Memory Scanning task)	Habitual consumers (descendant BAC 0.86-0.64 g/L) = Placebo	
N = 69 (21-29 years old) Men	Habitual consumers (3.9 SDU and 2.3 occasions/week)	↓ Associative learning	Habitual consumers (0.8 g/L) < Placebo	[38]
		↓ Word fragment completion	Habitual consumers (0.3 g/L) < Placebo	
		↓ Free recall	Habitual consumers (0.3 g/L) < Placebo	
		= Visual memory (Picture recognition)	Habitual consumers (0.8 g/L) = Placebo	

N = 20 (21,8 mean years old) Men	--	↓ Long-term memory (ImPACT)	Alcohol consumers (ascendant BAC: 0.81-0.93 g/L) < Placebo	[39]
		↓ Long-term visual memory (ImPACT)	Alcohol consumers (ascendant BAC: 0,81-0.93 g/L) < Placebo	
		↓ Short-term memory (ImPACT)	Alcohol consumers (ascendant BAC: 0.81-0.93 g/L) < Placebo	
		↓ WM (ImPACT)	Alcohol consumers (ascendant BAC: 0.81-0.93 g/L) < Placebo	
		↓ Declarative memory (ImPACT)	Alcohol consumers (ascendant BAC: 0.81-0.93 g/L) < Placebo	
		= WM (ImPACT)	Alcohol consumers (descendant BAC 0.86-0.79 g/L) = Placebo	
		= Short-term memory (ImPACT)	Alcohol consumers (descendant BAC 0.86-0.79 g/L) = Placebo	
N = 72 (21-35 years old) Men/Women	BD (≥ 10 SDU and 1.5 occasions/week) / Light BD (< 6 SDU /week)	↓ Short-term memory (DSST-WAIS-R)	BD (0.92 g/L) < Placebo Light BD (0.82 g/L) < Placebo	[40]
		= Short-term memory (DSST-WAIS-R)	BD (0.42 g/L) = Placebo Light BD (0.36 g/L) = Placebo BD = Light BD	
N = 72 (21-30 years old) Men/Women	BD (2-24 SDU /week)	↓ WM	BD (0.9 g/L) < Placebo	[41]
N = 27 (21-35 years old) Men/Women	Habitual consumers (5 SDU /week)	= Short-term memory (DSST-WAIS-R)	Acute consumers of 0.8 g alcohol/kg weight = Placebo	[42]
N = 10 (19-29 years old) Men/Women	Habitual consumers (12-30 SDU /month)	= Visual WM [43]	Habitual consumers (0.6 g/L) = Placebo	[44]
N = 32 (18-30 years old) Men	Habitual consumers (31.8-36.3 SDU /week)	= WM (Spatial Span)	Habitual consumers (0.73 g/L) = Placebo	[45]

		= WM (Baddeley's Reasoning task)	Habitual consumers (0.73 g/L) = Placebo	
N = 91 (18-20 years old) Men/Women	--	↓ WM (Trail Making Test- Trail B)	Alcohol consumers (0.84 g/L) < Placebo	[33]
N = 19 (18-35 years old) Men	Habitual consumers (≥ 4 SDU and 2 occasions/month)	↓ Short-term memory (DSST- WAIS-R)	Placebo > habitual consumers (0.78 g/L)	[46]
N = 75 (21-35 years old) Men/women	Moderate drinkers / Heavy drinkers	= WM (Wais-III and IV)	Moderate-Heavy drinkers (0.63 g/L) = Moderate-Heavy drinkers (0.0 g/L)	[25]
		= WM (Trail Making Test- Trail A-B)	Moderate-Heavy drinkers (0.63 g/L) = Moderate-Heavy drinkers (0.0 g/L)	
N = 79 (18-19 years old) Men	Refrainers / BD (≥ 6 SDU and ≥ 2 occasions/month)	= Immediate visual memory (Wechsler-III)	Refrainers (0.0 g/L) = Refrainers (0.33 g/L) = BD (0.0 g/L) = BD (0.33 g/L)	[23]
		= WM (Wechsler-III)	Refrainers (0.0 g/L) = Refrainers (0.33 g/L) = BD (0.0 g/L) = BD (0.33 g/L)	
N = 93 (18-19 years old) Women	Refrainers / BD (≥ 5 SDU and ≥ 2 occasions/month)	↓ Immediate visual memory (Wechsler-III)	Refrainers (0.5 g/L) < Refrainers (0.0 g/L) BD (0.0 g/L) < Refrainers (0.0 g/L)	[23]
		= WM (Wechsler-III)	Refrainers (0.5 g/L) < BD (0.5 g/L) Refrainers (0.0 g/L) = Refrainers (0.5 g/L) = BD (0.0 g/L) = BD (0.5 g/L)	

