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Neuropsychological Performance of South African Treatment-naïve Adolescents with Alcohol Dependence

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Abstract

Background—Alcohol dependence (AD) in developmentally vulnerable adolescents is ubiquitous and confers a risk for long-term neurocognitive sequelae, yet comorbid substance use disorders and psychopathology can complicate interpretations. Here, we compare cognitive functioning in adolescents with and without AD, who are free from comorbid disorders.

Methods—English- and Afrikaans-speaking adolescents (13–15 years) of mixed ancestry and low socio-economic status were recruited from the Cape Town region of South Africa. Adolescents with psychiatric, developmental, or other substance use disorders (SUDs) were excluded. AD (n = 26) and control (n = 26) groups were matched on age, gender, language, and level of education. Neuropsychological testing in participants' home language followed detailed medical/psychiatric evaluation.

Results—Although our sample included participants who smoked tobacco, lifetime dosage of other drugs was negligible. When tobacco and other drug use as well as demographic variables were controlled, adolescents with AD performed more poorly on measures of verbal story memory, self-monitoring, and psychomotor speed and coordination.

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Author Disclosures

Contributors

Authors George Fein, Susan Tapert and Paul Carey designed the study and wrote the protocol. Author Helen L. Ferrett, George Fein and Susan Tapert managed the literature searches and summaries of previous related work. Authors Helen L. Ferrett and Kevin Thomas undertook the statistical analysis, and author Helen L. Ferrett wrote the first draft of the manuscript. All authors contributed to and have approved the final manuscript.

Conflict of Interest

No conflict declared.

Conclusions—These preliminary results, although relatively subtle, suggest that adolescents with AD may be at increased risk for failure to reach optimal levels of neuromaturation, and may be susceptible to cognitive problems associated with protracted alcohol consumption.

Keywords

alcohol; adolescents; neuropsychology; health effects

1. INTRODUCTION

Although most adults who drink begin experimenting with alcohol as teenagers, relatively little is known about alcohol's effects in adolescence, a critical phase of brain development that is characterized by accelerated neural maturation, dendritic pruning, and increased myelination, particularly in the hippocampal and frontal regions (Gogtay et al., 2004; Sowell et al., 2004). Additional research into the effects of drinking during this important transition period is warranted.

Most studies of the impact of alcohol dependence on the brain have examined individuals in treatment. Such samples represent a small proportion of alcoholics in the general population, and we have shown that treated adults are not a representative sample of adult alcoholics: they have more severe alcoholism and substantially more psychiatric comorbidity (Di Sclafani et al., 2008; Fein, 2006; Fein et al., 2004; Fein and Landman, 2005; Fein et al., 2006). A reasonable assumption is therefore that adolescents in treatment for alcohol use disorders (AUDs) are also heavier drinkers with more comorbidities than untreated adolescents with AUDs in the general population. For these reasons, it is crucially important to study untreated, community-dwelling adolescents with AUDs to understand the effects of alcohol on the adolescent brain.

To study community-dwelling adolescents with AUDs but with no comorbid substance use disorders (SUDs) is important because doing otherwise would make it impossible to disentangle the effects of alcohol on brain structure and function from those of other substances. This goal has been challenging in the United States because the majority of adolescents with AUDs also have substantial histories of other substance use (Substance Abuse and Mental Health Services Administration, 2007). Amongst adolescents in South Africa, however, alcohol (followed by tobacco) remains the most popularly used substance of abuse, despite some recent changes in trends amongst youth seeking treatment (Parry et al., 2004; Pluddemann, 2008). For instance, surveys in Cape Town schools show that untreated adolescents prefer alcohol to cannabis (the most frequently used illicit substance), and demonstrate lower incidence of mixed substance abuse than found in the USA. The reported lifetime prevalence of adolescent substance use indicates that 66% of males and 48% of females consume alcohol, compared to 32% of males and 13% of females who use cannabis. Recent (past month) alcohol use (particularly weekend binge-drinking) is reported by 32% of school-going adolescents (Flisher et al., 2003). The Cape Town region of South Africa thus provides an opportunity to study adolescents meeting criteria for AUD but with minimal other drug use history.

The existing literature suggests that although the adverse effects of AUDs in adolescents tend to be more subtle than those exhibited by adults with AUDs (Moss et al., 1994), heavy adolescent drinking is associated with poorer neuropsychological functioning in various cognitive domains, including attention, intelligence, processing speed, motor speed, visuospatial abilities, and aspects of memory and executive functioning (Brown and Tapert, 2004; Brown et al., 2000; Moss et al., 1994; Tapert and Brown, 1999; Tapert et al., 2002; Tarter et al., 1995; Zeigler et al., 2005).

Unfortunately, comparisons across studies and interpretations of the overall pattern of data are made difficult by the fact of between-study discrepancies in test batteries and definitions of neuropsychological domains, as well as by differences in sample demographic and clinical characteristics (e.g., treated vs. treatment naïve, age at testing, years of education, race, socio-economic status, and presence of psychiatric comorbidities). Furthermore, due to the interactive effects of other SUDs (cannabis and stimulant use, for example, also predict attentional difficulties), poorer neuropsychological performance cannot exclusively be attributed to alcohol consumption.

Longitudinal studies have helped clarify some of the inconsistencies. Tapert et al. (2002), for example, showed that visuospatial problems are not generic to all AUDs, but are primarily associated with the intensity of alcohol withdrawal. Testing the same adolescents at 4- and 8-year follow-up periods has confirmed that protracted and ongoing alcohol abuse is associated with ongoing attention and memory difficulties, specifically verbal and nonverbal retention in the context of intact learning and recognition abilities (Brown and Tapert, 2004; Brown et al., 2000; Tapert and Brown, 1999; Tapert et al., 2002). Although not all neuropsychological domains are affected by alcohol consumption, and most authors acknowledge that effects are sometimes subtle, prolonged and excessive consumption of alcohol by adolescents has profoundly negative educational, occupational, physiological and psychosocial sequelae. These consequences, which extend into adulthood, have been well documented in the international as well as in the South African literature (Parry et al., 2004; Shuckit, 2009; Zeigler et al., 2005).

