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Neural and psychological characteristics of college students with alcoholic parents differ depending on current alcohol use

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ABSTRACT

A significant proportion of college students are adult children of an alcoholic parent (ACoA), which can confer greater risk of depression, poor self-esteem, alcohol and drug problems, and greater levels of college attrition. However, some ACoA are resilient to these negative outcomes. The goal of this study was to better understand the psychobiological factors that distinguish resilient and vulnerable college-aged ACoAs. To do so, scholastic performance and psychological health were measured in ACoA college students not engaged in hazardous alcohol use (resilient) and those currently engaged in hazardous alcohol use (vulnerable). Neural activity (as measured by functional magnetic resonance imaging) in response to performing working memory and emotion-based tasks were assessed. Furthermore, the frequency of polymorphisms in candidate genes associated with substance use, risk taking and stress reactivity were compared between the two ACoA groups. College ACoAs currently engaged in hazardous alcohol use reported more anxiety, depression and posttraumatic stress symptoms, and increased risky nicotine and marijuana use as compared to ACoAs resistant to problem alcohol use. ACoA college students with current problem alcohol showed greater activity of the middle frontal gyrus and reduced activation of the posterior cingulate in response to visual working memory and emotional processing tasks, which may relate to increased anxiety and problem alcohol and drug behaviors. Furthermore, polymorphisms of cholinergic receptor and the serotonin transporter genes also appear to contribute a role in problem alcohol use in ACoAs. Overall, findings point to several important psychobiological variables that distinguish ACoAs based on their current alcohol use that may be used in the future for early intervention.

1. Introduction

One-fourth to one-third of college students meet the criteria to be an adult child of an alcoholic parent (ACoA; Kelley et al., 2008). The impact of growing up with one or more alcoholic parent/s can be traumatic for a child (Mackrill and Hesse, 2011). In fact, ACoA status is correlated with increased occurrences of depression (Kelley et al., 2010; Klostermann et al., 2011), anxiety (Woodford et al., 2011), alcohol abuse (LaBrie et al., 2007), low self-esteem (Neighbors et al., 2004), greater risk for developing substance use problems (Yoon et al., 2013; Eddie et al., 2015), and higher levels of college attrition (Kitsantas et al., 2008). Specifically, ACoA have been reported to have greater difficulty in adjusting to college life during their freshman year than students from non-alcoholic homes (Porter and Pryor, 2007). The difficulties in college experienced by many ACoA students may be due to poor psychological and/or social functioning (Kelley et al., 2008; Hill et al., 2001), cognitive difficulties affecting scholastic performance (Schroeder and Kelley, 2008), or a combination of these factors, which are likely to be also compounded by the propensity for hazardous alcohol use in this population (Yoon et al., 2013; Eddie et al., 2015).

The majority of research investigating neural-behavioral correlates of ACoA relates to reward sensitivity, motivation, behavioral disinhibition/impulsivity and risky decision making, given that these are risk factors for alcohol use disorder (AUD) (Schweinsburg et al., 2004; Bjork et al., 2008; Acheson et al., 2009; Andrews et al., 2011; Cservenka and Nagel, 2012; Yau et al., 2012; Dager et al., 2013; Kareken et al., 2013; Weiland et al., 2013). Studies comparing adolescents with a familial history of AUD (1st or 2nd degree biological relative without

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Table 1
Demographic and assessment measures for adult children of alcoholic parents based on current alcohol status.

Demographic measure		Non-hazardous	Hazardous
Gender (%)	Male	36.36	42.11
	Female	63.64	57.89
Age (range)		19–24	18–25
Race (%)	White	81.82	94.74
	Native American	0	5.26
	Asian American	4.55	0
	Multiracial	13.64	0

