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Executive Functioning in Alcohol Use Studies: A Brief Review of Findings and Challenges in Assessment

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Abstract

There is a wealth of research about the links between executive functioning (EF) and alcohol use. However, difficulty may arise in interpreting findings because of the variability between studies regarding the specific components of EF measured, as well as the variability of tasks used to examine each EF construct. The current article considers each of these problems within the context of a literature review that focuses on two topics: (1) the efficacy of EF in predicting alcohol use and alcohol-related consequences, and (2) the effect of acute alcohol intoxication on EF task performance. An additional goal was to identify and describe commonly used EF measures with the intention of providing alcohol researchers information on the assessment of different EF domains. Our findings indicate that there is strong evidence supporting a relation between EF difficulties (particularly response inhibition and information updating) and alcohol use, with additional evidence of a significant interaction between EF and implicit associations on alcohol use. In contrast, research supporting a link between set shifting abilities and later alcohol use is scarce. Additionally, this review found evidence of alcohol acutely affecting many EF processes (particularly response inhibition). Overall, there is a need to replicate these findings with commonly used EF tasks (*versus* developing numerous tasks within individual laboratories) to better advance our understanding of the relation between EF and alcohol use.

Keywords

Acute; alcohol; assessment; deficits; executive functioning; inhibition; shifting; updating

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CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

1. INTRODUCTION

1.1. Definition of Executive Functioning

Executive functioning (EF) is a higher-order cognitive construct involved in the self-regulation of goal-directed behavior [1], and is thus highly relevant for the avoidance of maladaptive behaviors. For example, strong EF skills protect against early initiation of substance use [2]. Despite the link between EF and health behaviors, there remains a lack of clarity about the specific components of EF that are most important in understanding and predicting behavior. The term “executive functioning” refers to a collection of many different cognitive abilities [3], including sustained and selective attention, mental flexibility, response inhibition, supervisory control of action, and resistance to interference [1], which has led to inconsistent use of the term in research and operationalization of the construct [3]. While some authors have examined individual EF processes [4], others have argued that “the sum is greater than its parts” [5]. One of the more influential models posits that EF is comprised of three higher-order factors: set shifting, information updating, and response inhibition [6]. Several additional constructs can be added to this tripart EF model, including fluency, planning, and insight; whereas, working memory is sometimes considered to overlap with the construct of information updating [7]. The term “executive functioning,” then, has come to represent both unique, distinct executive abilities, and a composite, umbrella construct. Understanding how EF relates to a given health behavior – for example, alcohol use – requires careful consideration of the distinct aspects of EF that are investigated to better identify which EF abilities most strongly impact alcohol use behaviors, and which are most affected by drinking.

1.2. Executive Functioning & Alcohol

Individual differences in EF are relevant to the etiology of alcohol use disorders, the consequences of alcohol use, and the treatment of alcohol use disorders [8, 9]. Studies have suggested that EF deficits may put individuals at risk for the development of substance use disorders [10], make them more likely to experience problems as a result of substance use [11, 12], and contribute to limited benefit from treatment [4]. The exact pathways through which these processes occur are unclear, as it is likely that multiple pathways exist, and may be concurrently active. It may be, for instance, that deficits in set shifting or information updating make it more difficult for an individual to engage multiple coping strategies in the face of cues associated with alcohol use. Similarly, it is possible that response inhibition difficulties might affect how easily an individual can resist an urge to go to the bar, socialize with friends who engage in drinking, or drink alcohol. This complexity is evidenced in the wide variability of EF processes that have been examined in relation to alcohol use. As alluded to above, some studies utilize a composite score derived from several tasks (*e.g.*, [13, 14]), while others refer to scores on a single task that measures one process. Understanding the differences in these studies, both in which tasks are used and which constructs are assessed, can help to clarify the specific ways in which alcohol use and EF are related.

A recently published review paper examined what is known about the links between EF and alcohol use [15]. The authors conducted a systematic review of alcohol-related EF

impairments in social drinkers (as opposed to a clinical population of drinkers) and concluded that EF is not impaired in heavy drinking non-clinical samples. This review focused on social drinkers, and included a review of only seven studies, one of which was based on self-report of executive dysfunction [16] utilizing a scale that has been shown to have inconsistent validity as a self-report instrument across populations (*e.g.*, [17]). The aforementioned review by Montgomery *et al.* [15] also included an empirical study that compared heavy social drinkers to light drinkers on a series of EF tests and found that heavier drinkers performed more poorly on measures of task switching and inhibitory control. That is, in contrast to the authors' review of the literature, which indicated that there were no deficits in EF among heavy social drinkers, the empirical data they presented suggested that there are indeed observable deficits among young, non-dependent heavy alcohol users [15]. The results of this paper highlight the difficulties facing researchers in this field as they try to interpret inconsistent findings across empirical studies examining alcohol use and EF. Hence, there exists a great deal of nuance in EF assessment and in the interpretation of studies reporting EF-related results, due, in part, to: (1) the variety of constructs assessed under the broader category of "executive functioning," and (2) the variety of measures used to assess each EF construct.

1.3. Purpose of the Paper

The purpose of this paper is to explore the inconsistencies in empirical findings and EF measurement problems within two areas of alcohol use research: (1) the relation between EF and subsequent drinking behaviors and (2) the acute effects of alcohol administration on EF abilities, as assessed in both laboratory-based and field studies. In addition, we will (3) describe tasks that are frequently used to assess specific EF processes. Our aim is to better enable researchers to compare findings across studies and tasks, and to make informed choices about which tasks to use when conducting research on EF and alcohol use. Areas that will not be covered include links between EF deficits, alcohol consumption, and aggression [18–21]; the *non-acute* effects of alcohol on EF, including the effects of chronic alcohol use, treatment, and abstinence on EF [22–31]; the impact of sex on EF and alcohol use; and links between EF, alcohol expectancies, and alcohol use.

2. METHOD

Empirical articles, review papers, reference lists and meta-analyses published prior to November 2013 were identified through searches in the PsycINFO and PubMed databases¹. Titles, abstracts, and papers were reviewed and papers were included only when alcohol use or drinking-related behaviors were the primary outcome variable. Papers were excluded, for instance, if prenatal exposure to alcohol, severe mental illness, or aging was the primary focus (*i.e.*, when alcohol use was a measured, but secondary, outcome). Similarly, papers were excluded if the focus was on cognitive functions other than executive functions (*e.g.*, prospective memory). All reviewed papers utilize behavioral, as opposed to self-report,

¹The following search terms were used in PsycInfo and PubMed: (1) executive AND function* AND ti(alcohol) NOT fetal NOT prenatal NOT business NOT executives NOT ti(schizophrenia) NOT ti(bipolar); In English, In Peer-Reviewed; (2) same search term replacing "executive AND function*" with "working memory"; (3) replacing with "response inhibition"; (4) replacing with "shifting"; (5) all searches additionally run with "drinking" instead of "alcohol".

measures of EF. While there is some evidence of a correlation between self-reported EF and performance on EF tasks, in at least some populations [32], there are questions about the validity of self-reported levels of EF [33] and the validity of self-report data in all cognitive domains [34, 35]; thus, for the purposes of this paper, we are focusing on behavioral measures. In total, 49 articles met the study criteria and were included in the current paper.

3. FINDINGS

3.1. The Relation Between EF and Subsequent Drinking Behaviors (n=15)

The first section of this paper reviews the literature exploring the link between EF deficits and several alcohol-related behaviors, including initiation of alcohol use, frequency or intensity of consumption, negative consequences associated with use, and information processing biases (*e.g.*, dwell time on alcohol-related stimuli in eye-tracking paradigms) (see Table 1). Studies listed in Table 1 are grouped with respect to the three higher-order EF factors: set shifting, information updating, and response inhibition. Each subsection lists papers in chronological order. Set shifting refers to the capacity of an individual to flexibly switch back and forth between mental tasks; information updating is the ability to monitor incoming information for relevance to the task at hand and act accordingly; inhibition is the ability to inhibit a dominant, automatic or prepotent response ([6]; for more information on the tasks that are listed in Tables 1 and 3). While statements regarding the contribution of EF deficits to the initiation of heavy alcohol use might be best derived from research on individuals who have yet to initiate alcohol use, to our knowledge, there are only two studies that have examined the role of EF in predicting alcohol use and related problems among alcohol-naïve² adolescents [36, 37]. These are denoted with an asterisk (*) in Table 1. The other studies we have examined in this section use cross-sectional data with alcohol-related dependent variables. In relying mostly on data from cross-sectional studies, we are unable to speak to temporal precedence. Still, we chose to include these studies in the review, as they provide meaningful data regarding relationships between EF and alcohol-related variables. The dearth of available research with alcohol-naïve individuals highlights the need for additional studies on this topic, as such studies could provide a more clear understanding of the link between pre-existing EF deficits and alcohol use.

Data from several short-term longitudinal studies examining individuals that have already initiated drinking provide evidence of a relation between EF and subsequent alcohol use. Results from such studies should be considered with a caveat, as their findings are complicated by the fact that alcohol use may be driving EF impairment, and most cannot speak to the relation between pre-existing EF deficits and initiation of drinking behaviors. Nevertheless, the data indicate that performance on tasks of response inhibition and planning predict drinking in the following week, above and beyond the predictive power of intention to drink [38]. In another study, lower EF was related to a variety of risk-taking behaviors, including hazardous drinking, above and beyond personality [39].