In sum, due to the high rates of polysubstance abuse and comorbid symptomatology present in the samples in the studies reviewed above, there is no clarity about what specific effects are attributable to alcohol to the exclusion of other substance use and psychiatric comorbidity. The aim of our Cape Town project is to clarify the effects of adolescent AUDs via study of relatively pure untreated adolescent AUDs. The current manuscript focuses on neuropsychological functioning.

2. MATERIALS AND METHODS

2.1. Participants

We recruited a heterogeneous sample of low socio-economic status mixed ancestry English or Afrikaans-speaking adolescents (ages 13–15 years) from 12 schools within a 25-km radius of the test site. Screening procedures included a psychiatric and medical history and physical examination (including urine analysis and breathalyzer testing) performed by a psychiatrist (PDC). The Schedule for Affective Disorders and Schizophrenia for School

Aged Children (6–18 Years) Lifetime Version (K-SADS-PL) (Kaufman, 1996) was used to ascertain current and past psychiatric diagnoses, as reported by the participants. The Semi-Structured Assessment for the Genetics of Alcohol (SSAGA-II) (Bucholz et al., 1994) was used to confirm AD diagnosis and to obtain detailed substance use histories (alcohol, tobacco, and all other drugs).

Participants were assigned to one of two groups: a heavy drinking group meeting DSM-IV criteria for alcohol dependence (American Psychiatric Association, 2000), or a light/non-drinking control group. Exclusion criteria for both groups were: mental retardation, lifetime DSM-IV Axis I diagnoses other than AD (as defined in the K-S ADS-PL, including major depression, dysthymia, mania, hypomania, cyclothymia, bipolar disorders, schizoaffective disorders, schizophrenia, schizophreniform disorder, brief reactive psychosis, panic disorder, agoraphobia, separation anxiety disorder, avoidant disorder of childhood and adolescence, simple phobia, social phobia, overanxious disorder, generalized anxiety disorder, obsessive compulsive disorder, attention deficit hyperactivity disorder, conduct disorder, oppositional defiant disorder, enuresis, encopresis, anorexia nervosa, bulimia, transient tic disorder, Tourette's disorder, chronic motor or vocal tic disorder, alcohol abuse and dependence, substance abuse and dependence, post-traumatic stress disorder, and adjustment disorders), current use of sedative or psychotropic medication, signs or history of fetal alcohol syndrome or malnutrition, sensory impairment, history of traumatic brain injury with loss of consciousness exceeding 10 minutes, presence of diseases that may affect the CNS (e.g., meningitis, epilepsy, HIV), less than 6 years of formal education, and lack of proficiency in English or Afrikaans. Collateral information verifying the absence of medical, psychiatric and psychosocial problems was obtained from consenting parents by a social worker at the consent-explanation interview. Verification of regular school attendance was obtained from school reports, and school teachers were consulted at pre-screening interviews conducted by a social worker to verify whether participants' behaviour and performance at school were considered to be within normal parameters. Participants in the AD ($n = 26$) and control ($n = 26$) groups were individually matched for age (within 1 year), gender, language, socio-economic status, and level of education (within 1 year). The mean age of the sample was 14.5 years (± 0.6) and they had completed 8.0 years (± 0.9) of education. Females (62%) outnumbered males (38%), and the majority of the sample were right handed (92%) and Afrikaans-speakers (88%) (see Table 1).

2.2. Measures

2.2.1. Substance use—A revised version of the Timeline Followback procedure (TLFB) (Sobell and Sobell, 1992) assessed lifetime history of alcohol use and drinking patterns (i.e., frequency, quantity, and density of alcohol consumption), including every phase from when subjects first started drinking at least once per month to the present, including all periods of sobriety. A standard drink was defined as one beer or wine cooler, one glass of wine, one 1.5-ounce shot of liquor (alone or in a mixed drink).

2.2.2. Neuropsychological battery and composite scores—A general-purpose neuropsychological test battery was selected. Due to the unavailability of current, culturally appropriate, unbiased South African tests (Foxcroft, 2004; van Ommen, 2005), age-

appropriate international tests with established utility in cross-cultural and multilingual contexts and in SUD studies were selected. In consultation with an Afrikaans linguistics specialist, appropriate cultural and language adaptations were made to the tests. Examples of these adaptations included replacement of items/terminology unfamiliar to South Africans, simplifications of test instructions, and substitution of items to ensure equivalent difficulty levels in both Afrikaans and English. Test instructions, stimuli, and response booklets were translated into Afrikaans and back-translated into English by independent translators. Hand dominance was established with the Edinburgh Handedness Inventory (EHI) (Oldfield, 1971). Ten composite domain scores were derived from the individual neuropsychological tests as follows:

1. Verbal Story Memory: Immediate and delayed recall of story and thematic units and delayed recognition of Stories E and F from the Children's Memory Scale (CMS) (Cohen, 1997).
2. Verbal List Learning: Total learning, short-term percent retention, long-term percent retention, learning over trials and trial 5 scores of the Auditory Verbal Learning Test (AVLT) (Maj et al., 1993).
3. Self-Monitoring and Regulation: Phonemic (letters L, B, and S) and semantic (animal category) fluency error scores (Strauss et al., 2006); AVLT total error score; time and rule violation scores from the Tower of London (ToL) (Culbertson and Zillmer, 2001); error scores from the Children's Color Trails Test (CCTT) (Llorente et al., 2003); and error scores from the Stroop Color-Word test (SCWT) (Golden and Freshwater, 2002).
4. Planning and Problem Solving: ToL total correct score; CLOX test trial 1 (Royall et al., 1998); and Matrix Reasoning subtest from the Wechsler Abbreviated Scale of Intelligence (WASI) (Wechsler, 1999).
5. Psychomotor Speed and Coordination: dominant and non-dominant peg insertion time from the Grooved Pegboard Test (GPT) (Russell and Starkey, 1993) and CCTT Trail 1 time.
6. Attention and Concentration: AVLT trial 1 score; Coding subtest from the Wechsler Intelligence Scale for Children - fourth edition (WISC-IV) (Wechsler, 1991); numbers backward subtest (CMS); and Stroop Color-Word correct score.
7. Sequencing Ability: CCTT Trail 2 and total error score.
8. Expressive Language: Phonemic and semantic total words generated from the verbal generativity tests; and WASI Vocabulary subtest.
9. Visuospatial Construction: Rey-Osterrieth Complex Figure copy (ROCF) (Meyers and Meyers, 1996); CLOX trial 2; and WASI Block Design subtest.
10. Visual Memory: immediate and delayed recall of the ROCF; Trials 1–3, immediate and delayed recall of the CMS Dots subtest.

