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Cognitive and Functional Deficits of Alcohol Abuse: A Review

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Due to the high prevalence of alcohol abuse and alcohol-related mortalities worldwide, it is important to investigate the impact of alcohol abuse on cognitive and functional deficits. The aim of this review is to synthesize the disparate research on cognitive and functional deficits due to alcoholism and delineate the different moderating factors that affect these deficits. The review of the literature suggests that the relationship between alcohol abuse and cognitive and functional deficits is multifaceted and is moderated by age, gender, dose, and prior treatment history. The review also indicates limitations of the cited studies and suggests multiple directions for future research.

Alcoholism (i.e., Alcohol Use Disorders) is the third leading cause of preventable death in the United States, and the lifespan of people who suffer from alcoholism is roughly 12 years shorter than their non-alcoholic counterparts (Maddux & Winstead, 2008). According to the World Health Organization (2008), alcohol use disorders cause 88 million deaths yearly. In addition, 125 million people worldwide are estimated to have alcohol use disorders.

Based on the Diagnostic and Statistical Manual, 4th Edition (American Psychiatric Association [DSM-IV-TR], 2000) alcohol use disorders include both abuse and dependence. Alcohol abuse refers to a maladaptive pattern of alcohol use that is manifested through recurrent alcohol use in dangerous situations (e.g., driving drunk, operating machinery while intoxicated), despite legal, social or interpersonal problems, and resulting in a failure to fulfill major role obligations. Alcohol dependence is defined by a maladaptive pattern of alcohol use manifested by at least three of the following: tolerance, withdrawal, larger consumption of alcohol than intended, unsuccessful efforts to cut down use, a great deal of time spent on obtaining the alcohol or using it, important social/occupational/recreational activities being given up or reduced because of alcohol use, and continued use despite physical or psychological problems attributed to alcohol use. Tolerance is described as the need for significantly increased amount of alcohol to achieve intoxication or desired effect and diminished effect with continued use of the same amount of alcohol. Alcohol withdrawal symptoms include hand tremors, headache, seizures, vomiting, and nausea (Lessa & Scanlon, 2006). Alcohol withdrawal is also characterized by the consumption of more alcohol or a substance similar to it in order to avoid withdrawal symptoms. Recreational alcohol use does not involve a maladaptive relationship with alcohol like alcohol dependence and abuse.

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Chronic consumption of alcohol has been found to be associated with cognitive deficits, including difficulties learning new information (McGlinchey-Berroth, Fortier, Cermak, & Disterhoft, 2002; Ryan & Butters, 1980; Schottenbauer, Hommer, & Weingartner, 2007), problems with retaining information over periods of long delay (Rose, Shaw, Prendergast, & Little, 2010), and impairment in prospective memory (Heffernan, Moss, & Ling, 2002; Ling et al., 2003), verbal and non-nonverbal contextual memory (Sullivan, Shear, Zipursky, Sagar, & Pfefferbaum, 1997), short-term memory, general memory, and verbal memory (Ryan & Butters, 1980; Rosenbloom, O'Reilly, Sassoon, Sullivan, Pfefferbaum, 2005; Sullivan, Rosenbloom, & Pfefferbaum, 2000; Sullivan, Fama, Rosenbloom, & Pfefferbaum, 2002). It has also been found to be associated with functional deficits in the areas of visuospatial abilities, upper limb mobility, and gait and balance (Sullivan et al., 2000; Sullivan et al., 2002). In addition, alcohol abuse has been found to negatively impact executive functioning, decision-making, behavioral inhibition, task shifting, working memory, and problem solving (Hildebrandt, Eling, Brokate, & Lanz, 2004; Leckliter & Metarazzo, 1989; Noel, Bechara, Dan, Hanak, & Verbanck, 2007; Sullivan et al., 2000; Sullivan et al., 2002),