Assessment (Range)	Scale	Non-hazardous	Hazardous	t value	p value
CAST (0 − 30)		19.36 ± 0.99	20.00 ± 1.60	0.348	0.729
AUDIT (0–16 +)		2.68 ± 0.50	15.21 ± 1.51	− 8.337	*< 0.001*
ASSIST[a]**(0–27 +)**	Tobacco	3.82 ± 1.30	12.32 ± 2.26	− 3.372	*0.002*
	Alcohol	6.50 ± 0.92	22.53 ± 2.56	− 6.238	*< 0.001*
	Marijuana	2.64 ± 0.73	7.42 ± 1.52	− 2.965	*0.005*
	Cocaine	0.27 ± 0.27	1.74 ± 1.48	− 1.041	0.304
	Amphetamines	0.41 ± 0.41	2.11 ± 1.53	− 1.142	0.13
	Inhalants	0.00 ± 0.00	0.58 ± 0.33	1.907	0.064
	Sedatives	0.14 ± 0.14	4.63 ± 1.98	− 2.446	*0.019*
	Hallucinogens	0.00 ± 0.00	2.32 ± 1.34	− 2.316	0.07
	Pain Medication/opioids	0.27 ± 0.27	1.68 ± 0.79	− 1.786	0.082
Duke health profile (0 − 100)	Physical health	73.64 ± 4.03	64.74 ± 4.48	1.48	0.147
	Mental health	72.73 ± 5.06	52.11 ± 5.05	2.868	*0.007*
	Social health	71.36 ± 4.43	54.74 ± 5.43	2.397	*0.021*
	General health	72.08 ± 4.10	57.48 ± 4.27	2.46	*0.018*
	Perceived health	77.27 ± 5.43	50.00 ± 8.55	2.767	*0.009*
	Self esteem	68.18 ± 5.45	55.26 ± 5.37	− 1.677	0.102
	Anxiety	34.84 ± 5.12	49.46 ± 5.13	− 2.006	0.052
	Depression	32.73 ± 5.27	48.95 ± 4.64	2.275	*0.029*
	Anxiety and depression	32.15 ± 4.90	51.76 ± 5.44	− 2.686	*0.011*
	Pain	29.55 ± 6.29	47.37 ± 8.09	1.762	0.086
	Disability	0.00 ± 0.00	15.79 ± 5.48	− 3.108	*0.004*
BDI-II (0–63)		11.5 ± 1.94	19.53 ± 3.08	− 2.27	*0.029*
BAI (0–63)		8.09 ± 1.57	13.68 ± 2.15	− 2.136	*0.039*
PCL (17–85)		36.64 ± 3.25	47.95 ± 3.77	− 2.286	*0.028*
GPA (0–4)		3.45 ± 0.10	3.15 ± 0.20	− 2.193	*0.017*
ACT (0–36)		23.85 ± 0.83	24.10 ± 0.99	0.168	0.868
SAT Composite (0–2400)		1475.5 ± 113.22	1436 ± 82.73	0.087	0.939

Abbreviations: Children of Alcoholics Screening Test (CAST); Alcohol Use Disorders Identification Test (AUDIT); Alcohol, smoking and substance involvement screening test (ASSIST); Beck test for depression (BDI-II); Beck test for anxiety (BAI); PTSD Check List (PCL); Grade point average (GPA); American college testing (ACT); Scholastic assessment test (SAT).

[a] Scores of 0–3 suggest low risk, scores of 4–26 suggest moderate risk, and scores > 27 suggest high risk of experiencing severe substance use issues.

necessarily being raised by this relative) to those without such history have provided some indication that family history alone alters neural functioning during emotional or cognitive processing. For example, the frontal cortex (medial frontal gyrus, dorsolateral frontal cortex, dorsal anterior cingulate) is necessary for executive functioning that is critical for day-to-day and scholastic performance of tasks like working memory, and is less active in adolescents with family history of AUD (Cservenka and Nagel, 2012; Mackiewicz Seghete et al., 2013). Likewise, the activity of subcortical regions such as the amygdala, necessary for appropriate emotional processing and responding, is also reduced in young adults with a family history of AUD (Glen et al., 2007; Cacciaglia et al., 2013), suggesting reduced ability to engage appropriately with emotional stimuli. Thus, a family history of AUD may confer psychosocial, cognitive, and alcohol problems on offspring via poor frontal cortical-to-subcortical neural functioning.

Adult offspring of alcoholics have a 2.5 to 4.4 fold increase in the chance of developing AUD in their lifetime as compared to children of non-AUD parents, with greatest risk conferred by being raised by two alcoholic parents (Yoon et al., 2013). This risk is likely conferred by an interaction between genetic and environmental factors, given that genetic influences can contribute to alcohol use (Goate and Edenberg, 1998; Köhnke, 2008) and being raised by an alcoholic parent is associated with increased adverse childhood experiences, increased psychosocial behaviors associated with alcoholism in youth, and greater risk for adult AUD (Anda et al., 2002; Hussong et al., 2007). However, most research using youth or adult children of alcoholics normally involves individuals who are selected based on family history of AUD with no consideration for whether they were raised by an individual with AUD. Furthermore, participants in such studies are often without extensive current alcohol problems themselves, likely representing a resilient subpopulation of ACoA (Heitzeg et al., 2008). For example, previous research examining polymorphisms in genes of ACoA have shown relationships with brain derived neurotrophic factor polymorphisms and executive functioning (Benzerouk et al., 2013), and dopamine D2 receptor A1 allele expression with risk seeking (Ratsma et al., 2001). However, these prior studies used resilient, healthy ACoA so it is not clear whether genetic polymorphisms contribute to at-risk and resilient ACoA phenotypes.

In the few neural studies including adolescents with both low *and* high alcohol problems who have a family history of AUD, there appear to be effects on neural functioning that can be attributed to current alcohol use and other effects attributable to a family history of alcoholism (Heitzeg et al., 2008, 2010). For example, failure to deactivate the frontal cortex during response inhibition in a go/no-go task in adolescents with a positive family history of AUD is only apparent with current alcohol problems, whereas failure to deactivate the striatum is observed in adolescents with a family history of AUD regardless of current alcohol use (Heitzeg et al., 2010). Therefore, to effectively