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Associations Between Parental Alcoholism And Adult Internalized And Externalized Indicators Of Maladjustment

Stephanie Brezinski

ASSOCIATIONS BETWEEN PARENTAL ALCOHOLISM AND ADULT INTERNALIZED AND EXTERNALIZED INDICATORS OF MALADJUSTMENT

by

Stephanie Joy Brezinski
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This thesis, submitted by Stephanie Joy Brezinski in partial fulfillment of the requirements for the Degree of Master of Arts from the University of North Dakota, has been read by the Faculty Advisory Committee under whom the work has been done and is hereby approved.

_____
Alan King (Chairperson)

_____
Thomas Petros

_____
F. Richard Ferraro

This thesis is being submitted by the appointed advisory committee as having met all of the requirements of the School of Graduate Studies at the University of North Dakota and is hereby approved.

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Chris Nelson
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Title	Associations Between Parental Alcoholism and Adult Internalized and Externalized Indicators of Maladjustment
Department	Psychology
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Stephanie Joy Brezinski
June 18, 2019

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ABSTRACT

Associations between parental alcoholism and internalized and externalized indicators of maladjustment in adult children of alcoholics were examined. College students (N = 2,531) and respondents from the general population (N = 703) provided self-reports of various internalized and externalized indicators of maladjustment. Several hypotheses were tested: (1) CAST scores will be positively associated with symptom severity; (2) Combined maternal and paternal alcoholism will be associated with the most severe maladjustment compared to ACAs from a single parent and/or non-ACAs; (3) Male ACAs will exhibit relatively more externalized symptoms than internalized symptoms and female ACAs will exhibit relatively more internalized symptoms than externalized symptoms; (4) Paternal alcoholism will be more strongly associated with externalized symptoms as opposed to internalized symptoms, particularly among male ACAs, and maternal alcoholism will be more strongly associated with internalized symptoms as opposed to externalized symptoms, particularly among female ACAs. Overall, consistent with previous literature, the present study found mixed results regarding the relationship between differential gender effects on ACAs risk of various internalized and externalized maladjustment indicators; however, our results from both samples provide further evidence that ACAs, regardless of gender and/or ACA status, appear to be at increased risk of both internalized and externalized forms of maladjustment compared to non-ACAs.

CHAPTER I

INTRODUCTION

In 2016, an estimated 14.6 million Americans, 18 years and older, met criteria for alcohol use disorder (AUD; Substance Use and Mental Health Services Administration, 2016). In 2017, approximately 7.5 million children under the age of 17 lived with at least one parent with an AUD (Lipari & Van Horn, 2017). These individuals are commonly referred to as adult children of alcoholics (ACAs). Anda et al. (2002) reported that ACAs have a 3.5-5.6-fold risk of developing alcoholism, regardless if they were exposed to any other form of adverse childhood experiences (ACEs). According to the literature ACAs are at such an increased risk for developing alcohol use problems due to biological predispositions as well as environmental risk factors.

Alcohol-Related Risks

The literature identifies various genetic influences that may be involved in the development of alcoholism. For example, Dager et al. (2015) found a correlation between amygdala volume and the risk of AUD. The researchers found that individuals with AUD had smaller amygdala volumes compared to non-AUD individuals; however, they found that non-AUD individuals with a family history of AUD also showed reduced amygdala volume. This suggests that reduced amygdala volume is a pre-existing structural difference rather than alcohol-induced damage (Dager et al., 2015). Another study by Herting, Schwartz, Mitchell, and Nagel (2010) found that ACAs have a disrupted white matter microstructure. The authors reported that this disruption may decrease ACAs efficiency of cortical processing, which may increase their susceptibility of developing AUD (Herting, Schwartz, Mitchell, & Nagel, 2010). Family density of alcohol problems

also appears to increase a ACAs risk of developing alcohol use problems (Anda et al., 2002; Capone & Wood, 2008; Corte & Becherer, 2007; King & Chassin, 2007; Merikangas & Avenevoli, 2000; Perkin & Berkowitz, 1991; Schumacher, 1990; Slutske et al., 2008). Dawson, Harford, and Grant (1992) found that a ACAs risk of developing alcohol dependence increased depending on the relationship to the alcoholic family member. More specifically, a ACAs risk was increased by 45% with only second or third degree relatives with alcoholism, by 86% for individuals who only had first degree relatives with alcoholism, and by 167% for individuals who had first, second, and third degree relatives with alcoholism (Dawson, Harford, & Grant, 1992). This is consistent with Perkin and Berkowitz (1991) who reported multigenerational alcoholism increased a ACAs risk of alcohol use problems. ACAs with an alcohol-misusing parent and alcohol-misusing grandparent reported heavier alcohol consumption and more frequent intoxication compared to both non- ACAs and ACAs with just an alcohol-misusing parent or alcohol-misusing grandparent (Perkin & Berkowitz, 1991). Schumacher (1990) also found that ACAs with two alcoholic parents significantly increased the amount of daily alcohol consumption compared to non- ACAs and ACAs with only one alcoholic parent. The author found that ACAs with two alcoholic parents drank an average of 3.2 oz. of alcohol per day, three times the average amount of the general population (.94 oz.), compared to ACAs with only maternal alcoholism (1.1 oz.), only paternal alcoholism (1.0 oz.), and neither parent alcoholic (1.1 oz; Schumacher, 1990). Several studies demonstrated having two alcoholic parents greatly increased a ACAs risk of not only alcohol misuse, but also mental health issues, drug misuse, and suicide attempts (Anda et al., 2002; Slutske et al., 2008). Other research has found the higher the family density of

alcoholism, the earlier the age of onset of drinking (Hill, Shen, Lowers, and Locke, 2000). It is important to consider the severity of risk for ACAs with a family density of alcohol problems, especially ACAs with two alcoholic parents, because not only are they at higher risk genetically for alcohol use problems, but also at an increased risk environmentally (Slutske et al., 2008).

Several studies found that, compared to non- ACAs, ACAs with one or two alcohol abusing parents were at significantly greater risk of suffering from multiple ACEs (i.e. physical abuse, emotional abuse, sexual abuse, emotional neglect, physical neglect, or parental separation or divorce; Dube et al., 2001; Thompson, Alonzo, Grant, & Hasin, 2013). Several studies have demonstrated that exposure to ACEs increases the likelihood of alcohol-related problems (Grayson & Nolen-Hoeksema, 2005; Harter & Taylor, 2000; Lown, Nayak, Korcha, & Greenfield, 2011; Sheridan, 1995; Shin, Hassamal, & Peasley Groves, 2015). These findings suggest the risk of developing alcohol use problems is especially problematic for ACAs who not only have at least one alcoholic parent, but also experience other forms of ACEs.

Psychopathology-Related Risks

In addition to an increased risk of developing alcohol-related problems, the literature indicates that ACAs are also at a heightened risk of developing psychopathology, especially ACAs with a higher family density of alcoholism (Alterman, Renner, Cacciola, Mulvaney, & Rutherford, 2000; Barnow, Schuckit, Smith, Preuss, & Danko, 2002; Corte & Becherer, 2007; Diaz et al., 2008; Earls, Rich, Jung, & Cloninger, 1998; Haber et al., 2010; Molina, Donovan, & Belendiuk, 2010). Much of the literature demonstrates that ACAs are at an increased risk of various internalizing (e.g. depression,

anxiety) and externalizing (e.g. aggression, conduct disturbance, alcohol use, delinquency, ADHD) indicators of maladjustment. There is extensive literature demonstrating that ACAs are at higher risk of developing depression and/or anxiety and experiencing significantly less satisfaction with life (Belliveau & Stoppard, 1995; Corte & Becherer, 2007; Chen & Weitzman, 2005; Cuijpers, Langendoen, & Bijl, 1999; Diaz et al., 2008; Hall, Bolen, & Webster, 1994; Kelley et al., 2010; Klosterman et al., 2011; Reich, Earls, Frankel, & Shayka, 1993; Tomori, 1994). There is considerable evidence supporting the relationship between ACAs risk of developing various externalizing symptoms. Several studies have indicated an increased risk of conduct disturbance, aggression, anger, substance use, delinquency, ADHD, and oppositional defiant disorder in ACAs (Earls, Reich, Jung, & Cloninger, 1988; Edwards, Eiden, Colder, & Leonard, 2006; Haber, Jacob, & Heath, 2005; Haber et al., 2010; Hussong et al., 2007; Kuperman, Scholosser, Lidral, & Reich, 1999; Merikangas & Avenevoli, 2000; Obot & Anthony, 2004; Reich, Earls, Frankel, & Shayka, 1993; Tomori, 1994).