Additionally, research suggests that abstinence allows for recovery of some cognitive impairments associated with alcohol abuse (e.g., Brandt, Butters, Ryan, & Bayog, 1983; Bates, Voelbel, Buckman, Labouvie, & Barry, 2005; Mann, Gunther, Stetter, & Ackerman, 1999; Rosenbloom, Rohlfing, O'Reilly, Sassoon, Pfefferbaum, & Sullivan, 2007). Bates et al. (2005) found a modest recovery in the cognitive domains of executive functioning, verbal processing speed, and verbal ability domains, and a medium recovery in the memory domain of abstaining alcoholics. Rosenbloom and colleagues (2007) found improvements on measures of general memory and ataxia in abstaining alcoholics. Brandt and colleagues (1983) showed that alcoholics with a prolonged period of abstinence (more than 5 years) improved in short-term memory and non-verbal memory skills. In addition, Mann and colleagues (1999) found significant improvement on measures of perceptual-motor speed, verbal knowledge, non-

verbal reasoning, and spatial imagination in alcoholics who abstained from alcohol for a 5-week period. The aforementioned studies will be explored in more details further along in this review.

Effects of Alcoholism on Memory

Prospective Memory

Prospective memory is an important part of everyday cognitive functioning and is defined as the type of memory in which a person has to remember to do something in the near future, similar to a mental "to do" list (Heffernan et al., 2002). There is evidence that individuals with heavy alcohol use have impaired prospective memory compared to individuals with low-level alcohol use. An alcohol abuser with deficits in prospective memory may forget to perform important tasks from their "to do" list, such as attending doctor's appointments and completing work responsibilities.

A study conducted by Heffernan and colleagues (2002) investigated the effects of alcohol use on memory. The authors recruited a sample of 60 college students and asked them to complete the Prospective Memory Questionnaire (PMQ), a self-report measure that assesses prospective memory and effects of alcohol abuse. The alcohol-abusing group was found to have significantly impaired prospective memory functioning. The heavy alcohol use group self-reported significantly higher levels of prospective forgetting for short-term, long-term, and internally cued prospective memory. However, the authors cautioned about interpretation of the results obtained because the PMQ is a self-report measure and can therefore be biased.

Similar to Heffernan and colleagues (2002), Ling and colleagues (2003) conducted an Internet-based study to investigate prospective memory function in alcohol abusers. Seven-hundred sixty-three participants completed the PMQ, the Everyday Memory Questionnaire (EMQ), and a demographic questionnaire. Results indicated a significant impairment in long-term aspects of prospective memory with increased cognitive failures among the heavy alcohol abusing group, corroborating previous results by Heffernan and colleagues (2002). Findings by Ling and colleagues (2003) add further support to the negative association between prospective memory deficits and alcohol abuse.

Contextual Memory

Another avenue of research on memory deficits in alcohol abusers has been the assessment of contextual memory, which is defined as the memory for the source of information, the temporal positioning of information, and the context in which that information was presented (Sullivan et al., 1997). Sullivan and colleagues (1997) assessed contextual memory in alcoholics using an order recognition test of verbal and non-verbal items. In order to test their contextual memory, the participants were shown 233 nouns and then were asked the question, "Which words did you see more recently?" (Sullivan et al., 1997, p. 198). Alcoholics were found to have significant deficits in contextual memory as compared to healthy controls. This study has important implications because it suggests that alcohol abuse has a negative impact on contextual memory.

Effects of Alcoholism on Executive Functioning

Bechara and Martin (2004) proposed that there is an underlying deficit in executive functioning components of working memory and decision-making in people who abuse substances. Bechara and Martin (2004) found performance differences between individuals with substance dependence and control participants on measures of working memory and decision-making with the substance dependent individuals performing significantly worse than controls. Thus, the authors established evidence to support the notion that individuals who abuse substances have underlying working memory and decision-making deficits.

Hildebrandt and colleagues (2004) further explored executive functioning deficits in people with long-term, heavy alcohol consumption diagnosed with alcohol dependence according to the ICD-10. Hildebrandt and colleagues (2004) assessed executive functioning (i.e., working memory, behavioral inhibition, and task shifting) using a two-back paradigm; participants were instructed to watch a computer screen presenting double-digit numbers from 10 to 99 and to press a response key when the number was the same as the previous number, two numbers back. The authors found that people with a history of long-term heavy alcohol consumption showed no impairment in working memory as compared to the matched healthy controls, but showed deficits in behavioral inhibition and task shifting. These results suggest that subdividing executive functioning into its constituent parts should be an integral part in studying executive functioning deficits in alcohol abusers (Hildebrandt et al., 2004).