2.3. Procedures

All study procedures were approved by the Committee for Human Research of Stellenbosch University. After eligibility was established, written consent from parents and written assent from participants was obtained. Participants were transported from their homes or schools to the testing site. After physical and psychiatric screening, urine analysis and breathalyzer testing, the subjects completed demographic self-report questionnaires and neuropsychological testing (approximately 3 hours). Tests were individually administered by a clinical psychologist (HLF) in the participants' preferred language. Participants were provided with meals and refreshments, and at the conclusion of the testing session were compensated for their time with gift vouchers. Confidentiality of all study information was maintained with the exception of statutory reporting requirements in newly-identified or ongoing threats to the safety of minor participants. Two of the 54 recruited subjects were excluded as screen failures: one was diagnosed with current posttraumatic stress disorder and the other tested positive for cannabis use.

2.4. Statistical Analysis

All data were checked and cleaned before analyses. First, descriptive statistics and exploratory group comparisons were computed using Statistica 8.0 (StatSoft, 2008). SPSS Version 17.0 (SPSS Inc., 2008) was used for all other analyses. Depending on variable type and normality of score distributions, *t*-tests, ANOVAs, or chi-square tests were used to ascertain differences between the AD and control groups. For all ANOVAs, the assumptions of normal distributions, homogeneity of variance, independent observations, and interval measurement scales were met, thereby reducing the risk of Type I errors (Edenberg et al., 2005).

Composite neuropsychological scores were computed to reduce the number of variables initially examined. A hybrid method (described by (Medina et al., 2007)) grouped measures into domains based on theoretical assumptions (Lezak et al., 2004), then average domain *z*-scores were computed. Goodness-of-fit was assessed by calculating Cronbach's alpha coefficients for each domain. We derived 10 composite neuropsychological domains, with Cronbach's alphas ranging from .47 to .96. The tests in each composite are listed in the Methods section, and in Table 2 along with descriptive statistics for each measure.

After confirming that all assumptions for regression analyses were met, a series of multiple regression analyses was conducted to ascertain whether alcohol group status and/or lifetime dosage of alcohol predicted performance on each domain score. For all analyses, the neuropsychological domain score was the outcome variable, and substance use and demographic variables were hierarchically entered as predictors: Either (i) alcohol group status or (ii) lifetime dosage of alcohol were entered on Step 1; lifetime tobacco dose and lifetime dose of all other drugs (including cannabis) were entered on Step 2; and completed years of education, participant age at testing, and language of test administration were entered on Step 3.

When alcohol group status significantly predicted performance within a neuropsychological domain, post-hoc multiple regression analyses were done on each of the measures within

that domain to determine whether AD predicted performance even if lifetime tobacco use, lifetime other substance use, level of education, age at testing, and language of test administration were controlled. The same procedures were followed as with the domain scores. Z-scores were used for all neuropsychological measures, as Kolgorov-Smirnov tests suggested non-normal score distributions.

3. RESULTS

3.1. Demographic and substance use characteristics

ANOVA and χ^2 analyses confirmed that the AD and control groups were successfully matched on major demographic characteristics (see Table 1). Within both the AD and control groups, some participants had experimented with tobacco and cannabis (see Table 1), and several control participants had experimented with alcohol. Although groups did not differ in the age of initiating experimenting with various substances, groups did differ in terms of the volume of tobacco and other substance use (see Table 1). For instance, many more controls than AD adolescents had never used cigarettes ($\chi^2(1) < 6.24, p = .012$) or cannabis ($\chi^2(1) = 15.44, p < .001$). Average lifetime tobacco and cannabis usage was much higher in the AD group than in the control group (Welch's $F(1, 25.085) = 5.59, p = .026$; Welch's $F(1, 25.235) = 11.26, p = .003$, respectively).

As expected, AD adolescents had substantially greater alcohol involvement than controls. For example, many more controls had never used alcohol or been intoxicated ($\chi^2(1) = 12.38, p < 0.001$; $\chi^2(1) = 48.15, p < .001$, respectively). Average lifetime alcohol usage was much higher in the AD group than in the control group (Welch's $F(1, 25.001) = 46.60, p < .001$).

Although none of the participants were diagnosed with psychiatric disorders specified in the K-SADS-PL, sub-threshold and threshold symptoms were tallied in order to provide some indication of possible sub-diagnostic indicators of psychiatric vulnerability. Across the entire sample, some symptoms were endorsed for each diagnostic category addressed in the K-SADS-PL, with the exception of mania, enuresis, encopresis, anorexia or bulimia. No difference in symptom counts for individual diagnoses was found between the AD and control groups; however, when externalizing symptom groups were clustered together (namely ADHD, oppositional defiant disorder and conduct disorder), the AD group demonstrated a tendency to endorse more symptoms than the control group ($F=5.07, sig. = .03$).

3.2. Multiple regression analysis: Domain scores

The mean scores shown in Table 2 indicate that participants in the AD group performed more poorly than those in the control group across all composite domains. In hierarchical regression analyses, after controlling for lifetime tobacco and other substance use, education, age, and language of test administration, AD group status was associated with poorer performance in the domains of Verbal Story Memory ($\beta = -.31, p = .041$), Self-Monitoring ($\beta = .28, p = .045$), and Psychomotor Speed and Coordination ($\beta = .34; p = .03$) (see Table 3). In these domains, alcohol use, other substance use, and the listed demographic variables together account for 26% to 38% of the variability in performance. Poorer

performance in these domains suggests poorer recall rates on story memory tasks; a tendency to commit more repetition, intrusion, and rule-violation errors across various tests; and slower completion times on motor tasks. AD group status was not statistically significantly associated with performance in the domains of Verbal List Learning, Planning and Problem-Solving, Attention and Concentration, Sequencing Ability, Expressive Language, Visuospatial Construction, and Visual Memory.