There is inconsistency in the literature evaluating ACAs risk of reporting psychopathology. Several studies have demonstrated no differences between ACAs and non- ACAs on psychopathology risk (Bijttebier, Goethals, & Ansoms, 2006; Drapkin, Eddie, Buffington, & McCrady, 2015; Jones, Perera-Diltz, Salyers, Laux, & Cochrane, 2007; Nicholas & Rasmussen, 2006; Reich, Earls, Frankel, & Shayka, 1993; Schumacher, 1990). Some research suggests that depressive symptoms reported by ACAs may even be better attributed to a history of parental mood disorders rather than to a history of parental alcoholism (Preuss, Schuckit, Smither, Barnow, & Danko, 2002). The

lack of consensus on the association between parental alcoholism and its effects on ACAs psychopathology highlights an important direction for future research.

Sex Differences in Parent

Most of the available literature indicates that paternal alcoholism significantly increases a ACAs risk of alcohol consumption. Corte and Becherer (2007) found having a family history of paternal alcoholism increased the likelihood of offspring problematic alcohol consumption whereas a family history of maternal alcoholism did not. Chassin, Curran, Hussong, and Colder (1996) found a strong relationship between paternal alcoholism and adolescent substance use. The authors found that adolescents with a history of paternal alcoholism were not only more likely to use substances, but also increased their substance use more rapidly compared to non- ACAs (Chassin, Curran, Hussong, & Colder, 1996). Another study showed that paternal alcoholism is more strongly associated with offspring alcohol problems, particularly for males (Ohanessian & Hesselbrock, 2004), while other research suggests paternal alcoholism is correlated with an increased risk of alcohol consumption for female offspring only (Coffelt et al., 2006). These studies illustrate how a history of paternal alcoholism can lead to an increased risk of problematic alcohol consumption in both male and female ACAs. Other research suggests that maternal alcoholism is more strongly associated with ACAs alcohol consumption. For instance, Pearson, D'Lima, and Kelley (2012) found that maternal alcoholism was most strongly associated with problematic alcohol use, particularly for female offspring. In this same study, male offspring were at increased risk for problematic alcohol use when both parents were alcoholic (Pearson, D'Lima, & Kelley, 2012). Another study found that ACAs with alcoholic mothers were at greater

risk for problematic alcohol consumption compared to ACAs with alcoholic fathers (Weitzman & Wechsler, 2000). These studies demonstrate how a history of maternal alcoholism can also lead to an increased risk of problematic alcohol consumption in both male and female offspring.

In contrast to the research on ACA alcohol consumption, much of the research on ACA risk of developing psychopathology suggests maternal alcoholism is more predictive of increased risk. Corte and Becherer (2007) demonstrate a relationship between maternal alcoholism and internalizing problems, specifically, major depressive disorder, generalized anxiety disorder, and obsessive-compulsive disorder. Morgan, Desai, and Potenza (2010) found that while both male and female offspring were at increased risk of psychopathology regardless of the sex of the alcoholic parent, female offspring with a history of maternal alcoholism were at a considerably greater risk of developing psychopathology compared to male offspring. Anda et al. (2002) found that ACAs with two alcoholic parents or just alcoholic mothers were at increased risk of psychopathology, substance misuse, and suicide attempts. Another study found that maternal alcoholism was associated with higher rates of both externalizing and internalizing symptoms, with particularly higher rates of externalizing behavior problems in young ACAs (Mesman, Edge, McKelvey, Pemberton, & Holmes, 2017). These studies demonstrate how a history of maternal alcoholism is associated with an increased risk of ACA psychopathology.

Other research suggests paternal alcoholism is more strongly associated with ACA risk of developing psychopathology. One study demonstrated that not only does paternal alcoholism significantly increase a ACAs likelihood of substance use, but was

also found to be a strong predictor of anxiety and mood disorders (Cuijpers, Langendoen, & Bijl, 1999). Another study demonstrates an increased risk of conduct disorder in offspring with a history of paternal alcoholism compared to offspring without a history of paternal alcoholism (Haber et al., 2010). Some research suggests that ACAs with both maternal and paternal alcoholism were at greatest risk for both internalizing and externalizing disorders, particularly anxiety disorders and ADHD (Earls, Reich, Jung, Cloninger, 1988). Taken together these studies demonstrate an inconsistency in the literature related to the relationship between sex of the alcoholic parent and risk of ACA problematic alcohol consumption and psychopathology, providing evidence of the need for future research to examine how sex of the alcoholic parent relates to offspring risk of problematic alcohol consumption and psychopathology.

Sex Differences in Offspring

According to the literature male offspring are more likely to experience conduct disturbance and aggression compared to female offspring. Edwards, Eiden, Colder, and Leonard (2006) found that male ACAs demonstrated higher levels of childhood aggression compared to female ACAs. The researchers also found that males with two alcoholic parents exhibited aggression longer than males in the control group (Edwards, Eiden, Colder, & Leonard, 2006). Kearns-Bodkin and Leonard (2008) also showed that men with a history of parental alcoholism, particularly maternal alcoholism, were more physically aggressive in their marriages. These studies demonstrate how a history of parental alcoholism can lead to aggressive behavior in male offspring. Related to conduct disturbance, Haber et al. (2010) demonstrated that male ACAs exhibited significantly higher rates of conduct disorder compared to male non- ACAs.

Based on the literature female offspring are more likely to experience depression and anxiety symptoms compared to male offspring. Several studies have found that not only does being female independently predict a greater risk of depression and anxiety symptoms, but also a family history of alcoholism, particularly on the maternal side, increases the risk (Abdel-Kalack & Alansari, 2004; Corte & Becherer, 2007). Christensen and Bilenberg (1999) also demonstrated that females with a history of maternal alcoholism had higher scores on depression and internalizing symptoms on the Child Behavior Checklist compared to males.

There is inconsistency in the literature regarding risk of alcohol consumption depending on the sex of the ACA. Several research studies indicate that male offspring are at a greater risk of alcohol use compared to female offspring. Schumacher (1990) found that while ACA, regardless of sex, were at increased risk of alcohol consumption, this result was most salient for male offspring. Male ACAs reported an average consumption of 2.1 oz. of alcohol each day compared to female ACAs who reported an average consumption of 1.1 oz. This suggests that, on average, male ACAs reported drinking more than twice that of the general population (.94 oz.; Schumacer, 1990). Alterman, Cacciola, Mulvaney, Rutherford, and Langenbucher (2002) found that male offspring with a high family density of alcoholism (i.e. two first degree relatives or two second/third degree relatives) were at the greatest risk of alcoholism themselves. Pearson, D'Lima, and Kelley (2012) also found that male ACAs with two alcoholic parents were at the highest risk of problematic alcohol use. Another study found that while both male and female ACAs were at an increased risk for problematic alcohol use, males were at a particularly high risk when there was a history of maternal alcoholism (Weitzman &

Wechsler, 2000). Several studies suggest that female ACAs may be at a higher risk for alcohol use compared to male ACAs. Curran et al. (1998) found female ACAs were at greater risk of alcohol use disorders compared to male ACAs. LaBrie, Kenney, Lac, & Migliuri (2009) demonstrated that female college students with a family history of alcoholism drank more heavily than peers without a family history of alcohol problems.

Other research suggests that both offspring are at risk for problematic alcohol use. Coeffelt et al. (2006) study showed that maternal alcoholism increases the risk of alcohol use in both male and female offspring. Another study found that paternal alcoholism was associated with male offspring alcohol use whereas both paternal and maternal alcoholism were associated with female offspring use (Crum & Harris, 1996). There is a lack of consensus on the relationship between parental alcoholism and ACA problem alcohol consumption and psychopathology, specifically as it relates to distinguishing between sex of the alcoholic parent and sex of the offspring. Future research is necessary to try and refine the equivocal literature.