Similarly, a study by Noel and colleagues (2007) looked at the performance of individuals who met the criteria for alcohol dependence according to the DSM-IV on the Iowa Gambling Task (IGT) in comparison to healthy controls. IGT is designed as a game in which four decks of cards, each of which has different rewards/punishments, are presented and the person has to choose one card out of a deck and they are either rewarded or punished for their choice. The goal of the task is for the participant to pick out the most risk-avoidant card deck. Results indicated that individuals with alcoholism had poor executive functioning, impairment in behavioral inhibition, and performed worse than control on the last 20 trials of IGT. The authors suggested that individuals with alcoholism display difficulties with decision-making, especially when these decisions involve risk. Moreover, the authors proposed that working memory in alcohol dependent individuals shows normal storage capacity, but the ability to manipulate information held in working memory is impaired.

Contrary to study findings by Hildebrandt and colleagues (2004) that found no evidence of deficits in working memory among alcohol dependents, Noel and

colleagues (2007) showed a partial deficit in working memory function among alcohol dependents. Both studies found deficits in behavioral inhibition among alcohol abusers; however, the study by Noel and colleagues (2007) indicated impairment in the manipulation of information component of working memory, whereas the study by Hildebrandt and colleagues (2004) did not. More research is needed on the executive functioning of working memory. Executive functioning, as was suggested by Hildebrandt and colleagues (2004), should be divided into its constituent parts in order for researchers to be able to reach a consensus about executive function impairment in alcohol abusers.

Tests of Premature-Aging Hypothesis and the Relation to Amount of Lifetime Alcohol Consumption

It has been hypothesized that the cognitive deficits seen in alcoholism are due to the premature aging of cognitive functioning caused by chronic alcohol consumption, a hypothesis known as the "premature-aging hypothesis" (Ryan & Butters, 1980). Ryan and Butters (1980) tested the "premature-aging" hypothesis by administering various learning and memory tasks to younger alcoholics (ages 34-49), older alcoholics (ages 50-59), and age-matched controls. Among the various tests administered by the researchers (Ryan & Butters, 1980), one was the Four-Word Short-Term Memory Test, in which participants are presented with four words and, following a distractor activity (i.e., count down from a three digit number by a certain number) are asked to recall these words. Their results provided evidence for the premature-aging hypothesis, showing that younger alcoholics consistently performed at a level similar to the 10 years older normal controls.

A study by Holden, McLaughlin, Reilly, and Overall (1988) looked to expand these findings. Using the Wechsler Adult Intelligence Scale (WAIS, 1955), which allows for discerning a person's mental age alongside their chronological one, Holden and colleagues (1988) tested participants who met the criteria for alcohol abuse or dependence according to the DSM-III. The results of the study indicated that the mental age of the alcoholic sample was seven years older than their actual chronological one, as compared to the age-matched sample. These results are in accord with the findings of Ryan and Butters (1980) and provide further evidence for the premature-aging hypothesis.

Nichols, Hochla and Parsons (1982) further explored the premature-aging hypothesis in females that met the criteria for alcoholism and were currently in a residential treatment facility. They found that alcoholic women performed significantly better than elderly non-alcoholic women on neuropsychological tests. The researchers, however, found that a subsample of female alcohol abusers who reported more severe alcohol intake performed comparably to non-alcoholic females that were 20-years their elder. Thus, the authors established partial support for the premature-aging hypothesis in females.

Shelton, Parsons, and Leber (1984) tested the premature-aging hypothesis by comparing the cognitive performance of middle-aged chronic alcoholics whose alcohol consumption was disruptive to their daily living to the performance of middle-aged controls and elderly controls. Cognitive performance was measured using a paired-associate learning test consisting of separate verbal and visuospatial subtests. Elderly controls were found to perform significantly worse than both the middle-aged controls and the middle-aged chronic alcoholics; middle-aged alcoholics performed on a similar level to middle-aged controls rather than the elderly controls. Therefore, the premature-aging hypothesis was not supported in this study.