Additionally, within the domains of Verbal Story Memory, Self-Monitoring, and Psychomotor Speed and Coordination, certain demographic factors accounted for a statistically significant proportion of the variance (see Table 3). For instance, years of education was associated with performance in all three domains (i.e., participants with more years of education performed better on tests in these domains), and language of test administration was associated with performance in the Self-Monitoring domain (i.e., those participants tested in English tended to perform better on tests in this domain). In contrast, the use of other substances (tobacco and cannabis) did not account for a significant portion of the variability in performance between participants in the AD and control groups within any of the composite neuropsychological domains. Furthermore, a separate set of similar multiple regression analyses using alcohol dosage, rather than group status, as the primary predictor variable did not detect dose-dependent relationships in any of the composite domains.

3.3. Multiple regression analysis: Individual neuropsychological tests

Measures comprising the domains significantly predicted by AD group were examined in regression follow-up regression analyses. When demographic and other substance use variables were held constant, AD group status was associated with: poorer performance on immediate and delayed recall of thematic units on the CMS Stories test ($\beta = -.35, p = .017$, and $\beta = -.32, p = .037$, respectively; Verbal Story Memory domain); more color-word errors on the SCWT ($\beta = .50, p = .001$; Self-Monitoring domain); and longer time to completion with the non-dominant hand on the GPT ($\beta = .39, p = .018$; Psychomotor Speed and Coordination domain). Overall, the regression models here demonstrated that, on these four dependent measures, alcohol use, other substance use, and the listed demographic variables together accounted for between 21% and 43% of variability in performance between AD and control participants. On these four dependent measures, the use of tobacco and other drugs was not significantly associated with performance. Age was, however, associated with number of SCWT color-word errors (i.e., older children made fewer errors); similarly, more years of education was associated with better performance on all four measures.

4. DISCUSSION

We report here on the substance use characteristics and neuropsychological performance of untreated adolescents from low socio-economic backgrounds in the Cape Town region. Participants with “pure” alcohol dependence were compared to light/non-drinking controls with no current or lifetime comorbid psychopathology; groups were matched for age, gender, language, socio-economic status and level of education.

Substance abuse patterns in our sample resemble trends reported for 2,930 high school students in Cape Town demonstrating a preference for alcohol (31%), followed by cigarettes (27%) and cannabis (7%) (Flisher et al., 2003). Onset age of between 12 and 13 years for drinking, smoking and using other drugs appears to be similar to international findings. In our sample, the age of first intoxication tends to mark the onset of regular drinking, which occurs towards the end of the 12th year. Our South African AD sample consumed less alcohol (78 units per month) than a sample of American adolescents who reportedly consumed 131 units in an average drinking month (Tapert and Brown, 1999); the latter sample, however, encompassed a wider age range (13–19 years) and included individuals with more comorbid drug use and externalizing behaviors. Our adolescent ADs indulged in a similar pattern of weekend binge-drinking found in the US (Moss et al., 1994). Although more AD participants than controls in our sample used tobacco (83% compared to 69%), comorbid substance use was substantially lower than in US adolescent AUD samples reported in previous studies. Tarter et al. (1995) demonstrated polysubstance abuse rates as high as 60% for cannabis and 26% for other drugs; in contrast, although 69% of our sample had experimented with cannabis, their lifetime consumption was 4 units, and none of our participants reported ever using any other type of illicit drug.

Adolescent substance use is often associated with psychiatric illness, particularly mood disorders, ADHD, conduct disorder and oppositional defiant disorders (Dawes et al., 2000). Although our adolescent AD group endorsed more symptoms relating to externalizing conditions than the control groups, none of the AD participants met the full criteria for any psychiatric disorder as defined by the K-SADS-PL. Our AD sample thus represented relatively pure AD, with minimal other drug use and no significant psychiatric symptomatology.

Neuropsychological testing demonstrated that AD predicted poorer functioning on 3 out of 10 composite domain scores (verbal story memory, self-monitoring and psychomotor speed and coordination), and on 4 of the individual tests (immediate and delayed recall of CMS story thematic units, SCWT color-word errors and GPT non-dominant hand peg insertion time).

Memory differences were present in individual test results and in the verbal story memory domain score. The association between alcohol use and poorer retention of verbal information found in American studies (e.g., (Brown and Tapert, 2004; Brown et al., 2000)) was confirmed in our study, although the differences we found were predominantly found in story memory rather than in list learning. The difference between controls and AD on the CLOX 1 test seemed to be an isolated test-specific result that could not be generalized to indicate inferior overall planning ability in AD participants.

In contrast to Moss et al.'s (1994) findings that 14–15-year-old AD participants made fewer perseverative and commission errors than controls, our AD participants demonstrated poorer self-monitoring and regulation abilities. Our study confirmed US findings (Nigg et al., 2006; Tarter et al., 1995) that AD is associated with poorer ability to inhibit prepotent responses, which is one of the indicators of neurobehavioral disinhibition that might render adolescents

vulnerable to adolescent substance involvement (Clark et al., 2005). In our sample, it is unclear whether the inhibitory difficulties precede or follow the onset of AD.

Interestingly, no gender-specific neuropsychological differences were found in our study, contradicting the findings elsewhere of greater female vulnerability to alcohol morbidity in adolescence (Brown and Tapert, 2004; Moss et al., 1994; Tarter et al., 1995). In the USA, for example, female vs. male adolescents with SUDs display poorer results in tests of sustained attention, intelligence, perceptual speed and language achievement (Tarter et al., 1995).