Project Objectives

Analysis in both samples will rely on a 2 (gender) x 4 (maternal, paternal, both, or neither ACA status) x (internalized versus externalized aggregates) mixed group design. This design will allow the examination of associations between parental alcoholism and adult internalized (depression, anxiety, body image preoccupation, dispositional mindfulness, life satisfaction, and self-esteem; Table 1) and externalized (alcohol consumption/abuse, trait hostility, mood volatility, conduct disturbance prior to age 15, lifetime aggression, and arrest history; Table 2) indicators of maladjustment. A similar follow-up analysis using an independent sample of national respondents will attempt to

replicate the college findings with the inclusion of several additional criterion measures. ACA status will be defined by paternal and/or maternal CAST scores of six or higher. A number of hypotheses will be tested: (1) CAST scores will be positively associated with symptom severity; (2) Combined maternal and paternal alcoholism will be associated with the most severe maladjustment compared to ACAs from a single parent and/or non-ACAs; (3) Male ACAs will exhibit relatively more externalized symptoms than internalized symptoms and female ACAs will exhibit relatively more internalized symptoms than externalized symptoms (gender x symptom cluster interaction); (4) Paternal alcoholism will be more strongly associated with externalized symptoms as opposed to internalized symptomatology, particularly among male ACAs and maternal alcoholism will be more strongly associated with internalized symptoms as opposed to externalized symptoms, particularly among female ACAs (gender x ACA status x symptom cluster interaction).

Analytic Strategy

Bivariate correlation analyses will be used in both samples to quantify the extent to which dimensional CAST scores are associated with each measures of adult maladjustment. An omnibus Multiple Analysis of Variance will be conducted followed by post-hoc ANOVAs employing a 2 (gender) x 4 (maternal, paternal, both, or neither ACA status) x (internalized versus externalized aggregates). An aggregate internalized and externalized score will be calculated for each participant using a standard score average. Internalized and externalized aggregate scores will be used as a within group factor to test hypotheses regarding differential impacts of ACA and/or gender. ACA status may have to be restricted to a dichotomous factor (ACA versus controls) in the

national sample if all eight cells cannot be populated with a minimum of 15 respondents. Relative risk analyses also attempted to estimate some practical implications of ACA status on several maladjustment risk factors.

CHAPTER II

METHODS

Participants and Procedure

Archival College Sample.

This sample ($N = 2, 531$) consists of college undergraduates who volunteered for extra credit toward a psychology course. Participants used the web platform SONA Systems to access the Qualtrics survey. A subset of initial respondents were excluded ($n = 198$) prior to analysis due to missing responses in the CAST questionnaire which was the focus of this study. This project was approved by the IRB and informed consent was collected from all participants. The results of this sample can be best generalized to college undergraduate ($M_{age} = 20.11$, $SD = 4.06$) men (24.0%) and women (76.0%). Ethnic diversity was limited in this Midwestern sample (Caucasian, 89.1%; African American, 2.5%; Asian, 2.1%; Native-American, 1.8%; Hispanic, 1.6%; Other, 3.1%).

National Sample.

Amazon's Mechanical Turk (MTurk) was used to generate a national sample ($N = 703$). This sample varied in gender (58.5%, women; 41.5%, men), age ($M_{age} = 36.19$, $SD = 12.15$) and ethnicity (White, 77.2%; Black, 8.0%; Hispanic, 5.3%; Asian, 6.0%; American Indian, 0.9%; & Multi-Racial, 1.9%). MTurk has been reviewed favorably as a crowdsourcing research platform (Buhrmester, Kwang, & Gosling, 2011; Gosling, Vazire, Srivastava, & John, 2004; Paolacci, Chandler, & Ipeirotis, 2010). A concern regarding this methodology has been the potential threat posed by server farms that rely on Virtual Private Servers (VPS) allowing individuals to disguise their origination and geolocation (Kennedy, Clifford, Burleigh, Waggoner, & Jewell, 2018).

Inclusion Criteria. The present MTurk sample was restricted to respondents who were at least 18 years of age, were located in the United States, had an HIT approval rate greater than 95%, and had completed at least 100 HITs.

Exclusion Criteria. Proxy/VPN detection software was used (https://iphub.info) to identify and exclude a subset of initial respondents who attempted to access the survey from outside of the United States or who were trying to disguise their international origin by using a VPS/VPN/Proxy (Burleigh, Kennedy, & Clifford, 2018). This protocol excludes respondents whose IP address is traced to a non-US location or is traced to a non-residential source, which suggests the respondent is likely using a VPS or proxy to mask their location and therefore inclusion criteria cannot be confirmed (Kennedy, Clifford, Burleigh, Jewell, & Waggoner, 2018). The protocol also flags, but does not exclude, respondents whose IP addresses are traced to a residential source, but the source has been reported as a potential VPS. These respondents are allowed to complete the survey, but some interpretive caution is warranted as there is uncertainty about whether a VPS is being used (Kennedy et al., 2018). With that said, given the limited research on the effectiveness of the proxy/VPN detection software, the protocol was adapted to minimize the risk of false positive exclusions by allowing MTurk workers flagged with IP addresses traced to non-residential sources (i.e., VPS/VPN/Proxy) to provisionally complete the survey. Respondents with IP addresses traced to non-US locations were still excluded from survey completion (n = 50).

Upon manually reviewing the origin of these non-residential IP addresses, all of the respondents who would have been kicked out by the original protocol were excluded from the final data set for either being traced back to an identified VPS server farm or

proxy website (n = 60). There was also a subset of respondents who were flagged as possible VPS usage and completed the survey who were traced back to server farms or proxy websites and were excluded from the final data set (n = 8). Additional precautions were taken to exclude respondents who failed an open-ended response question (n = 4), instructional manipulation check (n = 1), or whose IP address identified them as having both a duplicate IP address and geolocation (n = 22). Since IP addresses can only originate from one router, there is no way of confirming whether these respondents are individuals within the same household/organization or if they are the same individual completing the survey more than once (Ahler, Roush, & Sood, 2019).

Measures

An attempt was made to select criterion measures that were with literature definitions of either internalized or externalized symptoms of maladjustment. Two initial clusters were assembled with the intention of verifying whether or not the collective criterion measures could be meaningfully differentiated into internalized versus externalized symptom clusters. A preliminary verification of sample adequacy was conducted, Kaiser-Meyer Olkin = 0.782; Bartlett's Test of Sphericity, $\chi 2$ (91) = 4239.51, $p < 0.001$. This was followed by a factor analysis of the 12 maladjustment indicators (Eigenvalue > 1; factor loadings > .40; pairwise exclusions for missing data) using a Varimax rotation that forced the extraction of two principle components. An internalized maladjustment factor was extracted (α = .70; 21.04%) successfully using 6 of the indicators (DEPR, ANX, DIAG, TXS, FAT, LSI). A weak externalized maladjustment factor (α = .18; 14.69%) arose from six of the seven remaining indicators (KAT, BPAQ,

VOL, NARC, ANTI & MIND). Table 1 illustrates the factor loadings for a two-factor solution. Conduct disturbance (COND) failed to load sufficiently on either dimension.

Anxiety Symptoms (ANX)

A 13-item, customized survey was used in the college sample to measure anxiety symptoms using the DSM-5 criteria for panic disorder. Each of the symptoms were scored on a 5-point Likert scale ranging from 0 (Not Present) to 4 (Present Daily with Significant Distress). A total score was generated from the sum of these items.

Buss-Perry Aggression Questionnaire (BPAQ)

The Buss-Perry Aggression Questionnaire (BPAQ) is 29-item, self-report instrument designed to assess physical aggression, verbal aggression, trait anger, and trait hostility. Reliability has been established with scores ranging from .72-.89 for the subscales (Buss & Perry, 1992). Several studies have linked BPAQ scores to aggressive, angry, and bullying behavior (Archer & Webb, 2006; Gerevich, Bacskai & Czobor, 2007; Palmer & Thakordas, 2005).