There are also studies examining the relation of EF to implicit biases for alcohol-related information. These studies indicate that EF abilities and implicit associations –

²10 days of drinking total; never more than 2 drinks in a week.

operationalized as an individuals' tendency to pair alcohol-related stimuli with positively valenced words – interact to predict self-report of alcohol use [40] and dwell time on alcohol-related pictures [41]. Similarly, poor inhibitory control (Go/No-Go task) interacts with self-report behavioral approach sensitivity to predict alcohol use, and furthermore poor inhibitory control also interacts with poor decision-making (Iowa Gambling Task) to predict alcohol use [42]. That is, for those individuals who are low in inhibitory control, being motivated to approach novel stimuli or having reduced decision-making skills may represent independent pathways to conferring vulnerability to alcohol use [42]. Yet, in the same study, researchers found that performing well on a working memory task (N-back), in combination with having strong behavioral approach sensitivity was related to more frequent alcohol use, indicating that, for those with strong working memory, personality constructs such as approach sensitivity may be less relevant in the prediction of alcohol use [42].

Several studies have investigated the impact of cognitive training on drinking behaviors, as a form of intervention. One such study found that improving working memory through cognitive training leads to reduced alcohol use one month later, most notably for adults with the strongest implicit associations [43]. In addition, protocols focusing solely on improving impulse control (*via* a Go/No-Go paradigm) appear to be inferior to protocols that also involve active devaluation of alcohol-related stimuli with respect to the ability to reduce drinking behavior [44]. However, these findings are in contrast to evidence suggesting that EF does not interact with implicit attitudes to predict alcohol use [45].

Researchers investigating the link between EF and relapse vulnerability indicate that, among individuals who have already developed an alcohol use disorder, poorer working memory (Alpha Span task), reduced response inhibition (Hayling Test), and reduced prefrontal perfusion (measured by single-photon emission computed tomography [SPECT] neuroimaging) predict relapse after alcohol abstinence [46]. Yet, in a 6-month longitudinal study, response inhibition (Go/No-Go) did not predict relapse among abstinent alcoholics [47], and in two additional studies, performance on a battery of standardized tests of EF (Trail Making Test-Part B, Stroop Test, Phonemic Word Fluency Test [FAS], Wisconsin Card Sort, Booklet Category Test, Verbal Abstraction Test) did not predict treatment outcome [48, 49].

3.2. Summary of EF's Role in Predicting Alcohol Use

Taken together, these data provide compelling evidence that EF deficits may create vulnerability for engaging in alcohol use. There are also interactions between EF and implicit associations, which generally support a dual process framework, in which automatic associations may be moderated by cognitive control [50]. In addition, there is evidence to support a link between EF deficits and relapse risk, but here the data appear to be somewhat inconsistent. Notably, differences in blood flow to regions of the prefrontal cortex (a neural region that contributes preferentially to EF) have been shown to correlate with relapse after alcohol abstinence, indicating that underlying differences in the ways in which these regions function might moderate alcohol use behaviors and risk. Nevertheless, additional clarity is needed regarding the relation between EF deficits and relapse risk. In summary, EF appears to be relevant not only for initiation and maintenance of alcohol use, but for maintenance of

abstinence after drinking cessation; however, published results on these phenomena are limited.

Findings highlight the importance of (1) using longitudinal research methods that incorporate alcohol-naïve individuals (children, adolescents) in order to more clearly assess the ways in which pre-existing deficits in EF influence alcohol use; (2) utilizing a multi-method assessment of different EF components (see [42], in which inhibitory control and working memory differentially predict drinking); (3) taking personality variables (*e.g.*, approach sensitivity) into account. In addition, it is important to assess the relation of EF to drinking behavior across various developmental stages, as age of participants may be of particular importance. For example, when college-age students are tested, EF has a small, but significant, effect on binge drinking in the past week [38], indicating that for college-age students, other variables may be more important (such as peer/environmental factors). By contrast, in a study of children, EF abilities predicted drinking above and beyond other cognitive factors and family history of alcoholism [12]. A final consideration includes the need for future studies on these topics to incorporate a developmental framework that considers how EF and frontal lobe system changes which occur across the lifespan [51, 52] might increase vulnerability to alcohol use problems in certain populations (*e.g.*, adolescents).

3.3. Acute Effects of Alcohol Administration on EF Abilities, as Assessed in Both Laboratory-Based and Field Studies (n=35)

Table 2 describes laboratory alcohol administration and field studies that examine the acute effects of alcohol on EF. Table 2 is divided into the same three sections as Table 1, which reflect three higher-order factors within EF: set shifting, information updating, and response inhibition. Some studies (*e.g.*, [10]) are listed in two sections in Table 2 because they measured more than one EF construct. Within each category, studies are listed in roughly ascending order of the dose of alcohol used. By separating the available studies into these categories, we see that there is a relative dearth of published information on alcohol's acute effects on set shifting (n=7 studies), while there are several reports of alcohol's effects on both information updating (n=17) and response inhibition (n=18).

Another noticeable pattern in Table 2 is related to the doses of alcohol used in published research. Very few studies use or report on doses of alcohol less than 0.6 g/kg, and of those, only one study [53] reports on a dose as low as 0.2 g/kg. This may be because laboratory and field studies of alcohol's acute effects recruit heavy drinkers for safety reasons (*e.g.*, these drinkers are more likely to demonstrate an ability to endure alcohol administration without serious adverse effects), and for heavy drinkers, a dose of 0.2 g/kg is potentially less likely to have noticeable cognitive influence due to alcohol tolerance. Similarly, only 2 studies [10, 54]

The third notable element of Table 2 is the range and number of different tasks used to assess EF among drinkers after alcohol administration. Several of the tasks used are not standardized measures typically utilized in clinical settings and were instead either developed for the studies, or otherwise used only for research. The use of experimental tasks contributes to some of the difficulty in evaluating results and summarizing findings across

studies. In order to provide readers a better understanding of common EF tasks, including many of those listed in Table 2, we present in Table 3 a list of frequently used EF measures, along with the component of EF that they assess.

Despite the range of methods in studies conducted to date, Table 2 shows evidence that alcohol acutely affects several EF processes. Some individuals appear particularly prone to EF impairment due to alcohol, such as those with ADHD [57] or those who score highly on measures of sensation-seeking [58]. It may also be that those who exhibit one type of EF deficit might be at greater risk for impairment on tasks assessing other EF domains after consuming alcohol. In one study, alcohol led to greater impairment in response inhibition for those who had lower working memory [10]. Understanding which combinations of EF deficits emerge in response to alcohol administration may help to understand the order in which processes are affected and who might be most affected by alcohol.

One component of EF that is not consistently demonstrated as being vulnerable to the acute impairing effects of alcohol is visuospatial working memory (VSWM). Of the 35 studies, there are three reports of alcohol having an effect on VSWM, and five [59–62] that report no effect of alcohol on VSWM. All 8 of these studies assessed EF in participants who had consumed moderate or high doses of alcohol (most studies in the range of 0.4 g/kg to 0.10 g/kg), which limits understanding of how alcohol at either lower or higher doses influences VSWM. Although 3 out of 8 studies (40%) observed an effect of alcohol on VSWM, results are inconsistent and more research is needed to better understand these effects.

Comparatively, there appears to be more consistent support that response inhibition is affected by acute alcohol, with 16 out of 20 studies reporting this effect. It is always possible that there is a bias in reporting (*i.e.*, a “file drawer problem”), but alcohol’s effect on response inhibition, as measured by Go/No-Go tasks, is one of the clearer effects emerging from this examination of the literature. Notably, response inhibition is measured in a variety of ways. For example, some tasks measure the capacity of an individual to inhibit engagement following particular cues (*e.g.*, the No-Go condition of the Go/No-Go task) and others measure disengagement (*e.g.*, letting go of an already pressed button); see [63] for an in-depth discussion. Researchers wishing to examine elements of response inhibition, and the influence that alcohol has on different components of this phenomenon, could enhance our understanding by selecting tasks that are specific to the question being asked, and by being aware of the differences in types of response inhibition tasks.

3.4. Summary of Acute Effects of Alcohol on EF

Alcohol has clear acute effects on many different EF components, including updating, set shifting and response inhibition, although there are more consistent findings within some elements of EF (*i.e.*, alcohol reliably affects verbal and auditory working memory) than others (visuospatial working memory is not as reliably affected). Accordingly, it has been suggested that acute alcohol ingestion, at moderate doses, produces greater impairment of rehearsal strategies, such as those used in verbal working memory, but does not have as great of an influence on sustained focus, which is required by tasks evoking VSWM [62]. By contrast, the findings regarding alcohol’s effect on set shifting are less equivocal, with 7 of 8 studies finding an influence of a range of alcohol doses (0.04 – 0.15%) on set shifting

abilities. Response inhibition is the domain that has the greatest number of published studies (n=20), and taken together, their results indicate that alcohol has a reliable influence on inhibition. It is notable that the majority of studies that examine alcohol's acute influence on inhibition come from a single lab; hence, replication with varied populations in diverse settings is warranted to bolster ecological validity. Nevertheless, it has been posited that response inhibition and set shifting share common underlying cognitive processes and neural substrates [64, 65], and as such, it seems quite plausible that evidence supporting alcohol's effects on these two processes would be similarly compelling.