In contrast to the US studies, no differences were found in attentional, visual memory or visuospatial abilities. This may indicate that the neuropsychological consequences of AD in Capetonian adolescents are less pervasive (i.e., they affect fewer cognitive domains) than in adolescents in the United States, possibly due to the absence of comorbid other SUDs and psychiatric symptoms. This underscores the value of this study in terms of the uniqueness of the sample, which differs from those traditionally studied in the adolescent AD field in that our participants were treatment-naïve and free of comorbid psychiatric disorders and other SUDs. Strengths of the study include enhanced chances that differences are attributable to alcohol consumption and not to the influence of other substances or comorbid psychiatric illnesses. Limitations of the study include its cross-sectional design and relatively small sample size, which limit our understanding of whether the effects may be transient or more enduring. Although when demographic factors were controlled, AD predicted neuropsychological performance on 3 domains, the interaction effects of AD and demographic variables (specifically years of completed education and test language) cannot be ruled out. The possibility of pre-existing inhibitory problems also cannot be ruled out. Longitudinal follow-up of these adolescents would help demonstrate whether the findings are a result of alcohol abuse by examining the trajectory of neuropsychological performance in the context of adolescent neural maturation, ongoing ADs, other SUDs or spontaneous remission. Progressively deteriorating function would implicate repeated toxic insults (in the form of excessive doses of alcohol) at this critical phase of neurodevelopment. In individuals who cease abusive drinking one could ascertain whether (or to what degree) recovery of function occurs, or whether brain maturation is arrested at this stage.

The current data, although generally consistent with the notion that heavy alcohol use in adolescence is associated with decreased volume in hippocampal and prefrontal regions, as well with frontal white matter abnormalities (De Bellis et al., 2000; De Bellis et al., 2005), need to be confirmed by more detailed studies of 'pure' samples such as this one. To that end, we will augment these data in the near future with analysis structural MRI and electrophysiological data on brain function and the presence of neurophysiological endophenotypes, as well as with measures of behavior and personality. Such data in this unique sample will prove invaluable in determining the characteristics of such factors in 'pure' adolescent AD.

Although a substantial proportion of individuals who drink excessively during adolescence transition into normative drinking patterns or abstinence, others develop adult AD and/or other SUDs, as well as other psychiatric co-morbidity (Clark, 2004). In addition to these risk

factors, although the neuropsychological difficulties demonstrated in our sample are relatively subtle, they may have important clinical and public health implications. Memory difficulties are likely to affect our youth's chances of reaching optimal potential in educational settings, thereby increasing their risk of experiencing occupational difficulties. Difficulties in self-monitoring and response inhibition may result in interpersonal complications. The early onset of AD is a source of concern in our community. Early onset (before the age of 15 years) of SUDs has been associated with a variety of negative health, safety and psychosocial problems (Zeigler et al., 2005). Examples of the problems associated with alcohol misuse that are relevant in the South African context are increased risk for injury and death (for example, from interpersonal violence, motor vehicle accidents and drownings associated with excessive alcohol use); increased probability of participating in sexually risky behaviors, with consequences such as becoming pregnant unintentionally, giving birth to children suffering from Fetal Alcohol Spectrum disorders, or contracting sexually transmitted diseases and being infected with HIV; increased probability of experiencing academic difficulties, failing to pass school grades, failing to attend school regularly, dropping out of high school, and being involved in criminal activities (Parry et al., 2004). It is also a source of concern that adolescents with diagnoses of AD have not sought or been referred to treatment. In sum, developmentally vulnerable adolescents who consume excessive amounts of alcohol and who suffer from AD may be at increased risk of failure to develop optimal levels of neuro-cognitive functioning and to thrive in numerous aspects of life.

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REFERENCES

- American Psychiatric Association. DSM-IV-TR: Diagnostic and Statistical Manual of Mental Disorders. Washington, DC: American Psychiatric Publishing; 2000.
- Brown SA, Tapert SF. Adolescence and the trajectory of alcohol use: basic to clinical studies. *Ann N Y Acad Sci.* 2004; 1021:234–244. [PubMed: 15251893]
- Brown SA, Tapert SF, Granholm E, Delis DC. Neurocognitive functioning of adolescents: effects of protracted alcohol use. *Alcohol Clin Exp Res.* 2000; 24(2):164–171. [PubMed: 10698367]
- Bucholz KK, Cadoret R, Cloninger CR, Dinwiddie SH, Hesselbrock VM, Nurnberger JI Jr, Reich T, Schmidt I, Schuckit MA. A new, semi-structured psychiatric interview for use in genetic linkage studies: a report on the reliability of the SSAGA. *J Stud Alcohol.* 1994; 55(2):149–158. [PubMed: 8189735]
- Clark DB. The natural history of adolescent alcohol use disorders. *Addiction.* 2004; 99(Suppl 2):5–22. [PubMed: 15488102]
- Clark DB, Cornelius JR, Kirisci L, Tarter RE. Childhood risk categories for adolescent substance involvement: a general liability typology. *Drug Alcohol Depend.* 2005; 77(1):13–21. [PubMed: 15607837]