Schottenbauer and colleagues (2007) further tested the premature-aging hypothesis by looking at the performance on a Selective Reminding Task (SRT) of participants who met the DSM-III-R criteria for alcohol dependence and comparing their performance to the performance of healthy controls. The authors described the SRT as a measure of learning and memory impairment. Alcoholics were found to have deficits in both learning and memory as compared to controls. Results indicated that age significantly predicted memory deficits among alcoholics, but lost statistical significance after controlling for dose effect (i.e., years of heavy drinking). These findings suggest that dosage is an important predictor variable of cognitive deficits alongside age and should be taken into consideration in future studies.

Pfefferbaum and colleagues (1992) performed a brain imaging study using Magnetic Resonance Imaging on participants who met alcohol dependence criteria on the Research Diagnostic Criteria (RDC, Spitzer, Endicott, & Robins, 1975) and discovered an age-related, not dose-related, structural damage in alcohol dependent individuals as compared to age matched healthy controls. Age significantly predicted structural damage above and beyond lifetime dosage of alcohol consumption, suggesting that even when the levels of lifetime alcohol consumption are comparable between younger and older alcoholics, older alcoholics seem to manifest greater structural damage in their brains. The significance of the neuroanatomical study by Pfefferbaum and colleagues (1992) lies in the finding of actual morphological differences in the brain structures among alcohol abusers contingent on their age and not dosage. Age and chronic alcohol abuse, as separate or compounded factors, have been linked to cause neuroanatomical damage.

The premature-aging hypothesis has been extensively researched (e.g., Nichols Hochla & Parson, 1982; Ryan & Butters, 1980; Schottenbauer et al., 2007; Shelton, Parsons, & Leber, 1984). Some studies provide support for the premature-aging hypothesis (Holden, McLaughlin, Reilly, & Overall, 1988; Ryan & Butters, 1980), while others provide partial or no evidence for this hypothesis (Nichols Hochla & Parson, 1982; Shelton, Parsons, & Leber, 1984). Even though researchers have not reached a consensus on the validity of the premature-aging hypothesis, age seems to have an effect on the cognitive and functional deficits associated with alcohol abuse. It also seems to have an effect on the

structure of the brain as has been found by Pfefferbaum and colleagues (1992). Dosage, likewise, seems to have an effect on the cognitive and functional deficits of alcohol abuse and will be explored further along in this review. More research is needed in order to pinpoint the mechanism by which age seems to have an effect on cognitive and functional deficits associated with alcohol abuse and the structural changes that are associated with it.

Effects of Gender on the Cognitive and Functional Deficits

A study by Sullivan and colleagues (2000) tested and compared the performance of 71 recently detoxified (1 month) alcoholic males on several neuropsychological measures to healthy male controls. All of the alcoholic males met the RDC criteria for alcohol dependence. Study participants underwent a thorough neuropsychological evaluation, which assessed executive functioning using the Wisconsin Card Sorting Task, short-term memory and production using the Brown-Peterson distractor tests, upper limb mobility by measuring grip strength and fine finger movement, declarative memory by using the delayed Wechsler Memory Scale stories, visuospatial abilities by the Hidden Figure Test, and gait and balance by using the Ataxia Battery. Consistent with previous research findings (e.g., Bechara & Martin, 2004; Hildebrandt et al., 2004; Noel et al., 2007; Ryan & Butters, 1980; Schottenbauer et al., 2007), Sullivan and colleagues (2000) found that recently detoxified alcoholic males exhibited deficits in measures of executive functioning, working memory, short-term memory, upper limb mobility, visuospatial abilities, and gait and balance as compared to healthy male controls. Furthermore, alcoholic males were found to be more vulnerable to deficits in upper limb mobility when age was taken into consideration.

This finding suggests that age, in combination with alcohol dependence, has a deleterious effect on functional deficits with older alcoholic males exhibiting more deficits in upper limb motor mobility as compared to younger male alcoholics. In addition, the authors found that age did not have an effect on cognitive functioning, but did have a predictive effect on deficits found in gait and balance, which is suggestive of age-related cerebellar damage due to alcohol abuse. Sullivan and colleagues (2000) found a dosage effect of alcohol consumption on gait and balance, but not on cognitive functioning. This finding contrasts previous findings reported by Schottenbauer and colleagues (2007) showing that lifetime alcohol consumption significantly predicted learning and memory performance as measured with SRT.