Children of Alcoholics Screening Test (CAST)

A 30-item, self-report assessment tool developed to identify individuals who were raised by at least one alcoholic parent (Jones, 1983). The individual completes the assessment for both the mother and the father answering questions such as: *did you ever wish that a parent would quit drinking* and *do you ever resent a parent's drinking*? Respondents answer in the affirmative and leave the question blank if not applicable. Scores range from 0 to 30, with scores of six and above indicating an alcohol abusing parent. Several studies have demonstrated well-established reliability and validity for the

use of the CAST in research settings (Charland & Cote, 1998; Clair & Genest, 1992; Dinning & Berk, 1989; Sheridan, 1995; Roosa, Sandler, Beals, & Short, 1988).

Conduct Disorder (COND)

Using a customized survey to measure the DSM-5 criteria for Conduct Disorder (COND), a history of CD symptoms prior to age 15 will be assessed. Respondents answer questions such as *have you ever started physical fights at school* and *have you ever deliberately destroyed others' property* by selecting either (a) no, (b) yes, before age 15, or (c) yes, at or after age 15. Each of the 15 symptoms will be scored as 0 or 1 with a total score generated from the sum.

Depression Symptoms (DEP)

A 12-item, customized survey was used in the college sample to measure depression symptoms using the DSM-5 criteria for major depressive disorder. Each of the symptoms were scored on a 5-point Likert scale ranging from 0 (Not Present) to 4 (Present Daily with Significant Distress). A total score was generated from the sum of these items.

Goldfarb Fear of Fat Scale (FAT)

The Goldfarb Fear of Fat Scale (GFFS) is a 10-item, self-report measure assessing fear of weight gain (Goldfarb, Dykens, & Gerrard, 1985). The items are scored using a 4-point Likert scale ranging from 1 (very untrue) to 4 (very true) with higher scores suggesting greater fear of gaining weight. Reliability and validity have been widely established (Anderson, Williamson, Duchmann, Gleaves, & Barbin, 1999; Cook, et al., 2013; Goldfarb, Dykens, & Gerrard, 1985; Lewis, Cash, Jacobi, & Bubb-Lewis, 1997; Morrison & O'Connor, 1999; Osman, Chiros, Guiterrez, Kopper, & Barrios, 2001;

Rushford, 2006).

Hypomanic Personality Scale – Mood Volatility Factor (VOL)

The Mood Volatility factor of the Hypomanic Personality Scale provides an estimate of affective, behavioral, and/or cognitive instability of the respondent (Eckbald & Chapman, 1986). The items are scored using a dichotomous format (T/F) with scores ranging from 0 to 15. Extensive reliability and validation have been demonstrated (Eckbald & Chapman, 1986; Schalet, Durbin, & Revelle, 2011; Stanton et al., 2017; Walsh, DeGeorge, Barrantes-Vidal, & Kwapil, 2015).

Khavari Alcohol Test (KAT)

The Khavari Alcohol Test (KAT) is a self-report instrument estimating the quantity and frequency of both typical alcohol consumption and instances of heavy alcohol consumption. Using 12 oz. of beer, 6 oz. of wine, and one 2 oz. shot of liquor as a single drink estimate, respondents answer the quantity of alcohol consumption questions *how many do you usually consume when drinking* and *what is the most drinks you usually consume when drinking*? Using the following frequency index: daily, 3-4 times per week, twice a week, once a week, 3-4 times a month, twice a month, once a month, 3-4 times a year, twice a year, once a year, or not currently drinking, respondents provide answers to the frequency of alcohol consumption questions *how often do you usually drink alcohol* and *how often do you consume this "most" amount*? This measure represents an abbreviated various of the original KAT (Khavari & Farber, 1978).

Levenson Self Report Psychopathy Scale (ANTI*)*

The Levenson Self-Report Psychopathy Scale has become a widely used brief self-report index of psychopathic tendencies (Levenson, Kiehl, & Fitzpatrick,

1995). Recent factor analytic analyses summarize convergent and discriminant validity for this measurement index that reflects some of the content area intended for the PCL-R (Salekin, Chen, Sellbom, Lester, & MacDougall, 2014).

Michigan Alcoholism Screening Test (MAST)

The MAST is a 25-item, self-report instrument for screening alcoholism risk (Selzer, 1971). The instrument consists of yes/no questions such as: *have you gotten into fights while drinking? have you ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking?* According to Selzer (1971) ratings of 0-3 points suggests no alcoholism risk, four points suggests questionable alcoholism risk, and 5+ points suggests considerable alcoholism risk. Several studies have identified the MAST as an appropriate screening measure for identifying problematic drinking and alcoholism with well-established reliability and validity (Minnich, Erford, Bardhoshi, & Ataley, 2018; Teitelbaum & Carey, 2000; Zung, 1982).

Mindfulness Attention Awareness Scale (MIND)

The Mindfulness Attention Awareness Scale is a 15-item, self-report measure used to estimate the extent to which a respondent is unaware of what is occurring in the present moment (Brown & Ryan, 2003). The items are scored using a 6-point Likert scale ranging from 1 (almost always) to 6 (almost never), with higher scores indicating a greater level of mindfulness. Reliability and validity is well-established in both college and community populations (Brown & Ryan, 2003; McKillop & Anderson, 2007).

Narcissistic Personality Inventory (NPI)

The Narcissistic Personality Inventory is a widely used brief index of narcissistic personality attributes (Raskin & Terry, 1988). Recent psychometric analyses contribute to growing support for the reliability and validity of this instrument for use in a wide range of clinical and nonclinical samples (Hasanvand, Javanmard, & Goodarzi, 2015).

Personality Inventory for DSM-5 – Depressivity and Anxiousness Domains (PID-5)

The Personality Inventory for DSM-5 (PID-5) is a 220-item questionnaire measuring personality trait facet scales that contribute to five primary personality domains (i.e., antagonism, detachment, psychoticism, disinhibition, negative affectivity; Krueger Derringer, Markon, Watson, & Skodol, 2012). The items are scored using a 4-point Likert format ranging from 0 (Very False or Often False) to 3 (Very True or Often True). Adequate reliability and validity have been established for the measure (Quilty, Ayearst, Chmielewski, Pollock, & Bagby, 2013; Russell, 2016; Anderson et al., 2013; Hopwood, Schade, Krueger, Wright, & Markon, 2012; Wright & Simms, 2014). For the purposes of this study, we used the depressivity and anxiousness facets to assess depression and anxiety symptoms.

Psychiatric Diagnostic History (DIAG)

A history of psychiatric diagnoses will be estimated by asking respondents: *have you been diagnosed with any of the following mental health conditions (leave blank if answer is no or is not applicable)*? Respondents provide estimates for attention-deficit hyperactivity disorder, depression, bipolar disorder, borderline personality disorder, post-traumatic stress disorder, obsessive-compulsive disorder, panic attacks, and schizophrenia.

Psychiatric Treatment History (TXS)

Psychiatric treatment histories were estimated from affirmative responses to a customized question: *Have you been prescribed any of the following types of psychiatric medication (leave bubble blank if answer is no or not applicable)?* Options include antidepressants, mood stabilizing drug (for bipolar disorder), electroconvulsive trial, anti-anxiety medication, stimulant (for ADHD), and/or anti-psychotics. Past treatment with counseling/ psychotherapy, and/or psychiatric hospitalization are also included in the list.

Satisfaction with Life Scale (LSI)

The Satisfaction with Life Scale is a brief, 5-item measure of one's life satisfaction up until the day of testing (Diener, Emmons, Larsen, & Griffin, 1985). The items are scored using a 7-point Likert scale from 1 (strongly disagree) to 7 (strongly agree). Satisfactory reliability and validity have been demonstrated (Adler & Fagley, 2005; Deiner, Emmons, Larsen, & Griffin, 1985; Pavot, Diener, Colvin, & Sandvik, 1991; Pavot & Diener, 1993; Steger, Frazier, Oishi, & Kaler, 2006).

Aggregate Score – Internalized (INT)

An aggregate score was calculated as a mean standard score for the six internalized symptom indicators assessed in the present study (i.e., LSI, DEP, ANX, DIAG, TXS, and FAT).

Aggregate Score – Externalized (EXT)

An aggregate score was calculated as a mean standard score for the six externalized symptom indicators assessed in the present study (i.e., BPAQ, NPI, ANTI, KAT, MIND, and VOL).