N = 136 (18-19 years old) Men/Women	Refrainers / BD (Men ≥ 6 SDU, women ≥ 5 SDU; and ≥ 2occasions/ month)	↓ Immediate visual memory (Wechsler-III)	Men BD (0.38 g/L) < Men BD (0.0 g/L)	[23]
		= WM (Wechsler-III)	Women Refrainers (0.38 g/L) < Women Refrainers (0.0 g/L)	
N = 154 (18-19 years old) Men/Women	Refrainers / BD (Men ≥ 6 SDU, women ≥ 5 SDU; and ≥ 2occasions/ month)		Refrainers (0.0 g/L) = Refrainers (0.38 g/L) = BD (0.0 g/L) = BD (0.38 g/L)	[24]
		↓ Immediate visual memory (Wechsler-III)	BD (0.4 g/L) < Refrainers (0.0 g/L)	
			BD (0.8 g/L) < BD (0.0 g/L) = Refrainers (0.0 g/L)	
		↓ WM (Wechsler-III)	BD (0.8 g/L) < BD (0.4 g/L) = BD (0.0 g/L) = Refrainers (0.0 g/L)	

Table 1.
Effects of acute alcohol consumption (one BD episode with different BACs) on memory in the studies carried out in this field [37, 39, 42, 43 and 46].

ascendant and descendant BACs, impairment was reported in long-term memory, short-term memory, and WM declarative memory with ascendant BAC but not with descendant BAC.

Finally, the values for cognitive performance—with (BAC)—in **Table 1** show the absence of effects or impairing effects in every sample, including for BAC of 0.0 g/L (refrainers and binge drinkers consuming refreshment/placebo). For example, in Vinader-Caerols et al. [23], male IVM performance was refrainers (0.0 g/L) = refrainers (0.33 g/L) = BD (0.0 g/L) = BD (0.33 g/L) and women’s performance was BD (0.0 g/L) < refrainers (0.0 g/L).

4. Discussion

The key findings of this review will now be discussed. Among the types of memory reviewed, word fragment completion, free recall, and IVM appear to be the most sensitive to the effects of acute alcohol, as they are affected by moderate doses of alcohol (BAC = 0.3–0.38 g/L) in adolescents and young adults (e.g., [23, 38]). However, higher doses of alcohol (BAC levels of BD, i.e., around 0.8 g/L) are necessary for a significant impairment in other memories, such as WM (e.g., [24]) and short-term memory (e.g., [40]). A plausible explanation for the lack of effects reported with BACs under 0.8 g/L (e.g., [23, 25, 40, 44, 45]) is that the brain of binge drinkers employs compensatory mechanisms in additional brain areas to

perform the tasks adequately and that these resources are undermined at higher BACs (e.g., [24, 33, 36, 40, 41]).

In contrast to the present review, others have attempted to provide an overview of affected (and unaffected) neuropsychological functions in adolescents and young binge drinkers, without evaluating the acute effects of alcohol and considering only the subjects' history of BD (e.g., [31]). However, the interaction between a BD history of consumption and the effects of acute alcohol exposure on learning and memory needs to be studied, as some long-term effects of repeated alcohol exposure in adolescents (such as alcohol tolerance or damaged cognitive abilities) are observed more readily—if at all—following an acute dose of alcohol [23].

It is known that tolerance can develop early in adolescents and young adults without alcohol use disorder [47, 48]. Considering the scarcity of studies that have evaluated the phenomenon of tolerance in healthy adolescents and the potential vulnerability of females to the neurotoxic effects of alcohol, we performed a study [23] in which we observed that binge drinkers performed better in IVM than refrainers when given alcohol (showing the development of alcohol tolerance) and binge drinkers performed worse than refrainers after consuming a nonalcoholic control drink (as their memory would have been damaged). Thus, adolescent women are more vulnerable to the neurotoxic effects of alcohol than men, because the cognitive tolerance effect of alcohol on IVM develops in BD women but not in BD men. The phenomenon of women beginning to drink earlier and progressing more rapidly than men from drinking onset to problematic drinking, known as the “telescoping effect” [49–51], would explain why adolescent women develop cognitive tolerance earlier than men.