There is a clear need for additional studies examining the acute effects of alcohol on EF. We lack a clear understanding of the relation between alcohol-induced EF impairments in the acute phase, to drinking behaviors that occur in the short- and long-term. For example, how acute effects of alcohol contribute to loss of control over drinking, or how lasting changes in EF contribute to the development of alcohol use disorders. Such studies may help to further illuminate critical issues in alcohol research, including the identification of individual differences and genetic susceptibilities that contribute to not only the development of alcohol use disorders, but those that predict treatment response, effects of alcohol on EF in relation to other alcohol-related behaviors of concern (e.g., binge drinking, decision making, risk taking, aggressive behaviors, *etc.*), and effects of alcohol-related neurotoxicity.

4. TASKS AND MEASUREMENT ISSUES

The final section of this paper will provide an overview of measurement issues associated with the study of EF, as this is an integral part of being able to synthesize and interpret this area of research. The EF measures in the reviewed studies are summarized in Table 3, including a brief description of the measure, the aspect(s) of EF assessed by each, and notes on administration and appropriateness for repeated assessment. Table 3 covers tests commonly used in clinical and research settings. Tasks used only for research that have not been widely adopted are not considered in Table 3. The wide range of assessments covered in this review is in part reflective of the larger taxometric difficulties associated with defining and isolating the sometimes overlapping components of EF (see [66]).

Other methodological and psychometric concerns complicate test selection as well, creating a difficult balancing act between reliability/validity, availability of normative data, ease of administration, and resistance to practice effects. For example, the Wisconsin Card Sorting Test, a task of set-shifting and rule acquisition, is relatively well-normed across a wide age range (5 years – 89 years; [34]) and is one of the most commonly administered measures in neuropsychological, forensic, and overall clinical assessments [67]. However, the task can be cumbersome and lengthy (15 – 30 minutes) to administer, highly frustrating for the examinee, and may be considerably susceptible to practice effects, particularly in higher functioning individuals [68, 69].

In addition, the quality and availability of normative data is of paramount concern [35, 70], and both aspects may vary widely along multiple sociodemographic variables, such that a task with robust norms for Caucasian individuals aged 20 – 40 years may not have the same psychometric properties when administered to an individual who differs in age or ethnic

background, and may thus be less valid in this context. Even determining which variables are relevant to performance on a task (*e.g.*, age, sex, ethnicity, educational attainment, native language, estimated/premorbidity IQ, *etc.*) can be stymieing. Length and ease of test administration, perceived difficulty of the test (in regards to rapport and examinee compliance/effort), and availability of alternate forms or computerized adaptations (although such an adaptation may in itself alter psychometrics; see [71]) are additional aspects to weigh when selecting the most appropriate EF test.

In sum, inconsistent use of tests or norms across studies, poor availability of normative data across a range of sociodemographic categories, use of tests without normative data, and use of unpublished or “home-grown” research tasks impedes the ability to aggregate individual studies and interpret meta-data. Greater uniformity of assessment procedures across studies, use of more robust normative data, and greater efforts to replicate prior studies could be invaluable in properly ascertaining the population effects of alcohol on EF. The adoption of widely available, psychometrically robust EF test batteries or subtests (*e.g.*, NIH EXAMINER; [7]), across multiple laboratories would also support this goal. Moreover, the use of such batteries will promote “big data to knowledge” efforts, which are likely to grow increasingly popular and effective in the near future.

5. GENERAL DISCUSSION AND FUTURE DIRECTIONS

The relation between EF and alcohol is complex, but there are a few points that may be inferred from the literature. First, we see that premorbid differences in EF have been shown to predict initiation of alcohol use and the subsequent experience of alcohol-related problems in prospective studies. Additional studies are needed on this topic to improve our understanding of how pre-existing EF deficits in alcohol-naïve individuals contribute to the initiation of alcohol use behaviors. In addition, there are relatively few studies examining the influence of EF on alcohol use in non-naïve drinkers. Only a small number of studies have examined each of the primary domains of EF (updating, shifting, inhibition), which reduces our ability to assess the importance of each EF domain to the development of alcohol use problems. In particular, there is a relative dearth of research on the role of set shifting in predicting alcohol use; yet, studies examining adults who are intoxicated indicate that set shifting is reliably and adversely affected. Taken together, these findings suggest that there are several areas that are ripe for future research. Studies that improve our ability to utilize EF assessments to better identify individuals who may have the greatest vulnerabilities, and could therefore benefit from preventative interventions, may reduce rates of alcohol use initiation and subsequent consequences. The changes that occur in EF processing which support the transition from naïveté to alcohol initiation and then to regular alcohol use are critical to understand, as research on the sequential order of impairment would be useful in developing prevention, intervention and treatment strategies. If it can be determined that some EF processes are more likely to influence drinking, future research might examine the utility of cognitive remediation strategies for alcohol-naïve adolescents in preventing early initiation of alcohol use. Further, with more research, additional patterns might emerge, such as determining which EF components are more likely to predict initiation of alcohol use *versus* alcohol-related negative consequences, or likelihood of successful alcohol treatment.

Second, we identified the literature that shows that alcohol has clear and distinct acute effects on EF.

Compared to other EF components, such as response inhibition and set shifting, the acute effect of alcohol on visuospatial working memory (VSWM) is less well supported. While there is some evidence that visual memory is relatively robust (*e.g.*, [72]) and relatively insensitive to various neurological impairments [73], other research indicates that VSWM can be affected by several disease processes, drugs of abuse, and medical interventions [74–76]. Currently, it appears that the acute alcohol effects on VSWM may be less clinically important relative to other aspects of EF with respect to successful alcohol use monitoring or cessation. Individuals likely rely on their capacity to inhibit prepotent responses when considering whether or not to initiate a drinking episode. They might also rely on information updating and set shifting when attempting to recall sobriety or moderation goals in the face of alcohol cues/urges. By contrast, VSWM may not have as direct an influence on an individual who is attempting to act in accordance with predetermined plans around drinking. Alcohol's acute effects on VSWM, however slight, may still have an effect on other behaviors that occur in the context of alcohol use (*e.g.*, sexual decision-making).

Each of the laboratory-based studies in Table 2 assessed EF at a single time point following alcohol ingestion. This represents a gap in our knowledge, for it is likely that changes in cognition over the course of a drinking episode are dynamic. For example, impaired control over drinking is believed to occur after the first or second drink of the drinking session [77, 78], and the presence of impaired control predicts greater risk for developing an alcohol use disorder [79–82]. Thus, improving our understanding of the changes in EF that occur throughout the course of a drinking episode (with respect to the effects of ongoing consumption, as well as changes that may occur during the ascending and descending limbs) will likely have clinical and scientific import. We also need to develop a better understanding of the EF processes that are affected at relatively lower doses of alcohol. There are methodological challenges associated with answering this question, as lower doses of alcohol are metabolized quickly, limiting the amount of time available for neurocognitive assessment.

There is a general need for replication of findings, particularly with the use of standardized tests rather than reliance on home-grown research tasks, as this will improve the applicability of research findings and comparisons across studies. Studies that utilize neuroimaging tools to identify the neural correlates of EF deficits may be better able to detect subtle differences in EF than studies that rely solely on traditional EF tests [83]. Neuroimaging studies could therefore further advance our ability to understand the link between subtle EF deficits and alcohol use behaviors, and might also improve our ability to identify individuals at risk for developing alcohol use disorders. It will also be important for future studies to take family history of alcoholism into account, as it is likely that there are familial, biological, neurological, or genetic factors that contribute to both cognitive deficits and the initiation and maintenance of alcohol use disorders [84]. In addition, future studies in this area should seek to understand how developmental changes in EF and frontal lobe systems might interact with developmental periods of environmental, social, and behavioral change (*e.g.*, adolescence/early adulthood) to increase alcohol use risk [51, 52].

In sum, we examined the literature describing the links between alcohol use and EF, and provide information about different tasks that are used in the study of these constructs. There is compelling evidence that EF deficits place individuals at greater risk for a variety of alcohol-related behaviors, including initiation of alcohol use and the experience of alcohol-related problems; and that once an individual consumes alcohol, there are subsequent changes in several EF processes that may contribute to negative alcohol-related consequences.