CHAPTER III

RESULTS

Descriptive Statistics

Descriptive statistics for the college and national samples are presented in Tables 2 and 3 respectively. Gender differences in the maladjustment indicators were common with women and men tending to score higher respectively on the internalized and externalized maladjustment indicators. Table 4 presents the CAST distributions for maternal, paternal, and parental alcoholism symptoms. The prevalence of CAST-defined (> 5) paternal and maternal alcoholism varied within the college (12.8% & 8.1%) and national (32.5% & 20.2%) samples.

Bivariate Correlation Analyses

Bivariate CAST score associations with the internalized (Table 5) and externalized (Table 6) maladjustment indicators ranged from small (r = .05) to medium (r = .44) in effect size. Associations were pervasive across almost all of the internalized indicators in both samples. Externalized links were largely restricted to trait aggression, mood volatility, and dispositional mindfulness.

Gender differences in correlation strength were found for selected internalized maladjustment indices. Parental drinking was more closely associated with mental health diagnostic history for women than men in the college sample. The relationship between maternal alcoholism and a number of symptom indicators (depression, anxiety, and both diagnostic and treatment history) was stronger for the men in the national sample.

Gender differences in correlation strength were more limited for the externalized maladjustment indicators. Maternal drinking was more strongly associated with alcohol

intake, antisocial attributes, and dispositional mindfulness for the men in the national sample. The link between alcohol intake and paternal drinking was stronger for the men in the college sample. Trait aggression associations with maternal drinking was stronger for the women in the college sample.

Multivariate Analysis of Variance

Independent 2 (gender) x 4 (ACA status) MANOVAs were conducted for the internalized dependent measure cluster within the two samples. MANOVAs were not conducted for the externalized cluster since those maladjustment indicators were not found to be internally consistent. Significant gender, $F(7, 1718) = 6.52$, $p < .000$, Pillai's Trace = .026 ($\eta_p^2 = .026$), and ACA status, $F(21, 5160) = 4.027$, $p < .000$, Pillai's Trace = .013 ($\eta_p^2 = .016$), main effects were found within the college sample. The gender by ACA interaction was not significant, $F(21, 5160) = 1.04$, $p = .399$, Pillai's Trace = .013 ($\eta_p^2 = .004$). Significant gender, $F(6, 637) = 6.79$, $p < .000$, Pillai's Trace = .060 ($\eta_p^2 = .060$), and ACA status, $F(18, 1917) = 7.32$, $p < .000$, Pillai's Trace = .193 ($\eta_p^2 = .064$), main effects were found within the national sample. The gender by ACA interaction was not significant, $F(18, 1917) = 1.27$, $p = .198$, Pillai's Trace = .035 ($\eta_p^2 = .012$).

Analyses of Variance

Subsequent ANOVAs for the internalized measures (Table 7) indicated a significant interaction for ACA status and mental health treatment history in both samples. Female and male ACAs, regardless of status, reported significantly more treatment history compared to non-ACAs in both samples (Tables 8 and 9). Male ACAs with two alcoholic parents in the national sample also reported more treatment history compared to single parent ACAs. There were significant main effects for ACA status on

almost all other internalized maladjustment indicators in both samples (Table 7). Subsequent Scheffe post-hoc comparisons found significant differences between ACA status and a number of internalized symptom indicators (Tables 8 and 9). There were also significant main effects for gender on almost all internalized measures in both samples. Subsequent Scheffe post-hoc comparisons found females reported significantly higher anxiety and body image preoccupation in both samples and significantly higher depression and diagnostic history in the college sample. No other significant gender effects were observed.

The externalized ANOVAs (Table 7) indicated a significant interaction for alcohol intake in the college sample. Male ACAs with paternal alcoholism reported more alcohol intake compared to males with maternal alcoholism or non-ACAs. No significant differences for alcohol intake were observed for females. There were significant main effects for several of the other externalized maladjustment indicators (Table 7). Subsequent Scheffe post-hoc comparisons found significant differences between ACA status and trait aggression, mood volatility, antisocial personality traits and dispositional mindfulness between both samples (Tables 8 and 9). There were also significant main effects for gender on almost all externalized measures in both samples. Males reported significantly higher alcohol intake, antisocial and narcissistic personality traits compared to females in both samples and significantly higher aggression in the national sample. No other significant gender effects were observed.

Relative Risk Analyses

Relative risk analyses were conducted for eight maladjustment risk factors: diagnostic history, treatment history, conduct disturbance, drug use, suicide attempts,

alcoholism, and alcohol-related arrests (Table 11). Non-ACAs were used as the unexposed group in all analyses. In both samples, ACAs were found to be at greater risk for nearly all maladjustment risk factors, regardless of ACA classification (Table 11).

CHAPTER IV

DISCUSSION

There is an inconsistency in the literature related to the relationship between differential gender effects of sex of the alcoholic parent and sex of the ACA related to various internalized and externalized maladjustment risk factors, which highlights an important direction for future research. One of the main goals of the present study was to examine how these differential gender effects may relate to offspring risk of maladjustment. In order to achieve this aim, we improved upon previous literature by employing a 2 (gender) x 4 (neither, maternal, paternal, or both ACA status) design to examine not only how gender of the alcoholic parent and/or ACA relate to maladjustment risk, but also examining the effect of family density of alcoholism on maladjustment risk (i.e., one alcoholic parent vs. two alcoholic parents). Another main goal of the present study was to replicate college sample findings with a national sample collected via Amazon's MTurk.

Overall, consistent with previous literature, our results from both samples indicated ACAs appear to be at increased risk of both internalized and externalized forms of maladjustment compared to non-ACAs. More specific hypotheses were tested to evaluate specific differential gender and ACA status effects: (1) CAST scores will be positively associated with symptom severity; (2) Combined maternal and paternal alcoholism will be associated with the most severe maladjustment compared to ACAs from a single alcoholic parent or non-ACAs; (3) Male ACAs will exhibit relatively more externalized symptoms and female ACAs will exhibit relatively more internalized symptoms; (4) Paternal alcoholism will be more strongly associated with externalized

symptom (particularly for males), and maternal alcoholism will be more strongly associated with internalized symptoms (particularly for females). Relative risk analyses also attempted to estimate some practical implications of ACA status on several maladjustment risk factors.

CAST scores were predicted to be positively associated with symptom severity. This hypothesis was generally supported for the internalized symptoms for both samples. Our results suggest both men and women tended to experience greater internalized symptom severity as CAST scores increased, with a few exceptions. Parental drinking was more closely associated with mental health diagnostic history for women than men in the college sample. The relationship between maternal alcoholism and a number of symptom indicators (depression, anxiety, and both diagnostic and treatment history) was stronger for the men in the national sample. This hypothesis was partially supported for the externalized symptoms. The link between alcohol intake and paternal drinking was stronger for the men in the college sample. Our results indicated maternal drinking was more strongly associated with alcohol intake, antisocial attributes, and dispositional mindfulness for the men in the national sample. Trait aggression associations with maternal drinking was stronger for the women in the college sample.

Our second hypothesis predicted that combined maternal and paternal alcoholism would be associated with more severe maladjustment compared to single parent alcoholism. This hypothesis was partially supported. Both samples found ACAs with two alcoholic parents reported more severe internalized symptoms (depression, anxiety, treatment history, life satisfaction) compared to ACAs with paternal alcoholism alone. The national sample found ACAs with two alcoholic parents reported more antisocial

personality traits compared to single parent ACAs. No other significant differences were observed between dual and single parent alcoholism in either sample. A more prominent and consistent finding in our results is that ACAs, regardless of status (i.e., dual vs. single parent alcoholism), appear to be at increased risk of various internalized and externalized maladjustment compared to non-ACAs.

Our relative risk analyses provide further evidence of this. Both samples demonstrated that ACAs are at increased risk of nearly all maladjustment risk factors compared to non-ACAs. In both samples, parental alcoholism of any type increased an ACAs risk of nearly all maladjustment risk factors compared to non-ACAs with a few exceptions. Paternal and dual alcoholism did not increase an ACAs risk of alcohol-related arrests in the college sample. Paternal and/or dual alcoholism did not increase an ACAs risk of conduct disturbance, diagnostic history, or one suicide attempt in the national sample.