A study by Sullivan and colleagues (2002) tested the performance of 43 detoxified (3.6 months) females on several neuropsychological measures and compared them to healthy female controls and the males from the Sullivan and colleagues (2000) study. The study used the same neuropsychological measures as the Sullivan and colleagues (2000) study. As compared to the nonalcoholic control females, the alcoholic females exhibited many of the same cognitive and functional deficits as the alcoholic males did in the Sullivan et al. (2000) study - such as deficits in verbal and non-verbal working memory, visuospatial processing, and gait and balance. Sullivan and colleagues (2002), however, found that alcoholic females had a lifetime alcohol consumption that was 2.5 times lower than the alcoholic males in the Sullivan and colleagues (2000) study. Thus, female alcoholics who were detoxified for 3.6 months and had a lifetime consumption that was 2.5 times lower than the alcoholic males in the Sullivan et al. (2000) study still exhibited many of the same cognitive and functional deficits, suggesting that there is a difference in the way alcohol consumption affects the cognitive and functional deficits in the two genders. Females consume less alcohol, but still show the same signs of cognitive and functional deficits as males do, suggesting that alcohol has an especially deleterious effect on females. However, the findings of this study should be interpreted with caution because the Sullivan and colleagues (2000) study was not aimed at investigating a gender difference in the way alcohol affects the two genders. In Sullivan et al. (2002) study an ad hoc comparison was performed between the males and females from the two different samples and the authors cautioned about the interpretation of the results. More research is warranted in order to further understand the relationship between alcoholism and cognitive and functional deficits in the two genders.

Effects of Treatment History and Amount of Alcohol Consumption

In a study with chronic alcoholics who met lifetime criteria for alcohol dependence, Fein and Landman (2005) compared alcoholic patients with and without history of alcohol treatment, the latter being referred to as treatment naïve alcoholics. Retrospective information about the participants' alcohol use was gathered using a lifetime follow-back interview procedure, in which subjects broke their drinking history into periods with consistent alcohol use. The two groups were further matched into pairs, based on the age at which they first met the criteria for heavy drinking. Results indicated that, compared to treatment naïve alcoholics, those with treatment histories had higher dose alcohol consumption (58% for males and 68% for females) during the period since they first met the criteria for heavy drinking. The study by Fein and Landman (2005) suggests that treatment naïve alcoholics and alcoholics with treatment histories are distinct populations in terms of their alcohol use, with dosage being an important differentiating factor between the two groups. Therefore, Fein & Landman (2005) cautioned against generalizing research findings found amongst alcohol abusers with treatment histories to those that have never had treatment.

Another study by Fein, McGillivrary, and Finn (2006) compared a sample of 58 treatment naïve alcohol dependent (TNAD) participants, who met DSM-IV criteria for current

alcohol dependence, to a matched sample of non-alcoholic controls (NAC) on a Simulated Gambling Task (SGT) that measures decision-making and behavioral inhibition. Results indicated that the two groups did not differ with respect to decision-making. Contrary to previous findings showing decision-making and behavioral inhibition impairment in alcohol abusing sample with treatment histories (Noel et al., 2007), the study by Fein and colleagues (2006) showed that treatment naïve young adults with alcohol dependence do not have global deficits in decision-making. The poor decision-making that treatment naïve young adults with alcohol dependence exhibit regarding their alcohol consumption seems to be more specific to drinking.

The aforementioned findings were further supported by the Smith and Fein (2010) study, which compared a sample of TNAD participants who met DSM-IV criteria for current alcohol dependence with a matched sample of NACs on performance in nine performance domains. The domains included attention, auditory working memory, verbal processing, abstraction/cognitive flexibility, psychomotor function, immediate memory, delayed memory, reaction time, and spatial processing. Smith and Fein (2010) did not find a significant difference between the two samples on any of the nine performance domains. The absence of any difference between the NAC group and the TNAD group, when most studies consistently reported discernible differences between alcoholics and healthy controls (e.g., Sullivan et al., 2000; Rosenbloom et al., 2005), further supports the findings of Fein and Landman (2005), which suggested that treatment naïve alcoholics and alcoholics with treatment histories are different populations. Therefore, future research must continue to investigate these two groups, the treatment naïve alcohol dependents and the non-alcoholic controls.