- Cohen, M. Children's Memory Scale: Manual. San Antonio: The Psychological Corporation; 1997.
- Culbertson, WC.; Zillmer, EA. Tower of London: Technical Manual. New York: Multi-Health Systems Inc.; 2001.
- Dawes MA, Antelman SM, Vanyukov MM, Giancola P, Tarter RE, Susman EJ, Mezzich A, Clark DB. Developmental sources of variation in liability to adolescent substance use disorders. *Drug Alcohol Depend.* 2000; 61(1):3–14. [PubMed: 11064179]
- De Bellis MD, Clark DB, Beers SR, Soloff PH, Boring AM, Hall J, Kersh A, Keshavan MS. Hippocampal volume in adolescent-onset alcohol use disorders. *American Journal of Psychiatry.* 2000; 157(5):737–744. [PubMed: 10784466]
- De Bellis MD, Narasimhan A, Thatcher DL, Keshavan MS, Soloff P, Clark DB. Prefrontal cortex, thalamus, and cerebellar volumes in adolescents and young adults with adolescent-onset alcohol use disorders and comorbid mental disorders. *Alcohol Clin Exp Res.* 2005; 29(9):1590–1600. [PubMed: 16205359]
- Di Sclafani V, Finn P, Fein G. Treatment-naïve Active Alcoholics Have Greater Psychiatric Comorbidity Than Normal Controls but less than Treated Abstinent Alcoholics. *Drug Alcohol Depend.* 2008; 98(1–2):115–122. [PubMed: 18620818]
- Edenberg HJ, Bierut LJ, Boyce P, Cao M, Cawley S, Chiles R, Doheny KF, Hansen M, Hinrichs T, Jones K, Kelleher M, Kennedy GC, Liu G, Marcus G, McBride C, Murray SS, Oliphant A, Pettengill J, Porjesz B, Pugh EW, Rice JP, Rubano T, Shannon S, Steeke R, Tischfield JA, Tsai YY, Zhang C, Begleiter H. Description of the data from the Collaborative Study on the Genetics of Alcoholism (COGA) and single-nucleotide polymorphism genotyping for Genetic Analysis Workshop 14. *BMC Genet.* 2005; 6(Suppl 1):S2. [PubMed: 16451628]
- Fein, G. Structural Brain MRIs in a Treatment-Naïve Active Drinking Alcohol Dependent Sample. Paper presented at the ISBRA meeting in Sydney; Australia. 2006.
- Fein G, Klein L, Finn P. Impairment on a simulated gambling task in long-term abstinent alcoholics. *Alcohol Clin Exp Res.* 2004; 28(10):1487–1491. [PubMed: 15597080]
- Fein G, Landman B. Treated and treatment-naïve alcoholics come from different populations. *Alcohol.* 2005; 35(1):19–26. [PubMed: 15922134]
- Fein G, McGillivray S, Finn P. Normal performance on a simulated gambling task in treatment-naïve alcohol-dependent individuals. *Alcohol Clin Exp Res.* 2006; 30(6):959–966. [PubMed: 16737453]
- Flisher AJ, Parry CD, Evans J, Muller M, Lombard C. Substance use by adolescents in Cape Town: prevalence and correlates. *J Adolesc Health.* 2003; 32(1):58–65. [PubMed: 12507802]
- Foxcroft C, Paterson H, le Roux N, Herbst D. Psychological assessment in South African: a needs analysis: The test use patterns and needs of psychological assessment practitioners. Pretoria, Human Sciences Research Council. 2004
- Gogtay N, Giedd JN, Lusk L, Hayashi KM, Greenstein D, Vaituzis AC, Nugent TF 3rd, Herman DH, Clasen LS, Toga AW, Rapoport JL, Thompson PM. Dynamic mapping of human cortical development during childhood through early adulthood. *Proc Natl Acad Sci U S A.* 2004; 101(21):8174–8179. [PubMed: 15148381]
- Golden, CJ.; Freshwater, SM. Stroop Color and Word Test: A Manual for Clinical and Experimental Uses. Los Angeles: Stoelting Co.; 2002.
- Kaufman, J.; Birmaher, B.; Brent, D.; Rao, U.; Ryan, N. The Schedule for Affective Disorders and Schizophrenia for School Aged Children (6–18 Years) Lifetime Version. <http://www.wpic.pitt.edu/ksads>. 1996.
- Lezak, M.; Howieson, D.; Loring, D. Neuropsychological assessment. New York: Oxford University Press; 2004.
- Llorente, AM.; Williams, J.; Satz, P.; D'Elia, LF. Children's Color Trails Test: Professional Manual. Odessa: Psychological Assessment Resources; 2003.
- Maj M, D'Elia L, Satz P, Janssen R, Zaudig M, Uchiyama C, Starace F, Galderisi S, Chervinsky A. Evaluation of two new neuropsychological tests designed to minimize cultural bias in the assessment of HIV-1 seropositive persons: a WHO study. *Arch Clin Neuropsychol.* 1993; 8(2):123–135. [PubMed: 14589670]

- Medina KL, Hanson KL, Schweinsburg AD, Cohen-Zion M, Nagel BJ, Tapert SF. Neuropsychological functioning in adolescent marijuana users: subtle deficits detectable after a month of abstinence. *J Int Neuropsychol Soc.* 2007; 13(5):807–820. [PubMed: 17697412]
- Meyers, J.; Meyers, K. *Rey Complex Figure and the Recognition Trial: Professional manual. Supplemental norms for children and adolescents.* Odessa: Psychological Assessment Resources; 1996.
- Moss HB, Kirisci L, Gordon HW, Tarter RE. A neuropsychologic profile of adolescent alcoholics. *Alcohol Clin Exp Res.* 1994; 18(1):159–163. [PubMed: 8198214]
- Nigg JT, Wong MM, Martel MM, Jester JM, Puttler LI, Glass JM, Adams KM, Fitzgerald HE, Zucker RA. 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 Adolesc Psychiatry.* 2006; 45(4):468–475. [PubMed: 16601652]
- Oldfield RC. Grooved Pegboard Test. *Neuropsychology.* 1971; 9:97–113.
- Parry CD, Myers B, Morojele NK, Flisher AJ, Bhana A, Donson H, Pluddemann A. Trends in adolescent alcohol and other drug use: findings from three sentinel sites in South Africa (1997–2001). *J Adolesc.* 2004; 27(4):429–440. [PubMed: 15288752]
- Pluddemann A, Parry C, Cerff P, Bhana A, Pereira T, Potgieter H, Gerber W, Nqini T, Petersen P. Monitoring alcohol and drug abuse trends in South Africa. *Drug and Alcohol Review.* 2008; 2(27):185–189. [PubMed: 18264880]
- Royall DR, Cordes JA, Polk M. CLOX: an executive clock drawing task. *J Neurol Neurosurg Psychiatry.* 1998; 64(5):588–594. [PubMed: 9598672]
- Russell, EW.; Starkey, RI. *Halstead Russell Neuropsychological Evaluation System (HRNES).* Los Angeles: Western Psychological Services; 1993.
- Shuckit MA. Alcohol-use Disorders. *The Lancet.* 2009; 373(9662):492–501.
- Sobell, LC.; Sobell, MB. *Allen and Litten. Totowa, NJ: Humana Press; 1992. Timeline Follow-back: A technique for assessing self-reported alcohol consumption. Measuring Alcohol Consumption: Psychosocial and Biochemical Methods; p. 41–72.*
- Sowell ER, Thompson PM, Toga AW. Mapping changes in the human cortex throughout the span of life. *Neuroscientist.* 2004; 10(4):372–392. [PubMed: 15271264]
- SPSS Inc. *SPSS Statistics 17.0, Rel.17.0.0.* Chicago: SPSS Inc.; 2008.
- StatSoft, I. *Statistica data analysis software system, Version 8.0.* 2008. www.statsoft.com
- Strauss, E.; Sherman, EMS.; Spreen, O. *A compendium of neuropsychological tests : administration, norms, and commentary.* Oxford; New York: Oxford University Press; 2006.
- Substance Abuse and Mental Health Services Administration. *Results from the 2006 National Survey on Drug Use and Health: National Findings (Office of Applied Studies, NSDUH Series H-32, DHHS Publication No. SMA 07-4293.* Rockville, MD: 2007.
- Tapert SF, Brown SA. Neuropsychological correlates of adolescent substance abuse: four-year outcomes. *J Int Neuropsychol Soc.* 1999; 5(6):481–493. [PubMed: 10561928]
- Tapert SF, Granholm E, Leedy NG, Brown SA. Substance use and withdrawal: neuropsychological functioning over 8 years in youth. *J Int Neuropsychol Soc.* 2002; 8(7):873–883. [PubMed: 12405538]
- Tarter RE, Mezzich AC, Hsieh YC, Parks SM. Cognitive capacity in female adolescent substance abusers. *Drug Alcohol Depend.* 1995; 39(1):15–21. [PubMed: 7587969]
- van Ommen CH. Putting the PC in IQ: Images in the Wechsler Adult Intelligence Scale -third edition (WAIS-III). *South African Journal of Psychology.* 2005; 35(3):532–554.
- Wechsler, D. *Manual for the Wechsler Intelligence Scale for Children - Third Edition (WISC-III).* San Antonio, TX: The Psychological Corporation; 1991.
- Wechsler, D. *Wechsler Abbreviated Scale of Intelligence.* San Antonio, TX: The Psychological Corp.; 1999.
- Zeigler DW, Wang CC, Yoast RA, Dickinson BD, McCaffree MA, Robinowitz CB, Sterling ML. The neurocognitive effects of alcohol on adolescents and college students. *Prev Med.* 2005; 40(1):23–32. [PubMed: 15530577]