Our third hypothesis predicted male ACAs would exhibit more externalized symptoms and female ACAs would exhibit more internalized symptoms. This hypothesis was partially supported. Several main effects of gender for both samples found females scored significantly higher on nearly all of the internalized maladjustment indicators compared to males and males scored significantly higher on nearly all of the externalized maladjustment indicators. Further, bivariate correlations found parental drinking was more closely associated with psychiatric diagnostic history for women than men in the college sample. Paternal drinking and alcohol intake was stronger for men in the college sample. Maternal drinking was more strongly associated with alcohol intake, antisocial attributes, and dispositional mindfulness for the men in the national sample.

With that said, there are several bivariate correlations that do not support this hypothesis. Trait aggression associations with maternal drinking was stronger for the women in the college sample. The relationship between maternal alcoholism and a number of symptom indicators (depression, anxiety, and both diagnostic and treatment history) was stronger for the men in the national sample. While these results do not support our hypothesis, they are consistent with previous research demonstrating males with alcoholic mothers have reported higher levels of internalizing symptoms (e.g., Chen & Weitzman, 2005; Corte & Becherer, 2007). Overall, consistent with previous literature, we found mixed results regarding how ACA gender is related to their risk of developing various forms of internalized and externalized maladjustment.

Our final hypothesis predicted paternal alcoholism would be more strongly associated with externalized symptoms, particularly for male ACAs, and maternal alcoholism would be more strongly associated with internalized symptoms, particularly for female ACAs. This final hypothesis was adequately not supported. While the significant interaction effect for alcohol intake in the college sample found male ACAs with paternal alcoholism reported more alcohol intake compared to males with maternal alcoholism or non-ACAs, this was the only significant gender x ACA status interaction between both samples supporting this hypothesis. As previously stated, a more prominent and consistent finding is that ACAs, regardless of status appear to be at increased risk of various internalized and externalized compared to non-ACAs. Overall, the limited interaction effects observed in the present study restricted our examination of our last hypothesis. This limitation may be minimized in future studies using a similar 2 x 4 design by ensuring adequate and equal sample sizes across gender and ACA status.

Limitations and Future Directions

While most of our hypotheses were partially supported and our results are generally consistent with previous research, there were a few inconsistencies in findings across the college and national samples. The differences in findings between the college and national sample may be attributable to a number of factors. First, while the overall number of participants in each sample were greater than most ACA studies, analyzing ACA status as four independent classifications resulted in unequal cell sizes between groups. It is important to note that unequal sample sizes can reduce statistical power and inflate Type I error rates, which may explain some of the differences observed (Rusticus & Lovato, 2014). Second, research has demonstrated that ACAs are at greater risk of suffering from multiple ACEs (e.g., physical abuse, emotional abuse) compared to non-ACAs (e.g., Dube et al., 2001; Thompson, Alonzo, Grant, & Hasin, 2013). Other than exposure to parental alcoholism, the present study did not account for other forms of ACEs that may have resulted in greater risk of adult maladjustment. Additionally, other research has demonstrated that ACA maladjustment may be better attributed to a history of parental psychopathology, rather than parental alcoholism alone (McCauley Ohannessian et al., 2004; Preuss et al., 2002). Results of the present study can be further clarified by measuring parental alcoholism, various forms of ACEs, and parental psychopathology. Future research in this area should focus on the contributions and strengths of all aforementioned factors in maladjustment. Third, while an internalized maladjustment factor was successfully extracted ($\alpha = .70$; 21.04%), the extracted externalized maladjustment factor was relatively weak ($\alpha = .18$; 14.69%), suggesting the measures used to evaluate externalized indicators of maladjustment may not have been

sufficient to find notable differences between groups. Finally, the college and national samples lacked equivalence in several ways. The college sample consisted of undergraduate students from one Midwestern university, which may limit the generalizability of the findings to a subset of college undergraduates. In comparison, the national sample was more heterogeneous in terms of age, gender, and ethnic diversity and may be more representative of and generalizable to the general population. Finally, different depression and anxiety scales were used for the national sample in hope of using more psychometrically robust measures than the measures used in the college sample.

Conclusions

To the author's knowledge, the present study was the first to employ a 2 (gender) x 4 (ACA status) design to examine ACAs risk of various maladjustment indicators while taking into account both differential gender effects of both the parent and ACA and various levels of ACA status (i.e., neither, mom, dad, or both alcoholics). While our gender x ACA status interactions were limited, it is evident from our results that treating ACA status as a dichotomous factor may inadvertently restrict our understanding of how differences in parental alcoholism (i.e., maternal vs. paternal or dual vs. single parent) may influence an ACAs risk of various internalized and externalized forms of maladjustment, which highlights a salient direction for future research. In the present study, some nuanced differences were observed between the samples, which may be explained by confounding sources not examined in the present study as previously described; however, our results were generally consistent with previous literature. Future research should take into account not only differential gender effects of both ACA and alcoholic parent, but also other potential sources of maladjustment risk factors to help

draw more definitive conclusions about the relationship between parental alcoholism and its effect on adult children of alcoholics.

APPENDICES

Appendix A

Table 1. Factor Loading Contrasts in a Two-Factor Internalized-Externalized Solution

Maladjustment Indicators	LABEL	Factor1	Factor 2
		Internalized	Externalized
Depression Symptoms	DEPR	**.762**	.100
Anxiety Symptoms	ANX	**.727**	.174
Diagnostic History	DIAG	**.445**	-.008
Treatment History	TXS	**.590**	-.012
Fear of Negative Evaluation	FNE	**.551**	-.044
Fear of Fat Scale	FAT	**.444**	.154
Life Satisfaction	LSI	**.533**	.203
Alcohol Consumption	KAT	-.106	**.426**
Trait Aggression	BPAQ	.236	**.413**
Mood Volatility	VOL	.293	**.555**
Narcissistic Traits	NARC	-.358	**.587**
Antisocial Traits	ANTI	.074	**.740**
Dispositional Mindfulness	MIND	-.388	**-.574**
Conduct Disturbance	COND	.236	.261

Note. Varimax rotation forcing a two-factor solution; Eigenvalue > 1; factor loadings > .4; pairwise exclusions for missing data.

Appendix B

Table 2. Descriptive Statistics for Predictor and Criterion Variables in the College Sample

Predictor & Criterion Variables	Label	Women	Men	*M*	*SD*	Range	Gender	
		n	*n*				*p*	*d*
Maternal Alcoholism	CAST	1920	606	1.33	4.28	0-28	**	.137
Paternal Alcoholism	CAST	1920	606	2.07	5.08	0-28	***	.197
Parental Alcoholism	CASTT	1920	606	1.70	3.72	0-28	***	.212
Internalized Maladjustment Indicators								
Depression Symptoms	DEP	1785	550	7.48	8.77	0-48	***	.235
Anxiety Symptoms	ANX	1712	514	7.19	9.68	0-52	***	.386
Diagnostic History	DIAG	2041	643	.294	.735	0-8	***	.175
Treatment History	TXS	2041	643	.560	1.19	0-8	***	.131
Fear of Negative	FNE	1764	545	15.78	7.71	0-29	***	.344
Fear of Fat Scale	FAT	1833	568	8.30	7.08	0-30	***	.528
Life Satisfaction	LSI	1857	578	19.96	6.44	0-30	***	.167
Externalized Maladjustment Indicators								
Alcohol Consumption	KAT	1962	619	.655	1.05	0-11.7	***	.408
Trait Aggression	BPAQ	1677	507	57.97	19.06	29-137	***	.226
Mood Volatility	HYPO	1821	563	6.28	3.70	0-15		
Narcissistic Traits	NARC	1805	563	44.60	10.21	16-80	***	.406
Antisocial Traits	ANTI	1742	537	63.95	12.35	25-125	***	.408
Dispositional Mindfulness	MIND	1796	552	39.95	14.79	15-90		

Note. * $p < .05$. ** $p < .01$. *** $p < .001$.