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References

1. Lezak, MD.; Howieson, DB.; Bigler, ED.; Tranel, D. *Neuropsychological Assessment*. 5. New York: Oxford; 2012.
2. Najam N, Moss HB, Kirisci L, Tarter RE. Executive cognitive functioning predicts drug use in youth. *J Indian Acad App Psychol*. 1997; 23(1–2):3–12.
3. Suchy Y. Executive functioning: Overview, assessment, and research issues for non-neuropsychologists. *Ann Behav Med*. 2009; 37(2):106–16. [PubMed: 19455377]
4. Bates ME. Utility of component-process approaches for understanding complex alcohol-related behavior within an executive functioning framework: Comment on Giancola (2000). *Exp Clin Psychopharmacol*. 2000; 8(4):598–600. [PubMed: 11127430]
5. Giancola PR. Executive functioning and alcohol-related aggression: Reply to Weingartner (2000), Bates (2000), Lyvers (2000), Cherek (2000), and Berman (2000). *Exp Clin Psychopharmacol*. 2000; 8(4):612–7.
6. Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A, Wager TD. The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: A latent variable analysis. *Cognit Psychol*. 2000; 41(1):49–100. [PubMed: 10945922]
7. Kramer JH, Mungas D, Possin KL, et al. Conceptualization and development of an executive function battery. *J Int Neuropsychol Soc*. 2014; 20:11–9. [PubMed: 24103232]
8. Bates ME, Bowden SC, Barry D. Neurocognitive impairment associated with alcohol use disorders: Implications for treatment. *Exp Clin Psychopharmacol*. 2002; 10(3):193–212. [PubMed: 12233981]
9. Giancola, PR.; Moss, HB. Executive cognitive functioning in alcohol use disorders. In: Galanter, M., editor. *Recent developments in alcoholism: Vol. 14. The consequences of alcoholism*. New York: Plenum Press; 1998. p. 227–251.
10. Finn PR, Justus A, Mazas C, Steinmetz JE. Working memory, executive processes and the effects of alcohol on Go/No-Go learning: Testing a model of behavioral regulation and impulsivity. *Psychopharmacol*. 1999; 146(4):465–472.
11. Day A, Metrik J, Spillane N, Kahler C. Working memory and impulsivity predict marijuana-related problems among frequent users. *Drug Alcohol Dependence*. 2013; 131(1–2):171–4. [PubMed: 23312340]
12. Nigg JT, Wong MM, Martel MM, et al. Poor response inhibition as a predictor of problem drinking and illicit drug use in adolescents at risk for alcoholism and other substance use disorders. *J Am Acad Child Adolescent Psychiat*. 2006; 45(4):468–75.
13. Glass JM, Buu A, Adams KM, et al. Effects of alcoholism severity and smoking on executive neurocognitive function. *Addiction*. 2009; 104(1):38–48. [PubMed: 19133887]
14. Thoma RJ, Monnig MA, Lysne PA, et al. Adolescent substance abuse: The effects of alcohol and marijuana on neuropsychological performance. *Alcohol Clin Exp Res*. 2011; 35(1):39–46. [PubMed: 20958330]

15. Montgomery C, Fisk JE, Murphy PN, Ryland I, Hilton J. The effects of heavy social drinking on executive function: A systematic review and meta-analytic study of existing literature and new empirical findings. *Hum Psychopharmacol Clin Exp*. 2012; 27(2):187–99.
16. Heffernan T, Ling J, Bartholomew J. Self-rated prospective memory and central executive deficits in excessive alcohol users. *Irish J Psychol Med*. 2004; 21(4):122–24.
17. Bennett PC, Ong B, Ponsford J. Measuring executive dysfunction in an acute rehabilitation setting: Using the dysexecutive questionnaire (DEX). *J Int Neuropsychol Soc*. 2005; 11(4):376–85. [PubMed: 16209417]
18. Giancola PR. Executive Functioning and Alcohol-Related Aggression. *J Abnormal Psychol*. 2004; 113(4):541–55.
19. Giancola PR. The underlying role of aggressivity in the relation between executive functioning and alcohol consumption. *Addict Behav*. 2007; 32(4):765–83.
20. Giancola PR, Parrott DJ, Roth RM. The influence of difficult temperament on alcohol-related aggression: Better accounted for by executive functioning? *Addict Behav*. 2006; 31(12):2169–87. [PubMed: 16563644]
21. Godlaski AJ, Giancola PR. Executive functioning, irritability, and alcohol-related aggression. *Psychol Addict Behav*. 2009; 23(3):391–403. [PubMed: 19769424]
22. Ngandu T, Helkala E, Soininen H, Winblad B, Tuomilehto J, Nissinen A, Kivipelto M. Alcohol drinking and cognitive functions: Findings from the cardiovascular risk factors aging and dementia (CAIDE) study. *Dementia Geriatric Cogni Disorders*. 2007; 23(3):140–9.
23. Townshend JM, Duka T. Attentional bias associated with alcohol cues: Differences between heavy and occasional social drinkers. *Psychopharmacol*. 2001; 157(1):67–74.
24. Weiss E, Marksteiner J. Alcohol-related cognitive disorders with a focus on neuropsychology. *Int J Disability Human Develop*. 2007; 6(4):337–42.
25. Gross AL, Rebok GW, Ford DE, et al. Alcohol consumption and domain-specific cognitive function in older adults: Longitudinal data from the Johns Hopkins Precursors Study. *J Gerontol: Series B: Psychol Sci Social Sci*. 2011; 66B(1):39–47.
26. Goldstein RZ, Leskovjan AC, Hoff AL, et al. Severity of neuropsychological impairment in cocaine and alcohol addiction: Association with metabolism in the prefrontal cortex. *Neuropsychologia*. 2004; 42(11):1447–58. [PubMed: 15246283]
27. Lawrence AJ, Luty J, Bogdan NA, Sahakian BJ, Clark L. Impulsivity and response inhibition in alcohol dependence and problem gambling. *Psychopharmacol*. 2009; 207(1):163–72.
28. Nowakowska K, Jabłkowska K, Adamiak G, Borkowska A. P01-58 Cognitive dysfunctions in patients with alcohol dependence. *Eur Psychiat*. 2009; 24:S446.
29. Oscar-Berman M, Marinkovi K. Alcohol: effects on neurobehavioral functions and the brain. *Neuropsychol Rev*. 2007; 17(3):239–57. [PubMed: 17874302]
30. Sullivan EV, Fama R, Rosenbloom MJ, Pfefferbaum A. A profile of neuropsychological deficits in alcoholic women. *Neuropsychol*. 2002; 16(1):74–83.
31. Zinn S, Stein R, Swartzwelder HS. Executive Functioning Early in Abstinence From Alcohol. *Alcohol Clin Exp Res*. 2004; 28(9):1338–46. [PubMed: 15365304]
32. Scheiser DM, Delis DC, Filoteo JV, et al. Are self-reported symptoms of executive dysfunction associated with objective executive function performance following mild to moderate traumatic brain injury? *J Clin Exp Neuropsychol*. 2011; 33(6):704–14. [PubMed: 21958432]
33. Mitchell M, Miller LS. Executive functioning and observed *versus* self-reported measures of functional ability. *Clin Neuropsychol*. 2008; 22(3):471–9. [PubMed: 17853131]
34. Strauss, E.; Sherman, EMS.; Spreen, O. A compendium of neuropsychological tests. 3. New York: Oxford University Press; 2006.
35. Faust, D.; Ahern, DC.; Bridges, AJ. Neuropsychological (brain damage) assessment. In: Faust, D., editor. *Coping with psychiatric and psychological testimony*. 6. New York: Oxford University Press; 2012. p. 363-469.
36. Squeglia LM, Pulido C, Wetherill RR, Jacobus J, Brown GG, Tapert SF. Brain response to working memory over three years of adolescence: Influence of initiating heavy drinking. *J Studies Alcohol Drugs*. 2012; 73(5):749–60.

37. Wetherill RR, Castro N, Squeglia LM, Tapert SF. Atypical neural activity during inhibitory processing in substance-naïve youth who later experience alcohol-induced blackouts. *Drug Alcohol Dependence*. 2013; 128(3):243–9. [PubMed: 23021773]
38. Mullan B, Wong C, Allom V, Pack SL. The role of executive function in bridging the intention-behaviour gap for binge-drinking in university students. *Addict Behav*. 2011; 36(10):1023–6. [PubMed: 21665370]
39. Pharo H, Sim C, Graham M, Gross J, Hayne H. Risky business: Executive function, personality, and reckless behavior during adolescence and emerging adulthood. *Behav Neurosci*. 2011; 125(6): 970–8. [PubMed: 22004262]
40. Thush C, Wiers RW, Ames SL, Grenard JL, Sussman S, Stacy AW. Interactions between implicit and explicit cognition and working memory capacity in the prediction of alcohol use in at-risk adolescents. *Drug and Alcohol Depend*. 2008; 94(1–3):116–24.
41. Friese M, Bargas-Avila J, Hofmann W, Wiers RW. Here's looking at you, bud: Alcohol-related memory structures predict eye movements for social drinkers with low executive control. *Soc Psychol Personality Sci*. 2010; 1(2):143–51.
42. Patrick ME, Blair C, Maggs JL. Executive function, approach sensitivity, and emotional decision making as influences on risk behaviors in young adults. *J Clin Exp Neuropsychol*. 2008; 30(4): 449–62. [PubMed: 18938681]
43. Houben K, Wiers RW, Jansen A. Getting a grip on drinking behavior: Training working memory to reduce alcohol abuse. *Psychol Sci*. 2011; 22(7):968–75. [PubMed: 21685380]
44. Houben K, Havermans RC, Nederkoorn C, Jansen A. Beer à no-go: learning to stop responding to alcohol cues reduces alcohol intake *via* reduced affective associations rather than increased response inhibition. *Addic*. 2012; 107(7):1280–87.
45. Littlefield AK, Vergés A, McCarthy DM, Sher KJ. Interactions between self-reported alcohol outcome expectancies and cognitive functioning in the prediction of alcohol use and associated problems: A further examination. *Psychol Addict Behav*. 2011; 25(3):542–6.
46. Noël X, Sferrazza R, Van dL, et al. Contribution of frontal cerebral blood flow measured by 99mTc-bicisate SPECT and executive function deficits to prediction treatment outcome in alcohol-dependent patients. *Alcohol Alcoholism*. 2002; 37(4):347–54. [PubMed: 12107037]
47. Camchong J, Stenger A, Fein G. Resting-state synchrony during early alcohol abstinence can predict subsequent relapse. *Cerebral Cortex*. 2013; 23(9):2086–99. [PubMed: 22819968]
48. Morgenstern J, Bates ME. Effects of executive function impairment on change processes and substance use outcomes in 12-step treatment. *J Studies Alcohol*. 1999; 60(6):846–55.
49. Moriyama Y, Mimura M, Kato M, et al. Executive dysfunction and clinical outcome in chronic alcoholics. *Alcoholism: Clin Exp Res*. 2002; 26(8):1239–44.
50. Wiers RW, Gladwin TE, Hofmann W, Salemink E, Ridderinkhof KR. Cognitive bias modification and cognitive control training in addiction and related psychopathology mechanisms, clinical perspectives, and ways forward. *Clin Psychol Sci*. 2013; 1(2):192–212.
51. Zimmerman ME, Brickman AM, Paul RH, et al. The relationship between frontal gray matter volume and cognition varies across the healthy adult lifespan. *Am J Geriatr Psychiat*. 2006; 14:823–33.
52. Blakemore SJ. Imaging brain development: the adolescent brain. *Neuroimage*. 2012; 61(2):397–406. [PubMed: 22178817]
53. Boha R, Molnar M, Gaal ZA, et al. The acute effect of low-dose alcohol on working memory during mental arithmetic: I. Behavioral measures and EEG theta band spectral characteristics. [Research Support, Non-U.S Gov't]. *Int J Psychophysiol*. 2009; 73(2):133–7. [PubMed: 19414050]
54. Guillot CR, Fanning JR, Bullock JS, McCloskey MS, Berman ME. Effects of alcohol on tests of executive functioning in men and women: a dose response examination. Randomized Controlled Trial Research Support, N.I.H. Extramural Validation Studies. *Exp Clin Psychopharmacol*. 2010; 18(5):409–17. [PubMed: 20939644]
55. Day A, Celio M, Lisman S, Spear L. Gender, history of alcohol use and number of drinks consumed predict craving among drinkers in a field setting. *Addict Behav*. 2014; 39(1):354–7. [PubMed: 24148138]