Table 1

Sociodemographic and substance use characteristics of the current sample.

	Control Group (n= 26)	AD Group (n= 26)
	M (SD) or %	M (SD) or %
Demographics		
Age (in years)	14.83 (0.63)	15.08 (0.64)
Years of completed education	8.04 (0.87)	8.04 (0.77)
% Afrikaans-speaking	88	88
% Female	62	62
% Right-handed	85	100
Tobacco Use		
% Never smoked tobacco *	69	35
% Lifetime <100 cigarettes	27	27
% Lifetime >100 cigarettes **	4	39
Smoking onset age	13.50 (0.54)	12.88 (1.36)
Lifetime tobacco dose ^{a*}	26.50 (123.26)	1412.42 (2987.17)
Other Substance Use		
% Never used cannabis ***	85	31
% Never used any other drugs	100	100
Cannabis use onset age	13.25 (1.50)	13.67 (0.91)
Lifetime cannabis dose ^{b**}	0.15 (.37)	3.69(5.37)
Alcohol Use		
% Never drunk alcohol ***	39	0
% Never been intoxicated ***	97	0
Drinking onset age	12.88 (1.26)	12.42 (1.47)
Alcohol lifetime dose ^{c***}	3.15 (5.10)	1909.31 (1423.80)
Age of first intoxication	-	12.77 (1.24)
Age of onset of regular drinking	-	12.96 (1.11)
Regular drinking duration (months)	-	21.54 (8.04)
Regular drinking frequency ^d	-	6.62 (2.64)
Regular drinking quantity ^e	-	13.15 (7.48)

Note. For all of the variables not presented as percentages, means are presented with standard deviations in parentheses.

* $p < .05$;

** $p < .01$;

*** $p < .001$

^aTotal number of cigarettes smoked in lifetime;

^bTotal number of 'joints' smoked in lifetime;

^cTotal number of standard units of alcohol consumed in lifetime;

^dDrinking days per month;

^eAverage standard units per drinking day.

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Table 2

Neuropsychological performance within composite domains.

	Control Group (<i>n</i> = 26)		AD Group (<i>n</i> = 26)	
	Range	<i>M</i> (<i>SD</i>)	Range	<i>M</i> (<i>SD</i>)
Verbal Story Memory (<i>a</i> = .96)	-0.87 – 2.55	0.33 (1.01)	-1.33 – 1.12	-0.33 (0.70)
CMS:				
Story Units Immediate Recall	13–67	30.62(15.51)	5–52	22.54(11.68)
Thematic Units Immediate Recall	2–13	5.88 (3.13)	0–9	3.69 (2.29)
Story Units Delayed Recall	11–60	27.42(14.05)	3–43	19.35(11.20)
Thematic Units Delayed Recall	1–11	5.31 (3.34)	0–7	3.04(1.95)
Story Recognition	17–28	21.62(3.02)	13–25	19.73(2.99)
Verbal List Learning (<i>a</i> = .52)	-1.09 – 1.00	0.10 (0.53)	-1.54 – 0.92	-0.10 (0.63)
AVLT:				
Total Words Learned	36–63	50.88 (7.91)	24–66	47.81 (9.90)
Short-Term Percent Retention	55–110	85.69(13.71)	67–128	92.81(16.67)
Long-Term Percent Retention	67–109	87.62(9.49)	33–120	84.85(20.74)
Learning Over Trials	4–33	18.96 (7.71)	-1–34	14.54(8.02)
Trial 5	8–15	12.27 (1.95)	7–15	11.50 (2.14)
Self-Monitoring (<i>a</i> = .62)	-0.72– 0.60	-0.17 (0.35)	-.57 – 1.56	0.17 (0.53)
Phonemic Fluency Errors	0–6	1.81 (1.74)	0–5	1.46 (1.30)
Category Fluency Errors	0–2	.23 (.51)	0–2	.35 (.56)
AVLT Total Errors	0–18	6.08 (4.89)	0–32	9.69 (6.75)
ToL:				
Time violations	0–2	.31 (.62)	0–2	.46 (.65)
Rule violations	0–2	.27 (.53)	0–6	.85 (1.49)
CCTT:				
Trail 1 Errors	0–1	.15 (.37)	0–1	.19 (.40)
Trail 2 Errors	0–3	.73 (.96)	0–4	1.04 (1.22)
SCWT:				
Word Errors	0–3	.31 (.74)	0–3	.42 (.86)
Color Errors	0–2	.27 (.53)	0–6	1.04 (1.56)
Color-Word Errors	0–6	1.58 (1.72)	0–10	3.46 (2.32)
Planning and Problem-Solving (<i>a</i> = .49)	-0.91 – 1.03	0.17 (0.54)	-1.40 – 0.85	-0.17 (0.52)
ToL Total Correct	2–7	3.58 (1.58)	1–6	3.42 (1.47)
CLOX Trial 1	9–14	12.38 (1.30)	8–14	11.42 (1.60)
WASI Matrix Reasoning	6–29	18.12 (5.55)	8–25	15.73 (4.85)
Psychomotor Speed and Coordination (<i>a</i> = .53)	-1.30 – 0.58	-0.20 (0.56)	-0.95 – 1.77	0.20 (0.81)
GPT:				
Dominant hand Time	52–84	67.62 (9.03)	58–99	72.92(10.88)