Although men and women have been included in some of the reviewed studies, only ours [23, 24] were carried out in order to specifically evaluate these gender differences in adolescents. In our second study [24], although the tolerance phenomenon was not evaluated (because refrainers did not consume an acute dose of alcohol), no gender differences were detected in IVM and WM performance with $BAC > 0.5$ mg/L. We suspect that an increased BAC overrides these cognitive differences between men and women. At the same time, the BAC is dependent on several factors such as rates of absorption, distribution, and elimination, as well as gender, body mass and composition, food effects, and type of alcohol. Therefore, careful extrapolation and interpretation of the BAC is needed [52].

The findings of the present review would be bolstered with a tighter control of factors that contribute to heterogeneity of results, such as:

- Not taking into account the gender factor. The inclusion of men and women in study samples is more representative of the population.
- Variability in the sample's history of consumption, which can encompass a wide range (refrainers, habitual consumers/moderate drinkers, light binge drinkers, heavy binge drinkers, etc.).
- The use of different tests/batteries for evaluating similar memories (e.g., CANTAB, ImPACT, WAIS-R).
- The registration of performance in ascendant/descendant BACs. For example, more deleterious effects are observed in ascendant BAC versus descendant BAC. Most of the studies either they evaluate memory performance in descendant BAC or they do not specify whether the BAC is ascendant or descendant.

- Variability in the age ranges in the studies. This variability (see **Table 1**), without a neat separation between adolescents and young adults, does not allow to properly compare these periods in order to find potential differences. Actually, there are not studies directly evaluating possible differences in the effects of acute BD on memory, comparing adolescents and young adults.

Several studies, using different paradigms (e.g., Stroop task, Go/No-Go task), have also shown that BD during adolescence is associated with poor inhibitory control (e.g., [7, 53]). Inhibitory control processes are developing during adolescence and youth, and a poor inhibitory function may predispose the individual to alcohol misuse [53]. Thus, impaired inhibitory control has been related to increased loss of control over drinking (i.e., a greater number of drinks per episode) [7], and this impairment seems to be related to the severity of alcohol-related problems [54, 55]. Likewise, acute and binge alcohol drinking may impair the inhibitory control and compromise the ability to prevent or stop behavior related to alcohol use. Then, poor inhibitory control can be both the cause and the consequence of excessive alcohol use. Adolescence and young adulthood may be a particularly vulnerable period due to the following reasons: (a) the weak or immature inhibitory functioning typical of this stage may contribute to the inability of the individual to control alcohol use and (b) alcohol consumption per se may alter or interrupt the proper development of inhibitory control leading to a reduced ability to regulate alcohol intake [53]. Therefore, inhibitory control training is a potential effective component of a comprehensive protocol for intervention strategies focused on this at-risk group of young adults who continue a BD trajectory into adulthood. Interventions targeting binge-drinking behavior should aim to inhibitory control training.

Increasing the knowledge about the effects of BD alcohol consumption pattern on memory and other executive functions in adolescents and young adults is also instrumental to designing programs and policy to reduce the impact of drinking in this highly vulnerable population in order to diminish the likelihood of participation in risky behaviors.

5. Conclusions

After reviewing the literature concerning the effects of one BD episode (with different BACs) on learning and memory performance in adolescents and young adults, the following conclusions can be drawn:

- Alcohol BD has differential effects depending on the type of memory. For example, IVM is more sensitive than other memories to the neurotoxic effects of acute doses of alcohol in adolescents and young adults with a BD history (IVM is affected by a moderate BAC, while WM score is undermined only by BAC levels of BD).
- BAC is an important factor to take into account when evaluating the acute effects of BD alcohol on memory performance in this type of studies.
- Women are more vulnerable to some of the detrimental effects of alcohol than men are. For example, an effect of cognitive alcohol tolerance on IVM has been observed in women but not in men. These gender differences emphasize the need to include females in studies when investigating the neurotoxic effects of alcohol in adolescents and youths.