56. Domingues S, Mendonça J, Laranjeira R, Nakamura-Palacios E. Drinking and driving: a decrease in executive frontal functions in young drivers with high blood alcohol concentration. *Alcohol*. 2009; 43(8):657–64. [PubMed: 20004344]
57. Weafer J, Fillmore MT, Milich R. Increased sensitivity to the disinhibiting effects of alcohol in adults with ADHD. [Controlled Clinical Trial Research Support, N.I.H. Extramural]. *Exp Clin Psychopharmacol*. 2009; 17(2):113–21. [PubMed: 19331488]
58. Fillmore MT, Ostling EW, Martin CA, Kelly TH. Acute effects of alcohol on inhibitory control and information processing in high and low sensation-seekers. *Comparative Study Research Support, N.I.H. Extramural Drug Alcohol Depend*. 2009; 100(1–2):91–9. [PubMed: 19004578]
59. 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. [Comparative Study Research Support, N.I.H. Extramural] *Alcoholism. Clin Exp Res*. 2006; 30(8):1363–71.
60. 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. *Clinical Trial Comparative Study Randomized Controlled Trial Research Support, Non-U.S. Gov't. Alcohol Clin Exp Res*. 2003; 27(5):773–9. [PubMed: 12766621]
61. Rose AK, Duka T. Effects of alcohol on inhibitory processes. *Behav Pharmacol*. 2008; 19(4):284–91. [PubMed: 18622175]
62. Saults JS, Cowan N, Sher KJ, Moreno MV. Differential effects of alcohol on working memory: Distinguishing multiple processes. *Exp Clin Psychopharmacol*. 2007; 15(6):576–87. [PubMed: 18179311]
63. Marczinski CA, Abroms BD, Van Selst M, Fillmore MT. Alcohol-induced impairment of behavioral control: differential effects on engaging *vs* disengaging responses. *Psychopharmacology (Berl)*. 2005; 182(3):452–9. [Research Support, N.I.H., Extramural Research Support, U.S. Gov't, P.H.S.]. [PubMed: 16075287]
64. Hedden T, Gabrieli JD. Shared and selective neural correlates of inhibition, facilitation, and shifting processes during executive control. *Neuroimage*. 2010; 51(1):421–31. [PubMed: 20123030]
65. Robbins TW. Shifting and stopping: fronto-striatal substrates, neurochemical modulation and clinical implications. *Philosophical Transactions of the Royal Society London Series B, Biological Sciences*. 2007; 362(1481):917–32.
66. McCabe DP, Roediger HL, McDaniel MA, Balota DA, Hambrick DZ. The relationship between working memory capacity and executive functioning: evidence for a common executive attention construct. *Neuropsychol*. 2010; 24(2):222–43.
67. Hogan, TP. 50 widely used psychological tests. In: Koocher, GP.; Norcross, JC.; Hill, SS., III, editors. *Psychologists' Desk Reference*. 2. New York: Oxford University Press; 2005. p. 101-104.
68. Tate RL, Perdices M, Maggioletto S. Stability of the Wisconsin Card Sorting Test and the determination of reliability of change in scores. *Clin Neuropsychol*. 1998; 12:348–57.
69. Basso MR, Bornstein RA, Lang JM. Practice effects on commonly used measures of executive function across twelve months. *Clin Neuropsychol*. 1999; 13:283–92. [PubMed: 10726600]
70. Mitroshina, M.; Boone, KB.; Razani, J.; D'Elia, LF. *Handbook of normative data for neuropsychological assessment*. 2. New York: Oxford; 2005.
71. Ahern, DC.; Faust, D. Challenging computerized testing and computer-based test interpretations. In: Faust, D., editor. *Coping with psychiatric and psychological testimony*. 6. 2012. p. 470-87.
72. Standing L, Conezio J, Haber RN. Perception and memory for pictures: Single trial learning of 2500 visual stimuli. *Psychonomic Sci*. 1970; 19(2):73–4.
73. Tombaugh T. The Test of Memory Malingering (TOMM): Normative data from cognitively intact and cognitively impaired individuals. *Psychol Assessment*. 1997; 9(3):260–8.
74. Dovis S, Van der Oord S, Wiers R, Prins P. ADHD subtype differences in reinforcement sensitivity and visuospatial working memory. *J Clin Child Adol Psychol*. 2014:1–16.
75. Koekkoek S, de Sonnevill LM, Wolfs TF, Licht R, Geelen SP. Neurocognitive function profile in HIV-infected school-age children. *Eur J Paediatr Neurol*. 2008; 12(4):290–7.

76. Raffa RB. Cancer 'survivor-care': II. Disruption of prefrontal brain activation top-down control of working memory capacity as possible mechanism for chemo-fog/brain (chemotherapy-associated cognitive impairment). *J Clin Pharm Therap.* 2013; 38(4):265–8. [PubMed: 23656522]
77. Heather N, Tebbutt JS, Mattick RP, Zamir R. Development of a scale for measuring impaired control over alcohol consumption: A preliminary report. *J Studies Alcohol.* 1993; 54(6):700–9.
78. Leeman RF, Patock-Peckham JA, Potenza MN. Impaired control over alcohol use: An under-addressed risk factor for problem drinking in young adults? *Exp Clin Psychopharm.* 2012; 20(2): 92–106.
79. Hagman BT, Cohn AM. Drinking correlates of DSM-IV alcohol use disorder diagnostic orphans in college students. *Am J Addic.* 2012; 21(3):233–42.
80. Kahler CW, Strong DR. A Rasch model analysis of DSM-IV alcohol abuse and dependence items in the national epidemiological survey on alcohol and related conditions. *Alcoholism: Clin Exp Res.* 2006; 30(7):1165–75.
81. Kahler CW, Strong DR, Read JP. Toward efficient and comprehensive measurement of the alcohol problems continuum in college students: The Brief Young Adult Alcohol Consequences Questionnaire. *Alcoholism: Clin Exp Res.* 2005; 29(7):1180–9.
82. Krueger RF, Nichol PE, Hicks BM, et al. Using latent trait modeling to conceptualize an alcohol problems continuum. *Psychol Assessment.* 2004; 16(2):107–19.
83. Sweet LH, Rao SM, Primeau M, Durgerian S, Cohen RA. Functional magnetic resonance imaging response to increased verbal working memory demands among patients with multiple sclerosis. *Human Brain Mapping.* 2006; 27(1):28–36. [PubMed: 16001441]
84. Nixon SJ. Executive functioning among young people in relation to alcohol use. *Curr Opin Psychiat.* 2013; 26(4):305–9.
85. Cahalan, D.; Cisin, I.; Crossley, H. American drinking practices: A national study of drinking behavior and attitudes. New Haven, Connecticut: College & University Press, Rutgers Center of Alcohol Studies; 1969. (Monograph No. 6)
86. Norman AL, Pulido C, Squeglia LM, Spadoni AD, Paulus MP, Tapert SF. Neural activation during inhibition predicts initiation of substance use in adolescence. *Drug Alcohol Depend.* 2013; 119(3): 216–23. [PubMed: 21782354]
87. Morgenstern J, Frey RM, McCrady BS, Labouvie E. Examining mediators of change in traditional chemical dependency treatment. *J Studies Alcohol.* 1995; 57(1):53–64.
88. Casbon TS, Curtin JJ, Lang AR, Patrick CJ. Deleterious effects of alcohol intoxication: Diminished cognitive control and its behavioral consequences. *J Abnormal Psychol.* 2003; 112(3): 476–87.
89. Grattan-Miscio KE, Vogel-Sprott M. Effects of alcohol and performance incentives on immediate working memory. [Randomized Controlled Trial Research Support, Non-U.S. Gov't]. *Psychopharmacol (Berl).* 2005; 181(1):188–96.
90. 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. *Neuropsychopharmacol.* 2006; 31(6):1301–9.
91. Tiplady B, Oshinowo B, Thomson J, Drummond GB. Alcohol and cognitive function: assessment in everyday life and laboratory settings using mobile phones. [Research Support, Non-U.S. Gov't]. *Alcohol Clin Exp Res.* 2009; 33(12):2094–102. [PubMed: 19740132]
92. Weissenborn R, Duka T. Acute alcohol effects on cognitive function in social drinkers: their relationship to drinking habits. [Clinical Trial Comparative Study Randomized Controlled Trial Research Support, Non-U.S. Gov't]. *Psychopharmacology (Berl).* 2003; 165(3):306–12. [PubMed: 12439627]
93. Cromer J, Cromer J, Maruff P, Snyder P. Perception of alcohol intoxication shows acute tolerance while executive functions remain impaired. *Exp Clin Psychopharmacol.* 2010; 18(4):329–39. [PubMed: 20695689]
94. Montgomery C, Ashmore KV, Jansari A. The effects of a modest dose of alcohol on executive functioning And prospective memory. *Hum Psychopharmacol: Clin Exper.* 2011; 26(3):208–15.
95. Lyvers MF, Maltzman I. Selective effects of alcohol on Wisconsin Card Sorting Test performance. *Br J Addic.* 1991; 86(4):399–407.