	Control Group (n = 26)		AD Group (n = 26)	
	Range	M (SD)	Range	M (SD)
Non-dominant hand Time	57–96	75.38(10.34)	62–118	83.81(14.91)
CCTT Trail 1 Time	10–37	23.27 (7.41)	8–49	23.73 (9.97)
Attention and Concentration (a = .47)	–1.65 – 1.16	0.08 (0.63)	–1.34 – 0.86	–0.08 (0.61)
AVLT Trial 1	2–11	6.38 (1.86)	2–12	6.65 (1.94)
WISC-IV Coding	29–71	52.69(8.96)	30–67	49.12 (9.38)
CMS Numbers Back	2–8	4.15 (1.67)	2–7	4.04 (1.18)
SCWT Color-Word Correct	18–46	32.12 (7.78)	11–46	29.04 (9.55)
Sequencing Ability (a = .79)	–1.00 – 1.83	–0.13 (0.79)	–0.93 – 3.70	0.13 (1.01)
CCTT:				
Trial 2 time	24–98	46.46(17.80)	30–147	51.27(22.71)
Total errors	0–3	.88 (1.03)	0–4	1.23 (1.24)
Expressive Language (a = .77)	–1.44 – 1.82	0.10 (0.91)	–1.22 – 2.22	–0.10 (0.74)
Phonemic Fluency: total words	14–39	26.92 (7.95)	16–43	25.85 (6.37)
Semantic Fluency: total words	9–25	14.88 (4.03)	9–21	14.15 (3.38)
WASI Vocabulary	17–52	31.65 (9.36)	21–53	29.69 (7.97)
Visuospatial Construction (a = .55)	–2.12 – 1.52	–0.00 (0.84)	–1.09 – 1.00	0.00 (0.60)
ROCF Copy	16–36	30.38 (4.53)	26–35	30.83 (3.02)
CLOX Trial 2	12–15	13.65 (1.06)	12–15	14.04 (.87)
WASI Block Design	9–64	27.62(13.36)	5–47	21.73(10.06)
Visual Memory (a = .83)	–1.75 – 1.25	–0.02 (0.77)	–1.53 – 1.26	0.02 (0.78)
ROCF:				
Immediate Recall	7–30	19.21 (6.94)	9–32	20.02 (6.46)
Delayed Recall	9–31	18.88 (6.72)	9–29	19.35 (6.31)
CMS Dots:				
Trials 1–3	10–24	18.00 (3.98)	8–24	18.27 (4.38)
Immediate Recall	2–8	6.08 (1.67)	3–8	6.23 (1.39)
Delayed Recall	4–8	6.23 (1.34)	1–8	5.92 (1.98)

Note. Data presented are z-scores for composite domains and raw scores for individual tests. CMS = Children's Memory Scale; AVLT = Auditory Verbal Learning Test; ToL = Tower of London; CCTT = Children's Color Trails Test; SCWT = Stroop Color-Word Test; WASI = Wechsler Abbreviated Scale of Intelligence; GPT = Grooved Pegboard Test; ROCF = Rey-Osterrieth Complex Figure test; WISC-IV= Wechsler Intelligence Scale for Children, 4th Edition.

Table 3

Regression analyses of selected neuropsychological domain scores.

	Verbal Story Memory			Self-Monitoring			Psychomotor Speed		
	β	<i>t</i>	sig.	β	<i>t</i>	sig.	β	<i>t</i>	sig.
Step 1									
Constant		1.94	.07		-1.90	.06	-1.47	.15	-1.47
Group	-.36	-2.74	.008**	.36	2.68	.010**	.28	2.07	.04*
Step 2									
Constant		1.91	.06		-1.95	.06		-1.4	.16
Group	-.34	-2.19	.034*	.26	1.68	.10	.35	2.22	.03*
Tobacco Dose	-.05	-.33	.75	.00	.01	.99	-.08	-.54	.60
Other Drugs	-.02	-.12	.91	.23	1.60	.12	-.11	-.73	.47
Step 3									
Constant		.54	.59		.33	.75		-.83	.41
Group	-.31	-2.1	.041*	.28	1.98	.045*	.34	2.23	.03*
Tobacco Dose	-.03	-.19	.85	-.03	-.21	.84	-.12	-.85	.40
Other Drugs	-.04	-.26	.80	.18	1.32	.20	-.12	-.83	.41
Education	.55	3.44	.001**	-.38	-2.45	.018*	-.40	-2.40	.02*
Age	-.29	-1.71	.10	.10	.64	.52	.28	1.57	.12
Language	.08	.65	.52	-.34	-2.75	.009**	-.25	-1.82	.08
	R ² = .13 for Step 1 R ² = .00 for Step 2 R ² = .19 for Step 3 R ² = .33 for Step 3 F=4.30**	R ² = .13 for Step 1 R ² = .04 for Step 2 R ² = .21 for Step 3 R ² = .38 for Step 3 F=5.06**	R ² = .08 for Step 1 R ² = .02 for Step 2 R ² = .17 for Step 3 R ² = .26 for Step 3 F=3.42*						

Notes:
 = change
 * p<.05
 ** p<.01