- Further research, particularly longitudinal studies, is necessary in order to confirm the abovementioned findings and to consolidate these conclusions.

In relation to the inhibitory control in binge drinkers, taking into account the scarcity of studies evaluating inhibitory control training on alcohol consumption (e.g., [56–58]) and the lack of them evaluating this kind of training on BD, future research should investigate long-term implementation of inhibitory control training, emphasizing the importance of this training as part of the intervention strategies focused on this at-risk group.

Acknowledgements

The authors wish to thank the “Generalitat Valenciana” [PROMETEO-II/2015/020] and the “Universitat de València” [UV-INV_AE18-779336] for the funding part of the work reviewed herein. They also wish to thank Mr. Brian Normanly for his editorial assistance.

Conflict of interest


The authors have no conflict of interest to declare.

Author details

Concepción Vinader-Caerols* and Santiago Monleón
Department of Psychobiology, University of Valencia, Valencia, Spain

*Address all correspondence to: concepcion.vinader@uv.es

IntechOpen

© 2019 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. 

References

- [1] Hibell B, Guttormsson U, Ahlström S, Balakireva O, Bjarnason T, Kokkevi A, Kraus L. Substance use among students in 35 European Countries (The 2007 European School Survey Project on Alcohol and other Drugs, ESPAD, Report). Stockholm: The European Monitoring Center for Drugs and Drug Addiction; 2007
- [2] Chavez PR, Nelson DE, Naimi TS, Brewer RD. Impact of a new gender-specific definition for binge drinking on prevalence estimates for women. *American Journal of Preventive Medicine*. 2011;**40**:468-471
- [3] National Institute of Alcohol Abuse and Alcoholism. NIAAA council approves definition of binge drinking. *NIAAA Newsletter*. 2004;**3**:3
- [4] Observatorio Español sobre Drogas (OED). Encuesta Escolar sobre Uso de Drogas en Estudiantes de Enseñanzas Secundarias (ESTUDES) 2014–2015. Madrid: Ministerio de Sanidad, Servicios Sociales e Igualdad; 2016
- [5] Gil-Hernandez S, Garcia-Moreno LM. Executive performance and dysexecutive symptoms in binge drinking adolescents. *Alcohol*. 2016;**51**: 79-87. DOI: 10.1016/j.alcohol.2016.01.003
- [6] Poulton A, Mackenzie C, Harrington K, Borg S, Hester R. Cognitive control over immediate reward in binge alcohol drinkers. *Alcoholism, Clinical and Experimental Research*. 2016;**40**:429-437. DOI: 10.1111/acer.12968
- [7] Carbia C, Corral M, Doallo S, Caamaño-Isorna F. The dual-process model in young adults with a consistent binge drinking trajectory into adulthood. *Drug and Alcohol Dependence*. 2018;**186**:113-119. DOI: 10.1016/j.drugalcdep.2018.01.023
- [8] Herman AM, Critchley HD, Duka T. Binge drinking is associated with attenuated frontal and parietal activation during successful response inhibition in fearful context. *The European Journal of Neuroscience*. 2018: 1-14. DOI: 10.1111/ejn.14108
- [9] Cohen-Gilbert JE, Nickerson LD, Sneider JT, Oot EN, Seraikas AM, Rohan ML, et al. College binge drinking associated with decreased frontal activation to negative emotional distractors during inhibitory control. *Frontiers in Psychology*. 2017;**8**:1650. DOI: 10.3389/fpsyg.2017.01650
- [10] Blakemore SJ. The social brain in adolescence. *Nature Reviews. Neuroscience*. 2008;**9**:267-277
- [11] Petit G, Kornreich C, Verbanck P, Campanella S. Gender differences in reactivity to alcohol cues in binge drinkers: A preliminary assessment of event-related potentials. *Psychiatry Research*. 2013;**209**:494-503. DOI: 10.1016/j.psychres.2013.04.005
- [12] López-Caneda E, Mota N, Crego A, Velasquez T, Corral M, Rodríguez Holguín S, et al. Neurocognitive anomalies associated with the binge drinking pattern of alcohol consumption in adolescents and young people: A review. *Adicciones*. 2014;**26**:334-359. DOI: 10.20882/adicciones.39
- [13] Gogtay N, Nugent TF 3rd, Herman DH, Ordonez A, Greenstein D, Hayashi KM, et al. Dynamic mapping of normal human hippocampal development. *Hippocampus*. 2006;**16**: 664-672. DOI: 10.1002/hipo.20193
- [14] DeMaster D, Pathman T, Lee JK, Ghetti S. Structural development of the hippocampus and episodic memory: Developmental differences along the anterior/posterior axis. *Cerebral Cortex*.