96. Christiansen P, Cole JC, Field M. Ego depletion increases ad-lib alcohol consumption: Investigating cognitive mediators and moderators. *Exp Clin Psychopharmacol*. 2012; 20(2):118–28. [PubMed: 22182418]
97. Birak KS, Terry P, Higgs S. Effect of cues associated with an alcoholic beverage on executive function. *J Studies Alcohol Drugs*. 2010; 71(4):562–9.
98. Lyvers M, Tobias-Webb J. Effects of acute alcohol consumption on executive cognitive functioning in naturalistic settings. [Research Support, Non-U.S Gov't]. *Addict Behav*. 2010; 35(11):1021–8. [PubMed: 20655148]
99. Tsujii T, Sakatani K, Nakashima E, Igarashi T, Katayama Y. Characterization of the acute effects of alcohol on asymmetry of inferior frontal cortex activity during a Go/No-Go task using functional near-infrared spectroscopy. [Randomized Controlled Trial Research Support, Non-U.S. Gov't]. *Psychopharmacol (Berl)*. 2011; 217(4):595–603.
100. Marcziński CA, Fillmore MT. Compensating for alcohol-induced impairment of control: effects on inhibition and activation of behavior. [Comparative Study Research Support, N.I.H., Extramural Research Support, U.S. Gov't, P.H.S.]. *Psychopharmacol (Berl)*. 2005; 181(2):337–46.
101. Marinkovic K, Rickenbacher E, Azma S, Artsy E. Acute alcohol intoxication impairs top-down regulation of Stroop incongruity as revealed by blood oxygen level-dependent functional magnetic resonance imaging. [Research Support, N.I.H., Extramural Research Support, Non-U.S. Gov't]. *Hum Brain Mapp*. 2012; 33(2):319–33. [PubMed: 21391268]
102. Abrams BD, Fillmore MT, Marcziński CA. Alcohol-induced impairment of behavioral control: Effects on the alteration and suppression of prepotent responses. *J Studies Alcohol*. 2003; 64(5): 687–95.
103. Weafer J, Fillmore MT. Individual differences in acute alcohol impairment of inhibitory control predict ad libitum alcohol consumption. [Clinical Trial Research Support, N.I.H. Extramural]. *Psychopharmacol (Berl)*. 2008; 201(3):315–24.
104. Fillmore MT, Marcziński CA, Bowman AM. Acute tolerance to alcohol effects on inhibitory and activational mechanisms of behavioral control. *J Studies Alcohol*. 2005; 66(5):663–72.
105. Fillmore MT, Blackburn JS, Harrison EL. Acute disinhibiting effects of alcohol as a factor in risky driving behavior. [Randomized Controlled Trial Research Support, N.I.H. Extramural]. *Drug Alcohol Depend*. 2008; 95(1–2):97–106. [PubMed: 18325693]
106. Fillmore M, Weafer J. Acute tolerance to alcohol in at-risk binge drinkers. *Psychol Addict Behav*. 2012; 26(4):693–702. [PubMed: 22023021]
107. Ostling EW, Fillmore MT. Tolerance to the impairing effects of alcohol on the inhibition and activation of behavior. *Psychopharmacol*. 2010; 212(4):465–73.
108. Loeber S, Duka T, Welzel H, Nakovics H, Heinz A, Flor H, Mann K. Impairment of cognitive abilities and decision making after chronic use of alcohol: The impact of multiple detoxifications. *Alcohol Alcoholism*. 2009; 44(4):372–81. [PubMed: 19487491]
109. Petrides M, Milner B. Deficits on subject-ordered tasks after frontal- and temporal-lobe lesions in man. *Neuropsychol*. 1982; 20(3):249–62.

Table 1
Effect of executive functioning on alcohol use initiation, frequency, and intensity (N=15 studies).

Authors	Design	N	Age (Years) M (SD)	EF Task(s)/Metric	Alcohol-Related DV	Finding
Updating	(n=6)					
Noël <i>et al.</i> , 2002 [46]	Longitudinal 3 mo. F/U	20	45.5 (7.5)	Alpha Span task		Individuals who ultimately relapsed had done more poorly on alphabetical portion of task
Thush <i>et al.</i> , 2008 [40]	Longitudinal 1 mo. F/U	81	16.34 (1.34)	WMC x implicit and explicit positive arousal expectancies	Alcohol use 1 month later	Implicit associations predict alcohol use for those with low WM; explicit associations predict alcohol use for those with high WM
Friese <i>et al.</i> , 2010 [41]	Cross-sectional	49	All males 25.2 (7.22)	Operation Span Task	Average fixation length; total dwell time; NOT 1 st fixation length or time to 1 st fixation	Implicit associations predict attention allocation for those with low WM capacity; not for those with high WM capacity
Pharo <i>et al.</i> , 2011 [39]	Cross-sectional	69 M, 67 F adolescents; 27 M, 30 F adults	Adol: 15.86 (1.06); Adults: 18-22 (19.8, 1.41)	COWAT; Digit Span	Risk-taking composite score (alcohol was on component)	Poorer performance on WM tasks was related to greater risk-taking
Houben <i>et al.</i> , 2011 [43]	Longitudinal 1 mo. training and 1 mo. F/U	48	44.3 (15.4)	SOPT; Backward Digit Span; Letter Span Task	AUDIT	WM training decreased alcohol use for those with high IAT
* Squeglia <i>et al.</i> , 2012, Study 2 [36]	Longitudinal 3 year F/U	40	12-16 at baseline assessment	fMRI activation in frontal and parietal areas during a visual WM task	Transition to heavy drinking (defined by [85] and modified for adolescents)	Lower baseline activation predicted transition to heavy drinking
Shifting	(n=1)					
Mullan <i>et al.</i> , 2011 [38]	Longitudinal 1 week F/U	153	20.1 (4.2)	Tower of Hanoi; Stroop; ICT; WCST	Binge drinking in past week	Poorer shifting was associated with increased drinking among those with intentions to drink
Inhibition	(n=10)					
Morgenstern & Bates, 1999 [48]	Longitudinal ^a	118	35.6 (9.1)	Composite score: SILS, TMT-Part B, WCST, FAS, CAT	Treatment outcome	EF did not predict treatment outcome
Moriyama <i>et al.</i> , 2002 [49]	Longitudinal 18 month F/U	37	51.6 (3.7)	Reaction time, Symbol Digit, Digit Span, Figure Position, TMT, Rule Shift, and other non-EF tasks	Drinking outcome (DSM-III-R alcohol-related problems)	EF did not predict drinking outcome but did predict occupational outcome
Noël <i>et al.</i> , 2002 [46]	Longitudinal 3 month F/U	20	45.5 (7.5)	Hayling task		Abstainers made fewer errors