Improvements with Abstinence and Potential Clinical Implications

A large study by Brandt and colleagues (1983) of 134 alcoholics with at least a 10-year history of daily alcohol consumption and at least 1-month abstinence at testing time revealed that alcoholics showed improvements on measures of short-term memory and psychomotor skills, but not long-term memory. At the follow-up phase of the study, the authors grouped the alcoholic abstainers into three groups: the "short-term abstinence" group, including participants who were abstinent between 1 and 2 months; the "long-term abstinence" group, including those who were abstinent between 1 and 3 years; and the "prolonged abstinence" group, for those who were sober for at least 5-years.

The authors found no significant difference between prolonged abstainers and non-alcoholic controls in terms of their short-term memory function, suggesting a recovery of short-term memory function among prolonged abstainers. The researchers, however, found a significant difference between prolonged abstainers and normal controls on measures of attention, perceptual speed, motor speed, visual scanning, and memory suggesting the irreversibility of the deficits in these areas even after prolonged abstinence. In addition, the researchers did not find a significant difference between prolonged abstainers and normal controls on a measure of non-verbal memory skills indicating recovery of these skills with prolonged abstinence.

A study by Rosenbloom and colleagues (2007) using various neuropsychological measures determined that alcoholics who met the DSM-IV criteria for alcohol dependence and were abstinent for 2 to 2.5 years showed significant improvement on measures of memory, and gait and balance as compared to controls. The alcoholic participants were self-reported abstainers. The participants were sober for over 4-months as they were tested at baseline. The authors (Rosenbloom et al., 2007) were also able to find structural changes in the brain of the abstaining participants with abstaining alcoholics showing a significantly decreased lateral ventricular volume as compared to relapsing alcoholics. Rosenbloom and colleagues (2007) concluded that both memory and ataxia could improve with sustained sobriety alongside structural changes in the brain.

A study by Bates and colleagues (2005) looked at the recovery of cognitive abilities of alcoholics who were in treatment for 6-weeks. The participants underwent a large neuropsychological evaluation before and after the 6-week treatment period. A modest recovery was reported in the domains of executive functioning, verbal processing speed, verbal ability domains, and medium recovery in the memory domain. Bates and colleagues (2005) cautioned that the improvements observed (with the exception of improvements in memory function) might be too minor to be of clinical relevance.

A study by Mann and colleagues (1999) investigated the pattern of cognitive deficits and their time-dependent recovery in a sample of 49 males who met the DSM-III-R criteria for alcohol dependence using a test-retest design. The authors also included a healthy sample of 49 males who were also tested and retested after a 5-week interval. The researchers (Mann et al., 1999) established significant improvement on measures of perceptual-motor speed, verbal knowledge, non-verbal reasoning, and spatial imagination. However, the authors did not find improvement in the area of short-term verbal memory. This is an interesting finding because it is in contrast to the findings of Brandt and colleagues (1983), who found that short-term memory recovers with abstinence and long-term memory does not. An explanatory factor for the results that Mann and colleagues (1999) observed is that perhaps not enough time passed between test and retest in order for the authors to be able to observe an improvement in short-term memory. The abstainers in the Brandt and colleagues (1983) study were abstinent for over 5-years and in the Mann and colleagues (1999) study the participants were only abstinent for 5-weeks.

The clinical implications of the recovery of some of the cognitive and functional deficits of alcohol abuse could potentially provide the ability for mental health practitioners

to tailor treatments for specific populations of alcohol abusers, as pointed out by Bates and colleagues (2005). The development of risk factor profiles that differentiate the cognitive and functional recovery among abstaining alcohol abusers can have a significant effect on how treatment is disseminated for the different profiles (Bates et al., 2005). The treatment process can be stifled if the treatment information is disseminated in such a way that an alcohol abuser with cognitive and functional deficits cannot assimilate it (Bates et al., 2005). The inability to assimilate the information that is disseminated as part of the therapeutic technique can influence the treatment outcome for people that suffer from alcohol abuse. The implication that the recovery of cognitive and functional deficits of alcohol abuse has is that specific therapeutic techniques can be better tailored to the needs of the people suffering from alcohol abuse.