2014;**24**:3036-3045. DOI: 10.1093/cercor/bht160

[15] Krogsrud SK, Tamnes CK, Fjell AM, Amlen I, Grydeland H, Sulutvedt U, et al. Development of hippocampal subfield volumes from 4 to 22 years. *Human Brain Mapping*. 2014;**35**: 5646-5657. DOI: 10.1002/hbm.22576

[16] Crean R, Vandewater S, Katner S, Huitron-Resendiz S, Taffe M. Chronic alcohol consumption impairs visuo-spatial associative memory in periadolescent rhesus monkeys. *Drug and Alcohol Dependence*. 2010;**114**:31-40. DOI: 10.1016/j.drugalcdep.2010.09.002

[17] Risher ML, Fleming RL, Boutros N, Semenova S, Wilson WA, Levin ED, et al. Long-term effects of chronic intermittent ethanol exposure in adolescent and adult rats: Radial-arm maze performance and operant food reinforced responding. *PLoS One*. 2013; **13**, 8(5):e62940. DOI: 10.1371/journal.pone.0062940

[18] Peeters M, Vollebergh WA, Wiers RW, Field M. Psychological changes and cognitive impairments in adolescent heavy drinkers. *Alcohol and Alcoholism*. 2014;**49**:182-186. DOI: 10.1093/alcalc/agt162

[19] Maurage P, Joassin F, Speth A, Modave J, Philippot P, Campanella S. Cerebral effects of binge drinking: Respective influences of global alcohol intake and consumption pattern. *Clinical Neurophysiology*. 2012;**123**: 892-901. DOI: 10.1016/j.clinph.2011.09.018

[20] Petit G, Maurage P, Kornreich C, Verbanck P, Campanella S. Binge drinking in adolescents: A review of neurophysiological and neuroimaging research. *Alcohol and Alcoholism*. 2014; **49**:198-206. DOI: 10.1093/alcalc/agt172

[21] Duka T, Gentry J, Malcolm R, Ripley TL, Borlikova G, Stephens DN,

et al. Consequences of multiple withdrawals from alcohol. *Alcoholism, Clinical and Experimental Research*. 2004;**28**:233-246. DOI: 10.1097/01.ALC.0000113780.41701.81

[22] Alfonso-Loeches S, Pascual M, Guerri C. Gender differences in alcohol-induced neurotoxicity and brain damage. *Toxicology*. 2013;**311**:27-34. DOI: 10.1016/j.tox.2013.03.001

[23] Vinader-Caerols C, Talk A, Montañés A, Duque A, Monleón S. Differential effects of alcohol on memory performance in adolescent men and women with binge drinking history. *Alcohol and Alcoholism*. 2017;**52**: 610-616. DOI: 10.1093/alcalc/agx040

[24] Vinader-Caerols C, Duque A, Montañés A, Monleón S. Blood alcohol concentration-related lower performance in immediate visual memory and working memory in adolescent binge drinkers. *Frontiers in Psychology*. 2017;**8**:1720. DOI: 10.3389/fpsyg.2017.01720

[25] Spinola S, Maisto SA, White CN, Huddleson T. Effects of acute alcohol intoxication on executive functions controlling self-regulated behavior. *Alcohol*. 2017;**61**:1-8. DOI: 10.1016/j.alcohol.2017.02.177

[26] Caldwell LC, Schweinsburg AD, Nagel BJ, Barlett VC, Brown SA, Tapert SF. Gender and adolescent alcohol use disorders on BOLD (blood oxygen level dependent) response to spatial working memory. *Alcohol and Alcoholism*. 2005;**40**:194-200. DOI: 10.1093/alcalc/agh134