Authors	Design	N	Age (Years) M (SD)	EF Task(s)/Metric	Alcohol-Related DV	Finding
Nigg <i>et al.</i> , 2006 [12]	Longitudinal 3 year F/U	498	12–14 or 15–17 at baseline	Stop Signal Task No influence of WCST	Alcohol problems, comorbid alcohol and drug use	Poor stop signal performance predicted alcohol problems, comorbid use
Patrick <i>et al.</i> , 2008 [42]	Cross-sectional	72	All females 21.1 (0.8)	N-back, GNG	Recent alcohol use	Among those with poorer performance on GNG, IGT predicted alcohol use; for those with better N-Back performance, BAS predicted alcohol use
Pharo <i>et al.</i> , 2011 [39]	Cross-sectional	69 M, 67 F adolescents; 27 M, 30 F adults	Adol: 15.86 (1.06); Adults: 18–22 yo (19.8, 1.41)	Stroop	Risk-taking composite score (alcohol was one component)	Poorer performance on Stroop task was related to greater risk- taking
Mullan <i>et al.</i> , 2011 [38]	Longitudinal 1 week F/U	153	20.1 (4.2)	Tower of Hanoi; Stroop; IGT; WCST	Binge drinking in past week	Poorer inhibition was associated with increased drinking among those with intentions to drink
Camchong, Stenger & Fein, 2012 [47]	Longitudinal 6 mo. F/U	69	Abstainers: 46.7 (6.8); Relapsers: 46.9 (7.25)	Affective GNG	Relapse to alcohol/drugs	No difference in GNG; relapsers had lower RSS in executive network
* Norman <i>et al.</i> , 2011 [86]	Longitudinal 5 year F/U	38	12–14 at baseline	GNG in fMRI	Transition to heavy alcohol use	Reduced activation in several brain regions, including frontal regions, predicted transition to heavy drinking
Houben <i>et al.</i> , 2012 [44]	Longitudinal 1 week F/U	57	20.91 (1.83)	GNG	Alcohol use in past week	Training of “no-go” response decreased alcohol use <i>via</i> change in implicit associations
* Wetherill <i>et al.</i> , 2013 [37]	Longitudinal 5 year F/U	60	12–14 at baseline; M=13.3	GNG in fMRI	Alcohol-induced blackouts	Greater activation in frontal cortices predicted alcohol- induced blackouts

Note: WM=working memory; COWAT=Controlled Oral Word Association Test; SOPT=Self-ordered pointing task; TMT= Trail Making Test; AUDIT=Alcohol Use Disorders Identification Test; fMRI=functional magnetic resonance imaging; IAT=Implicit Associations Task; IGT=Iowa Gambling Task; WCST=Wisconsin Card Sorting Task; GNG=Go/No-Go; BAS=behavioral activation system; SILS=Vocabulary Test from the Shipley Institute of Living Scale; FAS=Phonemic Word Fluency Test; RSS=resting state synchrony; CAT=Booklet Category Test; F/U=follow up;

* studies that utilize alcohol-naïve adolescents.

$a_{2,61} \pm 13.6$ days [87].

Table 2

Acute effects of alcohol on executive functioning (N=35).

Study	N	Age (Years): M (SD)	Dose	Alcohol Did Affect	Task	Did Not Affect	Task
Updating	(n=13)						
Boha <i>et al.</i> , 2009 [53]	32	22(2.3)	0.2 g/kg or 0.4 g/kg	(1) WM RT ^l (2) WM Correct Responses (+) ^l	Arithmetic task in scanner		
Fillmore <i>et al.</i> , 2009 [58]	10 M 10 W	23.2 (3.1)	0.0, 0.45, 0.65 g/kg	WM ^k	Number identification task		
Cashon <i>et al.</i> , 2003 [88]	32 undergrads	22.8 (2.3) in alcohol condition, 23 (2.3) in no-alcohol condition	Peak 0.06%	Perseveration ^b	N-Back Task		(1) Spatial Span Task (2) Baddeley's Reasoning Task
Rose & Duka, 2008 (Study 2) [61]	32 social drinkers	21.7, SD not reported	0.6 g/kg	(1) Reaction time in STMC ^{b,i} (2) Errors in STMC ^{b,c} (3) Scanning time in STMC ^{b,c}	SMS		
Grattan-Miscio & Vogel-Sprott, 2005 [89]	53 M 20 W	Range: 19–25, M(SD) not reported	0.62 g/kg (M); 0.54 g/kg (W)	(1) Long-term verbal memory ^g (2) Short-term visual memory (3) Long-term visual memory ^g (<i>p</i> =.08) (4) Visuospatial WM (5) Information Processing (6) Explicit memory	(1) Word Discrimination (2) Design Discrimination (3) Xs and Os ^c (4) Symbol Matching (5) Symbol Matching without a key	(1) Short-term verbal memory (2) Immediate WM	(1) Word Discrimination (2) Three Letters
Schweitzer <i>et al.</i> , 2006 [90]	20 M undergrads	21.8 (2.2)	0.65 g/kg alcohol	(1) Auditory WM (sequential presentation) (2) Visuospatial WM (sequential presentation)	(1) Sound presentation (2) Dot presentation	(1) Auditory WM (simultaneous presentation) (2) Visuospatial WM (simultaneous presentation)	(1) Sound presentation (2) Dot presentation
Saults <i>et al.</i> , 2007 [62]	36 M 36 W	Range: 21–30, M(SD) not reported	0.72 g/kg (M); 0.65 g/kg (F)	(1) Auditory WM (sequential presentation) (2) Visuospatial WM (sequential presentation)	(1) Sound presentation (2) Dot presentation	(1) Auditory WM (simultaneous presentation) (2) Visuospatial WM (simultaneous presentation)	(1) Sound presentation (2) Dot presentation

Study	N	Age (Years): M (SD)	Dose	Alcohol Did Affect	Task	Did Not Affect	Task
Paulus <i>et al.</i> , 2006 [59]	6 M 4 W	23.2 (0.9)	0.75 mL/kg (M); 0.68 mL/kg (F)			Visuospatial WM	fMRI task (2,4,6 colored dots)
Pihl <i>et al.</i> , 2003 [60]	41 social drinkers	20.85 (1.82) in alcohol condition, 20.2 (1.79) in placebo condition	Test at 0.08%	Acquired Association ^c	Acquired Spatial Association Task	(1) Non-spatial association (2) Visuospatial WM	(1) Acquired Non-Spatial Association Task (2) Random Object Span Task (like SOPT)
Tiplady <i>et al.</i> , 2009 [91]	30	22.8, no SD reported in everyday condition, 23.1, no SD reported in lab condition	M: 0.8 g/kg F: 0.7 g/kg	STMC	(1) Memory Scanning Task (2) Number Pairs		
Weissenborn & Duka, 2003 [92]	95 social drinkers	21.8 (SEM=0.3)	0.8 g/kg F: Mean=0.61 g/l M: Mean=0.56 g/l			Visuospatial WM	Self-Ordered Pointing Task
Finn <i>et al.</i> , 1999 [10]	69 M 80 W	FHP: 23.1 (2.9) FHN: 22.2 (1.8)	0.07% or 0.09%	Auditory WM ^a	Digit Span Backward (WAIS-R)		
Cromer <i>et al.</i> , 2010 [93]	20 social drinkers	22.8 (1.1)	0.10%	Visuospatial WM	Groton Maze Learning Test		
Shifting	(n=9)						
Montgomery <i>et al.</i> , 2011 [94]	40 social drinkers	20.15, no SD reported in alcohol condition; 19.4, no SD in placebo condition	0.4 g/kg	Planning	Jansari-Agnew-Akesson-Murphy (JAAM) task		
Lyvers & Maltzman, 1991 [95]	45 M 45 W	Range: 21–30, M(SD) not reported	0.05%	Perseveration Set-shifting	WCST		
Christiansen, Rose, Cole, & Field, 2012 [96]	80 undergrads	22.08 (4.53)	0.65 g/kg alcohol	Word generation	COWAT		
Brak, <i>et al.</i> , 2010 [97]	45 undergrads	20.5 (3.0)	0.65 g/kg (M); 0.57 g/kg (F)	Set-Shifting ^f	Shape Size Choice Task		
Weissenborn & Duka, 2003 [92]	95 social drinkers	21.8 (SEM=0.3)	0.8 g/kg F: Mean=0.61 g/l M: Mean=0.56 g/l	Planning	Tower of London		

Study	N	Age (Years): M (SD)	Dose	Alcohol Did Affect	Task	Did Not Affect	Task
Guillot <i>et al.</i> , 2010 [54]	94 M 91 W	25.6 (6.5)	.00%, .05%, .075%, or .10%	Perseveration ^d Set-Shifting (+) ^e	WCST TMT-B		Day <i>et al.</i>
Domingues <i>et al.</i> , 2009 [56]	96 tested with alcohol	Not reported	.01% - "over .06%"			Conceptualization, Mental Flexibility, Sensitivity to Interference, Environmental Autonomy	Frontal Assessment Battery
Day <i>et al.</i> , 2014 [55]	91	Men: 19.4 (0.78) Women: 19.3 (0.77)	0% - 0.29%	Set-Shifting	TMT-B, TMT Composite (B-A)	Attention	TMT-A
Lyvers & Tobias-Webb, 2010 [98]	86 bar patrons	22.1 (3.2)	0% - 0.15%	Perseveration	WCST PE	NPE	WCST
Inhibition	(n=20)						
Tsuji <i>et al.</i> , 2011 [99]	32	28.2 (5.05)	0.5 g/kg	Response Inhibition (RT and False Alarms)	Visual GNG in scanner		
Filmore <i>et al.</i> , 2009 [58]	10 M 10 W	23.2 (3.1)	0.0, 0.45, 0.65 g/kg	Response inhibition ^o	Cued GNG		
Marczynski <i>et al.</i> , 2005 [63]	12 M 12 W	23.4 (2.4)	0.0, 0.45, 0.65 g/kg	(1) Commission Errors/ (1) Reaction time ^k	(1) Eng. GNG (2) Diseng. GNG	Commission Errors	Disengagement GNG
Marczynski & Fillmore, 2005 [100]	9 M 8 W	23.5 (2.7)	0.0, 0.45, 0.65 g/kg	(1) Response inhibition (RT and Failures to Inhibit on No-Go)	Cued GNG		
Marinkovic <i>et al.</i> , 2012 [101]	10 M 10 W	24.9 (3.6)	Test between .04-.05%	(2) Reaction times (3) Accuracy on incongruent trials ($p=.07$)	Stroop task in fMRI		
Rose & Duka, 2008 (Study 1) [61]	32 social drinkers	21.3, no SD reported	0.6 g/kg	Inhibition of interference	Stroop Task		
Schweitzer <i>et al.</i> , 2006 [90]	20 M undergrads	21.8 (2.2)	0.65 g/kg alcohol	Response inhibition	Stroop GNG		
Abrams <i>et al.</i> , 2003 [102]	29 M 11 W	22.6 (1.6)	0.65 g/kg	Response inhibition	Cued GNG	Response alteration	Cued task: choice of two "go" options