Findings From Longitudinal Studies

A prospective study focusing on males was conducted in Denmark with 20-, 30-, and 40-year follow-ups. The sample of participants was chosen from a large Copenhagen birth cohort. At the 20-year follow-up of this cohort, Drejer, Theikjaard, Teasdale, Schulsinger, and Goodwin (1985) established that high-risk (i.e., having a father who is an alcoholic) 18- and 19-year-old males differed significantly from age-matched controls on measures of vocabulary, tests of categorizing ability, organization, and planning. This is an important finding, as the authors pointed out, because other researchers found these problems with prolonged alcohol abuse (e.g., Sullivan et al., 2000, 2002) and the results from this study show that it seems that high-risk males already have many of the deficits that were found in alcoholics prior to prolonged alcohol abuse. The 30-year follow-up studies on this cohort of high-risk males focused on the influence of genetic and environmental components of alcoholism (Knop et al., 1993) and the predictor variables of future alcoholism (Goodwin et al., 1994). The follow-up studies did not employ the use of neuropsychological batteries and therefore the results of the 20-year follow-up, which established that the high-risk males differ from age-matched controls on several neuropsychological measures, were not further explored. In a follow-up analysis by Knop and colleagues (2003), the authors concluded that the premorbid differences between the high and low-risk groups were only modestly related to problem drinking and alcohol dependence at age 30.

Discussion

The research on cognitive and functional deficits of alcohol abuse has been extensive and there seems to be a consensus amongst researchers that alcohol abuse includes cognitive and functional deficits in: learning, general memory, prospective memory, contextual memory, visuospatial abilities, upper limb mobility, gait and balance, executive functioning, decision-making, behavioral inhibition, behavioral inhibition, task shifting, working memory, and problem solving. However, there are some areas such as working memory, on which research has been inconclusive. Hildebrandt and colleagues (2004) did not find deficits in working memory among alcohol abusers, but other researchers determined that indeed there are deficits in working memory (Noel et al., 2007; Sullivan et al., 2000; Sullivan et al., 2002). Thus, more research is necessary in order to clarify how alcohol abuse impacts working memory. This can be done through clearly delineating working memory from executive functioning, as in the study by Hildebrandt and colleagues (2004), and classifying it in a way that would lead to consensus among different researchers.

The research on age and severity of cognitive and functional deficits of alcohol abusers seems to also fall into the area of inconclusive research findings. Researchers have found evidence to support the premature-aging hypothesis (Holden et al., 1988; Nichols Hochla & Parsons, 1982; Ryan & Butters, 1980) and neuroanatomical studies have reported age-related neuroanatomical damage even when dosage is controlled for (Pfefferbaum et al., 1992). The study by Schottenbauer and colleagues (2007) found that age had a predictive effect on the SRT performance of alcoholics, but this effect was significantly reduced once dose was entered into the equation. This suggests that age and dosage may have effects on cognitive functions and more research is needed to delineate what these effects are. However, some researchers (e.g., Sullivan et al., 2000) found that age and dosage did not have an effect on the cognitive functioning of alcoholics, but did have an effect on their functional capacities such as gait and balance. In addition, findings by Sheldon and colleagues (1984) did not provide support for the premature-aging hypothesis in their study of alcoholic males. These contrasting findings suggest that more research into this topic is imperative.

There are few studies covering the gender differences in cognitive and functional deficits of alcoholics. However, the study by Sullivan and colleagues (2002), which compared female alcoholics to male alcoholics, yielded important findings. The study showed that women tend to consume smaller amounts of alcohol for over a shorter period of time, but seem to exhibit many of the same cognitive and functional deficits as men do, such as upper limb strength, executive functioning, and speed. While these findings suggest that alcohol has a different effect in the two genders, more research is needed to further extrapolate what the differences between the genders are in terms of sensitivity to alcohol's deleterious effects.

Fein and Landman (2005) explored an avenue of research by noticing that researchers have focused on a convenience population (i.e., alcoholics in treatment or with a history of treatment) and did not focus on the larger population (i.e., naïve alcoholics or alcoholics who have not received treatment). Fein and colleagues (2006) and Smith and Fein (2010) did not report any cognitive or functional

differences between treatment naïve alcoholics and normal non-alcoholic samples, when directly comparing these two groups. These findings expand upon previous research that only examined alcoholics who have received treatment by including a sample of treatment naïve alcoholics. Further research is warranted in order to pinpoint at what level of consumption alcohol begins negatively impacting cognitive functioning. Also, research should be conducted to identify patient characteristics that distinguish treatment naïve alcoholics from alcoholics with treatment histories. This type of research can be enlightening because it can add other dimensions to understanding the differences between alcoholics with treatment and treatment naïve alcoholics.