[27] Squeglia LM, Schweinsburg AD, Pulido C, Tapert SF. Adolescent binge drinking linked to abnormal spatial working memory brain activation: Differential gender effects. *Alcoholism, Clinical and Experimental Research*. 2011;**35**:1831-1841. DOI: 10.1111/j.1530-0277.2011.01527.x

- [28] Zilles D, Lewandowski M, Vieker H, Henseler I, Diekhof E, Melcher T, et al. Gender differences in verbal and visuospatial working memory performance and networks. *Neuropsychobiology*. 2016;**73**:52-63. DOI: 10.1159/000443174
- [29] Janowsky JS, Chavez B, Orwoll E. Sex steroids modify working memory. *Journal of Cognitive Neuroscience*. 2000;**12**:407-414. DOI: 10.1162/089892900562228
- [30] Voyer D, Voyer SD, Saint-Aubin J. Sex differences in visual-spatial working memory: A meta-analysis. *Psychonomic Bulletin & Review*. 2017;**24**:307-334. DOI: 10.3758/s13423-016-1085-7
- [31] Carbia C, López-Caneda E, Corral M, Cadaveira F. A systematic review of neuropsychological studies involving young binge drinkers. *Neuroscience and Biobehavioral Reviews*. 2018;**90**:332-349. DOI: 10.1016/j.neubiorev.2018.04.013
- [32] Wechsler D. Wechsler Adult Intelligence Scale-III. San Antonio, EEUU: The Psychological Corporation; 2004
- [33] Day AM, Celio MA, Lisman SA, Johansen GE, Spear LP. Acute and chronic effects of alcohol on trail making test performance among underage drinkers in a field setting. *Journal of Studies on Alcohol and Drugs*. 2013;**74**:635-641. DOI: 10.15288/jsad.2013.74.635
- [34] Carbia C, Cadaveira F, López-Caneda E, Caamaño-Isorna F, Rodríguez Holguín S, Corral M. Working memory over a six-year period in young binge drinkers. *Alcohol*. 2017;**61**:17-23. DOI: 10.1016/j.alcohol.2017.01.013
- [35] Weissenborn R, Duka T. Acute alcohol effects on cognitive function in social drinkers: Their relationship to drinking habits. *Psychopharmacology*. 2003;**165**:306-312
- [36] Pihl RO, Paylan SS, Gentes-Hawn A, Hoaken PN. Alcohol affects executive cognitive functioning differentially on the ascending versus descending limb of the blood alcohol concentration curve. *Alcoholism, Clinical and Experimental Research*. 2003;**27**:773-779
- [37] Grattan-Miscio KE, Vogel-Sprott M. Effects of alcohol and performance incentives on immediate working memory. *Psychopharmacology*. 2005;**181**:188-196. DOI: 10.1007/s00213-005-2226-2
- [38] Söderlund H, Parker ES, Schwartz BL, Tulving E. Memory encoding and retrieval on the ascending and descending limbs of the blood alcohol concentration curve. *Psychopharmacology*. 2005;**182**:305-317. DOI: 10.1007/s00213-005-0096-2
- [39] Schweizer TA, Vogel-Sprott M, Danckert J, Roy EA, Skakum A, Broderick CE. Neuropsychological profile of acute alcohol intoxication during ascending and descending blood alcohol concentrations. *Neuropsychopharmacology*. 2006;**31**:1301-1309. DOI: 10.1038/sj.npp.1300941
- [40] Brumback T, Cao D, King A. Effects of alcohol on psychomotor performance and perceived impairment in heavy binge social drinkers. *Drug and Alcohol Dependence*. 2007;**91**:10-17. DOI: 10.1016/j.drugalcdep.2007.04.013
- [41] Sauls JS, Cowan N, Sher KJ, Moreno MV. Differential effects of alcohol on working memory: Distinguishing multiple processes. *Experimental and Clinical Psychopharmacology*. 2007;**15**:576-587. DOI: 10.1037/1064-1297.15.6.576
- [42] Holdstock L, de Wit H. Individual differences in responses to ethanol and