Study	N	Age (Years): M (SD)	Dose	Alcohol Did Affect	Task	Did Not Affect	Task
Weafer <i>et al.</i> , 2009 [57]	10 ADHD 12 Control	Control: 22.8 (1.1); ADHD: 22.8 (1.8)	0.65 g/kg	Response inhibition, particularly for ADHD	Cued GNG		
Weafer & Fillmore, 2008 [103]	14 M 12 W	21.9 (1.4)	0.65 g/kg	Response inhibition	Cued GNG		
Fillmore <i>et al.</i> , 2005 [104]	12 M 8 W	21.5 (1.0)	0.65 g/kg	(1) RT to Response inhibition/ (2) Response inhibition ^W	Cued GNG		
Fillmore <i>et al.</i> , 2008 [105]	7 M 7 W	23.5 (3.2)	0.65 g/kg	Response inhibition ^W	Cued GNG		
Fillmore & Weafer, 2012 [106]	20 M 20 W	23.1 (2.9)	0.65 g/kg	Response inhibition ^W	Cued GNG		
Ostling & Fillmore, 2010 [107]	32 adults	22.9 (2.4)	0.65 g/kg	(1) Response activation (2) Response inhibition	Cued GNG		
Birak, <i>et al.</i> , 2010 [97]	45 undergrads	20.5 (3.0)	0.65 g/kg (M); 0.57 g/kg (F)	(1) Response Inhibition (+) (2) Latency in RT (+)	Affective GNG		
Domingues <i>et al.</i> , 2009 [56]	96 tested with alcohol	Not reported	.01% - ".over .06%"			Inhibitory Control	Frontal Assessment Battery
Tiplady <i>et al.</i> , 2009 [91]	30	22.8, no SD reported in everyday condition, 23.1, no SD reported in lab condition	M: 0.8 g/kg F: 0.7 g/kg	Response Inhibition (RT & False Positives)	Visual GNG	Response Inhibition (False Negatives)	Visual GNG
Loeber & Duka, 2009 [108]	16 M 16 W	Alcohol: 21.3 (3.6) Placebo: 20.5 (3.4)	0.8 g/kg	Stop Signal RT	Stop Signal Task		
Finn <i>et al.</i> , 1999 [10]	69 M 80 W	FHP: 23.1 (2.9) FHN: 22.2 (1.8)	0.07% or 0.09%	(1) Response Inhibition (2) Approach (+)	(1) GNG False Alarm (2) GNG Hit Rates		
Guillot <i>et al.</i> , 2010 [54]	94 M 91 W	25.6 (6.5)	.00%, .05%, .075%, or .10%			Response Inhibition	GoStop Task

Note: (+)=participants did better rather than worse; in N column: M=men, W=women;

- ^a only for those high in WM;
- ^b under high WM load;
- ^c only on descending limb;
- ^d High dose performed more poorly on WCST PE and TE, Med dose performed more poorly on PE;
- ^e placebo & low dose performed better than at BL;
- ^f only in unfamiliar alcohol drink condition;
- ^g after 20 minute delay;
- ^h under conflict;
- ⁱ on ascending limb;
- ^j following invalid go cues;
- ^k only at 0.65 g/kg;
- ^l only for low dose (0.2 g/kg);
- ^m to no-go cues;
- ⁿ for both at-risk and no-risk drinkers;
- ^o among high sensation-seekers;

WCST = Wisconsin Card Sorting Task; WM = Working Memory; CANTAB = Cambridge Neuropsychological Test Automated Batteries; PM = Prospective Memory; JAAM = Jansari-Agnew-Akesson-Murphy Task; SOPT = Self-Ordered Pointing Task; COWAT = Controlled Oral Word Association Test; GNG = Go/No-Go; SMS = Sternberg Memory Scanning Task.

Table 3

Tasks of Executive Functioning.

Task	Construct Measured	Description	Age Ranges ¹	Administration
Wisconsin Card Sorting Task	Set Shifting, Rule Acquisition	Based on examiner feedback, examinees must learn an adapting set of rules to correctly sort the test stimuli	5 – 89 years	15 – 30 minute administration. Computerized version available. Task may be susceptible to practice effects in higher functioning individuals.
Trail Making Test	Set Shifting (set-shifting is predominant in Part B only, Part A is more an assessment of attention)	Examinees must quickly connect in order, a randomly distributed series of numbers (Part A) or letters and numbers (Part B) on a page	9 – 89 years	~5 minute administration.
Mental Arithmetic from WAIS-III	Working Memory (verbal)	Arithmetical word problems are presented orally to examinees who must solve the word problem without use of paper or pencil	16 – 89 years	3 – 8 minute administration
Self-Ordered Pointing Task [109]	Working Memory (visuospatial), Self-Regulation	Examinees must point to objects presented in a series of layouts without pointing to the same object twice	7 – 65 years (not inclusive for all ages in the range)	20 minute administration. Distributed by Millisecond Software for a fee.
Tower of London/Hanoi/Tower Test	Planning/Inhibition	Discs or beads must be moved under a set of constraining rules to replicate a series of patterns	Numerous versions exists with norms from early childhood to late adulthood	10 – 15 minute administration
Iowa Gambling Task	Planning	Examinees draw from decks of cards that differ in their level of reward/penalty, and must determine which deck offers the best odds to maximize winnings	18 – 79 years	15 – 20 minute administration. Computerized administration only.
Go/No-Go	Response Inhibition	A series of different tasks in which examinees must respond to one stimuli but withhold response to another stimuli	All ages due to qualitative nature of task	Variable stimuli and administration times. Often interpreted qualitatively.
Stop Signal	Response Inhibition	Examinees are required to initiate a motor sequence and stop the behavior at a signal, with reaction time as the target variable	Varies. CANTAB version 4 – 90 years	Variable stimuli and administration times. Computerized versions available.
Stroop Task	Response Inhibition; Resistance to Interference	A list of color names printed in discordant ink colors is presented to the examinee, who must ignore the words and identify the ink colors as quickly as possible	Varies by version within 5 – 94 years.	5 minute administration.
Controlled Order Word Association Test (COWAT)	Mental Flexibility; Set Maintenance	Examinees rapidly list words beginning with a target letter while avoiding proper nouns and variants involving suffixes	Varies by version within 6 – 95 years.	5 minute administration
Ruff Figural Fluency Test	Mental Flexibility	Examinees draw as many unique designs as possible	7 – 70 years	10 minute administration.

Task	Construct Measured	Description	Age Ranges ¹	Administration
		within a time limit by connecting dots in a matrix		
Porteus Maze Test	Planning	A path must be traced through a series of mazes without back-tracking	3+ years	15 – 60 minute administration.
CANTAB	Collection of tests	22 tests of various aspects of executive functioning administered on a computer touch-screen	4 – 90 years	2 – 10 minutes administration per subtest; each subtest can be administered individually. Computerized administration only.
Groton Maze Learning Test	Planning; Set Maintenance	Examinees must discover a hidden pathway through a computerized grid by following a set of rules	6 – 106 years	Portion of the Cogstate computerized test battery. www.cogstate.com
EXAMINER	Collection of tests	Includes 11 computerized and paper-and-pencil tests (assessing working memory, cognitive control, and fluency); administered independently or as a complete battery	3 – 90 years (with a few exceptions noted in the manual for some tests)	English and Spanish versions are available, with 3 alternate forms for each version. Tests are freely available: www.memory.ucsf.edu/resources/examiner
Delis-Kaplan Executive Function System (D-KEFS)	Collection of tests	9 tests of various aspects of executive functioning; tests can be administered independently or as a complete battery	8 – 89 years	90 minute administration for the full battery; some tests have alternate forms.

Note: For elaborate descriptions of the tasks, including psychometric data, normative data, and administration guidelines, see Strauss, Sherman, & Spreen (2006) or Lezak *et al.* (2012). Comprehensive descriptions and reviews can also be found in the Mental Measurements Yearbooks published by the Buros Institute

¹The age ranges provided here are for the most commonly available norms. Note that the quality of normative data may vary by age range and normative data for additional ages may be available in the research literature.