Appendix C

Table 3. Descriptive Statistics for Predictor and Criterion Variables in the National Sample

Predictors & Criterion	Label	Women	Men	*M*	*SD*	Range	Gender	
		n	*n*				*p*	*d*
Maternal Alcoholism	CASTM	411	292	3.53	6.84	0-28		
Paternal Alcoholism	CASTD	411	292	5.27	7.53	0-28		
Parental Alcoholism	CASTT	411	292	4.40	5.66	0-28		
Internalized Maladjustment Indicators								
Depression Symptoms	DEP	411	292	22.90	10.77	2-56		
Anxiety Symptoms	ANX	411	290	20.28	6.89	9-36	**	.243
Diagnostic History	DIAG	411	292	0.75	1.45	0-8	**	.232
Treatment History	TXS	401	285	1.53	2.17	0-8		
Fear of Fat Scale	FAT	395	275	9.63	7.23	0-30	***	.379
Life Satisfaction	LSI	407	289	17.45	8.07	0-30	*	.157
Externalized Maladjustment Indicators								
Alcohol Consumption	KAT	408	290	.85	1.70	29	***	.407
Trait Aggression	BPAQ	361	248	70.63	22.10	29-131	***	.340
Mood Volatility	HYPO	411	292	5.63	3.96	0-15		
Narcissistic Traits	NARC	386	277	42.12	12.78	16-75	**	.256
Antisocial Traits	ANTI	364	261	63.73	13.87	25-125	**	.253
Dispositional Mindfulness	MIND	396	274	36.29	16.75	15-84		

Note. * $p < .05$. ** $p < .01$. *** $p < .001$.

Appendix D

Table 4. Paternal, Maternal, and Parental CAST Alcoholism Distributions

Score	College Sample (N = 2531)			National Sample (N = 703)		
	CASTD	CASTM	CASTT	CASTD	CASTM	CASTT
0	73.8%	83.3%	67.6%	52.6%	65.6%	41.1%
1	5.5%	3.9%	2.7%	4.0%	5.5%	2.3%
2	3.0%	2.0%	2.3%	2.6%	3.3%	1.8%
3	2.0%	1.2%	1.3%	4.1%	3.0%	1.8%
4	1.8%	0.9%	1.0%	1.8%	1.1%	2.3%
5	1.1%	0.6%	1.3%	2.4%	1.3%	1.4%
6	1.2%	0.6%	0.7%	1.6%	0.7%	1.7%
7	0.9%	0.8%	0.9%	1.7%	0.7%	3.0%
8	0.9%	0.5%	0.6%	1.6%	1.4%	1.1%
9	0.9%	0.5%	0.6%	1.6%	0.7%	2.0%
10	1.1%	0.5%	0.5%	1.8%	1.1%	1.6%
11	0.8%	0.4%	0.3%	1.3%	0.7%	1.6%
12	0.6%	0.4%	0.4%	1.7%	1.3%	1.1%
13	0.7%	0.5%	0.4%	1.8%	1.0%	0.9%
14	0.6%	0.3%	0.6%	2.8%	0.6%	1.6%
15	0.5%	0.4%	0.0%	2.3%	1.3%	0.9%

Note. CASTD = Paternal CAST; CASTM = Maternal CAST; CASTT = Total CAST.

Appendix E

Table 5. Bivariate Correlation Matrix of CAST Scores and Internalized Maladjustment Indicators

Maladjustment Indicators	College Sample (n = 2531)			National Sample (n = 703)		
	Paternal	Maternal	Parental	Paternal	Maternal	Parental
	CASTD	CASTM	CASTT	CASTD	CASTM	CASTT
Women						
DEP	.159**	.156**	.198**	.248**	.186**	.291**
ANX	.144**	.165**	.194**	.160**	.089	.169**
DIAG	.054*	.098**	.094**	.277**	.149**	.254**
TXS	.124**	.151**	.172**	.209**	.178**	.247**
FAT	.107**	.081**	.120**	.073	.071	.097
LSI	.122**	.148**	.169**	-.136**	-.056	-.131**
Men						
DEP	.101*	.177**	.167**	.272**	.330**	.354**
ANX	.079	.141**	.132**	.248**	.292**	.318**
DIAG	-.026	-.003	-.020	.267**	.303**	.336**
TXS	.132**	.168**	.183**	.277**	.351**	.447**
FAT	.098*	.070	.106*	.153*	.185**	.201**
LSI	.096*	.115**	.130**	-.051	-.103	-.090

Note. CASTD = Paternal Alcoholism CAST; CASTM = Maternal Alcoholism CAST; CASTT = Total Parental Alcoholism. DEP = depression symptoms; ANX = anxiety symptoms; DIAG = mental health diagnostic history; TXS = mental health treatment history; FAT = Fear of Fat Scale; LSI = Life Satisfaction Index; KAT = Khavari Alcohol Test; BPAQ = trait aggression; HYPO = Mood Volatility; NARC = narcissistic traits; ANTI = antisocial traits; MIND = dispositional mindfulness. *$p < 05$. **$p < .01$. ***$p < .001$. Shading designates significant gender difference in coefficient

Appendix F

Table 6. Bivariate Correlation Matrix of CAST Scores and Externalized Maladjustment Indicators

Maladjustment Indicators	College Sample (N = 2531)			National Sample (N = 703)		
	Paternal	Maternal	Parental	Paternal	Maternal	Parental
	CASTD	CASTM	CASTT	CASTD	CASTM	CASTT
Women						
KAT	.020	.021	.025	.022	-.039	-.009
BPAQ	.104**	.123**	.143**	.127*	.134*	.177**
VOL	.100*	.094**	.122**	.206**	.120*	.221**
NARC	.015	.021	.022	-.064	-.033	-.066
ANTI	.035	.054*	.056*	.059	.007	.037
MIND	-.083**	-.088**	-.107**	.189**	.153**	.229**
Men						
KAT	.112**	.035	.096*	.099	.121*	.130*
BPAQ	.045	-.001	.032	-.002	.063	.034
VOL	.070	.116**	.112**	.197**	.195**	.232**
NARC	.025	.065	.053	.040	.074	.067
ANTI	-.028	.063	.016	.129*	.235**	.213**
MIND	-.016	-.061	-.046	.176**	.296**	.276**

Note. CASTD = Paternal Alcoholism CAST; CASTM = Maternal Alcoholism CAST; CASTT = Total Parental Alcoholism. DEP = depression symptoms; ANX = anxiety symptoms; DIAG = mental health diagnostic history; TXS = mental health treatment history; FAT = Fear of Fat Scale; LSI = Life Satisfaction Index; KAT = Khavari Alcohol Test; BPAQ = trait aggression; HYPO = Mood Volatility; NARC = narcissistic traits; ANTI = antisocial traits; MIND = dispositional mindfulness. *p < 05. **p < .01. ***p < .001. Shading designates significant gender difference in coefficient strength.

Appendix G

Table 7. Parental Alcoholism and Gender Interaction Analyses of Variance

	College Sample (N = 2531)						National Sample (N = 703)					
	ACA			Gender x ACA			ACA			Gender x ACA		
	F	p	η_p^2	F	p	η_p^2	F	p	η_p^2	F	p	η_p^2
	Internalized Maladjustment Indicators											
DEPz	15.63	**<.001**	.02	.656	.579	.00	22.53	**<.001**	.08	.401	.752	.00
ANXz	11.29	**<.001**	.01	1.05	.366	.00	11.59	**<.001**	.04	1.44	.228	.00
DIAGz	1.417	.236	.00	1.50	.210	.00	14.66	**<.001**	.06	.419	.740	.00
TXSz	22.84	**<.001**	.02	6.17	**<.001**	.00	32.89	**<.001**	.12	3.34	**< .05**	.01
FNEz	1.82	.140	.00	.587	.623	.00						
FATz	4.36	**< .01**	.00	.146	.932	.00	6.44	**<.001**	.02	.923	.429	.00
LSIz	11.43	**<.001**	.01	.570	.635	.00	4.73	**< .01**	.02	1.19	.309	.00
INTz	17.73	**<.001**	.03	.111	.954	.00	36.93	**<.001**	.13	2.05	.104	.00
	Externalized Maladjustment Indicators											
KATz	6.49	**<.001**	.00	5.61	**<.01**	.00	.928	.427	.00	1.96	.118	.00
BPAQz	4.16	**<.01**	.00	1.81	.142	.00	1.55	.198	.00	.501	.682	.00
VOLz	7.47	**<.001**	.01	.196	.899	.00	8.19	**<.001**	.03	.056	.983	.00
NARCz	1.10	.345	.00	.790	.499	.00	1.45	.225	.00	.327	.806	.00
ANTIz	1.94	.120	.00	.299	.826	.00	7.85	**<.001**	.03	2.55	.055	.01
MINDz	2.65	**<.05**	.00	.917	.432	.00	11.83	**<.001**	.05	2.00	.113	.00
EXTz	9.21	**<.001**	.01	.461	.709	.00	10.45	**<.001**	.04	1.28	.279	.00

Note. Significant effects bolded. Standard z scores were used in the analyses of these maladjustment indicators. DEP = depression symptoms; ANX = anxiety symptoms; DIAG = mental health diagnostic history; TXS = mental health treatment history; FNE = Fear of Negative Evaluation; FAT = Fear of Fat Scale; LSI = Life Satisfaction Index; KAT = Khavari Alcohol Test; BPAQ = trait aggression; HYPO = Mood Volatility; NARC = narcissistic traits; ANTI = antisocial traits; MIND = dispositional mindfulness.