- d-amphetamine: A within-subject study. *Alcoholism, Clinical and Experimental Research*. 2001;**25**:540-548. DOI: 10.1111/j.1530-0277.2001.tb02248.x
- [43] Luck SJ, Vogel EK. The capacity of visual working memory for features and conjunctions. *Nature*. 1997;**390**:279-281. DOI: 10.1038/36846
- [44] Paulus MP, Tapert SF, Pulido C, Schuckit MA. Alcohol attenuates load-related activation during a working memory task: Relation to level of response to alcohol. *Alcoholism, Clinical and Experimental Research*. 2006;**30**:1363-1371. DOI: 10.1111/j.1530-0277.2006.00164.x
- [45] Rose AK, Duka T. The influence of alcohol on basic motoric and cognitive disinhibition. *Alcohol and Alcoholism*. 2007;**42**:544-551. DOI: 10.1093/alcalc/agm073
- [46] Cash C, Peacock A, Barrington H, Sinnott N, Bruno R. Detecting impairment: Sensitive cognitive measures of dose-related acute alcohol intoxication. *Journal of Psychopharmacology*. 2015;**29**:436-446. DOI: 10.1177/0269881115570080
- [47] Saha TD, Chou SP, Grant BF. Toward an alcohol use disorder continuum using item response theory: Results from the National Epidemiologic Survey on alcohol and related conditions. *Psychological Medicine*. 2006;**36**:931-941. DOI: 10.1017/S003329170600746X
- [48] Schuckit MA, Smith TL, Hesselbrock V, Bucholz KK, Bierut L, Edenberg H, et al. Clinical implications of tolerance to alcohol in nondependent young drinkers. *The American Journal of Drug and Alcohol Abuse*. 2008;**34**:133-149. DOI: 10.1080/00952990701877003
- [49] Piazza NJ, Vrbka JL, Yeager RD. Telescoping of alcoholism in women alcoholics. *The International Journal of the Addictions*. 1989;**24**:19-28
- [50] Johnson PB, Richter L, Kleber HD, McLellan AT, Carise D. Telescoping of drinking-related behaviors: Gender, racial/ethnic, and age comparisons. *Substance Use & Misuse*. 2005;**40**:1139-1151. DOI: 10.1081/JA-200042281
- [51] Sugarman DE, Demartini KS, Carey KB. Are women at greater risk? An examination of alcohol-related consequences and gender. *The American Journal on Addictions*. 2009;**18**:194-197. DOI: 10.1080/10550490902786991
- [52] Perry PJ, Doroudgar S, Van Dyke P. Ethanol forensic toxicology. *The Journal of the American Academy of Psychiatry and the Law*. 2017;**45**:429-438
- [53] López-Caneda E, Rodríguez Holguín S, Cadaveira F, Corral M, Doallo S. Impact of alcohol use on inhibitory control (and vice versa) during adolescence and young adulthood: A review. *Alcohol and Alcoholism*. 2014;**49**:173-181. DOI: 10.1093/alcalc/agt168
- [54] Claus ED, Feldstein Ewing SW, Filbey FM, Hutchison KE. Behavioral control in alcohol use disorders: Relationships with severity. *Journal of Studies on Alcohol and Drugs*. 2013;**74**:141-151
- [55] Smith JL, Mattick RP, Jamadar SD, Iredale JM. Deficits in behavioural inhibition in substance abuse and addiction: A meta-analysis. *Drug and Alcohol Dependence*. 2014;**145**:1-33. DOI: 10.1016/j.drugalcdep.2014.08.009.
- [56] Bowley C, Faricy C, Hegarty B, Johnstone S, Smith JL, Kelly P, et al. The effects of inhibitory control training on alcohol consumption, implicit alcohol-related cognitions and brain electrical activity. *International Journal of Psychophysiology*. 2013;**89**:342-348. DOI: 10.1016/j.ijpsycho.2013.04.011

[57] Di Lemma LCG, Field M. Cue avoidance training and inhibitory control training for the reduction of alcohol consumption: A comparison of effectiveness and investigation of their mechanisms of action. *Psychopharmacology*. 2017;**234**: 2489-2498. DOI: 10.1007/s00213-017-4639-0

[58] Jones A, McGrath E, Robinson E, Houben K, Nederkoorn C, Field M. A randomized controlled trial of inhibitory control training for the reduction of alcohol consumption in problem drinkers. *Journal of Consulting and Clinical Psychology*. 2018;**86**: 991-1004. DOI: 10.1037/ccp0000312