Research in cognitive and functional deficits of alcohol use disorders can substantially benefit from longitudinal studies, which follow participants over time. By following participants with alcohol use disorders over time and conducting multiple sequential assessments of cognitive and functional deficits, longitudinal studies allow researchers to identify predictors and moderators of these deficits and distinguish between short- and long-term effects. The aforementioned Danish prospective study by Dreijer and colleagues (1985) was unique in that it employed a longitudinal design. However, none of the subsequent follow-up assessments (e.g., Goodwin et al., 1994; Knop et al., 1993) assessed participants' neuropsychological functioning, significantly limiting how much information regarding cognitive and functional deficits of alcohol abuse could be obtained from this prospective study. Thus, it is difficult to pinpoint what the cognitive and functional deficits of alcohol abuse are from this particular prospective study. Another limitation of the Danish cohort study was that it only focused on males, which limits the external validity of this study, since there appears to be a difference between how the genders respond to alcohol abuse (Sullivan et al., 2002). The Danish prospective study was ideal for discerning what the risk factors and predictors for alcohol abuse and dependence are, (e.g., Knop et al., 2003), but not the cognitive and functional deficits of alcohol abuse. Therefore, new prospective studies with both female and male cohorts are needed in order to disentangle what the cognitive and functional deficits of alcohol abuse are.

The findings presented in this review should be interpreted with caution because the nature of these studies is not of true experimentation. Therefore, the establishment of a causal relationship between alcohol abuse and cognitive and functional deficits was stifled. Likewise, the directionality of the deficits is hard to pinpoint. Are the cognitive and functional deficits seen in alcohol abusers a result of alcohol abuse or are they what led to the alcohol abuse in the first place? One way that the causal relationship and directionality between alcohol abuse and cognitive and functional deficits can be established is through true experimentation with animals. Random selection and random assignment can be accomplished in animal research and the internal validity of these types of studies would be quite strong. On the other hand, the external validity of such research would be thwarted because of the differences between humans and animals. Even with limited external validity, experimental studies with animals have a huge potential for elucidating the causal relationship between alcohol abuse and functional and cognitive deficits, and are therefore needed.

Another important limitation of the studies reviewed refers to the heterogeneous use of diagnostic criteria for alcohol dependence and abuse. Diagnosis of alcohol dependence or abuse was given based on a variety of diagnostic manuals, such as the ICD-10, DSM-III, DSM-IV, and RCD, which may account for the lack of consensus among different studies. Additionally, the cited studies did not always specify the longevity and the severity of the alcohol dependence or abuse. Future studies should agree on "gold standard" diagnostic criteria for alcohol dependence and abuse, in order to reach reliable and conclusive findings about the cognitive and functional deficits of alcohol abuse.

In addition, research that focuses on the recovery of cognitive and functional deficits of alcohol abuse is a very important avenue of research because it has implications for clinical practice. It is a necessary endeavor to establish the profile of risk factors that predict differences in cognitive recovery between individuals, as Bates and colleagues (2005) suggested. Establishing risk factors profiles can guide therapeutic interventions that could be better tailored for specific populations. Thus, more research is needed in order to identify the risk factors that predict differences in cognitive recovery of individuals. Further research in the area of recovery of functional and cognitive deficits of alcohol abuse can inform therapeutic work and help people with alcohol abuse recovery.

Conclusion

The relationship between alcohol abuse and cognitive and functional deficits may seem elementary on the surface; the more a person drinks the more deficits they exhibit. The results of numerous research studies, however, speak largely to the contrary. There are multiple variables that affect the cognitive and functional deficits of alcohol abusers including age, gender, treatment history, and dosage. There is documented recovery of some of the cognitive and functional deficits with abstinence and treatment. More encompassing research is needed in order to fully grasp and discern the effects of the aforementioned factors on cognitive and functional deficits of alcohol abusers. The details of this research could enlighten therapeutic practice and help to better understand and treat alcohol abuse.

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