Appendix H

Table 8. Parental Alcoholism Maladjustment Group Differences in College Sample

Criterion Indices	Dual Alcoholism CASTD > 5 CASTM > 5			Paternal Alcoholism CASTD > 5 CASTM < 6			Maternal Alcoholism CASTD < 6 CASTM > 5			No Alcoholism CASTD < 6 CASTM < 6		
	M	*SE*	*n*	*M*	*SE*	*n*	*M*	*SE*	*n*	*M*	*SE*	*n*
	Internalized Maladjustment Indicators											
DEPz	.620[d]	.142	73	.145[c]	.096	213	.338[b]	.115	108	-.132[a]	.026	1865
ANXz	.489[d]	.146	68	.606[c]	.095	210	.262[b]	.115	107	-.159[a]	.027	1771
DIAGz	.171	.139	81	-.084	.091	244	.078	.111	126	-.063	.025	2075
TXSz	.907[d]	.137	81	.071[c]	.090	244	.345[b]	.110	126	-.105[a]	.025	2075
FNEz	-.130	.147	71	.052	.094	217	.083	.114	112	-.115	.027	1834
FATz	.269[c]	.144	76	.026[b]	.093	224	-.028	.112	115	-.165[a]	.026	1908
LSIz	-.522[c]	.139	77	-.195	.093	232	-.484[b]	.113	114	.012[a]	.026	1930
INTz	.419[d]	.097	56	.073[c]	.063	160	.241[b]	.075	80	-.111[a]	.018	1436
	Externalized Maladjustment Indicators											
KATz	.195	.140	78	.501	.089	236	.122	.111	120	.093	.025	1993
BPAQz	.308[c]	.149	67	.248	.095	208	.309[b]	.126	102	.022[a]	.028	1716
VOLz	.433[c]	.140	78	.140	.096	224	.330[b]	.117	112	-.044[a]	.026	1891
NARCz	.304	.143	72	.076	.094	223	.230	.115	111	.096	.026	1883
ANTIz	.315	.140	72	.047	.096	214	.301	.113	111	.093	.027	1806
MINDz	.266[b]	.143	75	.092	.096	218	.174	.114	115	-.030[a]	.027	1862
EXTz	.284[c]	.072	81	.174	.047	243	.219[b]	.057	126	.034[a]	.013	2071

Note. Scheffe post-hoc cell differences ($p < .05$) designated by superscript letters that differ. Standard *z* scores were used in the analyses of these maladjustment indicators. DEP = depression symptoms; ANX = anxiety symptoms; DIAG = mental health diagnostic history; TXS = mental health treatment history; FNE = Fear of Negative Evaluation; FAT = Fear of Fat Scale; LSI = Life Satisfaction Index; KAT = Khavari Alcohol Test; BPAQ = trait aggression; HYPO = Mood Volatility; NARC = narcissistic traits; ANTI = antisocial traits; MIND = dispositional mindfulness.

Appendix I

Table 9. Parental Alcoholism Maladjustment Group Differences in National Sample

Criterion Indices	Dual Alcoholism			Paternal Alcoholism			Maternal Alcoholism			No Alcoholism		
	CASTD > 5 CASTM > 5			CASTD > 5 CASTM < 6			CASTD < 6 CASTM > 5			CASTD < 6 CASTM < 6		
	M	*SE*	*n*	*M*	*SE*	*n*	*M*	*SE*	*n*	*M*	*SE*	*n*
	Internalized Maladjustment Indicators											
DEPz	.621[d]	.110	76	.155[c]	.085	153	.374[b]	.124	66	-.228[a]	.048	408
ANXz	.380[d]	.111	76	.098[c]	.086	153	.273[b]	.125	66	-.204[a]	.048	406
DIAGz	.445[d]	.111	76	.191[c]	.086	153	.184[b]	.125	66	-.222[a]	.048	408
TXSz	.844[d]	.108	74	.144[c]	.083	150	.270[b]	.125	63	-.259[a]	.047	399
FATz	.395[b]	.115	72	-.056	.087	150	.093	.128	64	-.137[a]	.050	385
LSIz	-.022	.114	75	-.157[c]	.088	152	-.327[b]	.127	66	.104[a]	.049	403
INTz	.437[d]	.055	76	.062[c]	.043	153	.142[b]	.062	66	-.155[a]	.024	408
	Externalized Maladjustment Indicators											
KATz	.125	.113	75	.135	.087	153	.003	.127	65	-.007	.049	405
BPAQz	.259	.120	68	-.036	.093	137	.079	.135	61	-.004	.053	355
VOLz	.396[c]	.113	76	.167[b]	.087	153	.125	.127	66	-.136[a]	.049	408
NARCz	.192	.117	72	-.055	.090	146	-.134	.134	61	.043	.051	387
ANTIz	.516[b]	.116	71	-.166	.092	137	-.077	.133	58	-.023[a]	.052	359
MINDz	.464[d]	.114	73	.062[c]	.087	149	.258[b]	.132	61	-.192[a]	.049	388
EXTz	.308[b]	.059	76	.022	.046	153	.041	.067	66	-.052[a]	.026	408

Note. Scheffe post-hoc cell differences ($p < .05$) designated by superscript letters that differ. Standard *z* scores were used in the analyses of these maladjustment indicators. DEP = depression symptoms; ANX = anxiety symptoms; DIAG = mental health diagnostic history; TXS = mental health treatment history; FAT = Fear of Fat Scale; LSI = Life Satisfaction Index; KAT = Khavari Alcohol Test; BPAQ = trait aggression; HYPO = Mood Volatility; NARC = narcissistic traits; ANTI = antisocial traits; MIND = dispositional mindfulness.

Appendix J

Table 10. Relative Risks Posed by Parental Alcoholism Classifications

Maladjustment Risk	Paternal Alcoholism	Maternal Alcoholism	Dual Alcoholism
	College Sample		
Diagnostic History (> 0)	1.25*	1.46**	1.61**
Treatment History (> 0)	1.50***	1.73***	1.40*
Conduct Problems (> 2)	1.81***	2.29***	1.96**
Drug Use (> 0)	2.37***	3.23***	4.49***
Alcohol Abuse (MAST > 6)	1.64***	2.05***	2.23***
Alcohol-Related Arrest	1.37	2.68***	1.60
Suicide Attempt (1)	1.66**	2.75***	3.43***
Suicide Attempt (> 1)	2.34***	3.52***	3.26***
	National Sample		
Diagnostic History (> 0)	1.75**	1.54**	1.27
Treatment History (> 0)	1.59**	1.57***	1.54***
Conduct Problems (> 2)	1.36	4.64***	1.89***
Drug Use (> 0)	1.22**	1.30**	1.50***
Alcohol Abuse (MAST > 6)	1.63**	1.26*	1.87***
Alcohol-Related Arrest	2.03***	2.52***	3.45***
Suicide Attempt (1)	1.35	1.82**	1.19
Suicide Attempts (> 1)	2.84***	4.18***	4.50***

Note. * $p < .05$. **$p < .01$. *** $p < .001$. No Alcoholism Cell (CASTD =0; CASTM = 0) used as control condition in each *RR* estimation. Conduct disturbance symptoms all occurred prior to age 15.

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