



Externalizing Pathology and Substance Use Among Adolescents: The Intersection of Functional Brain Activity

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Abstract

Given the unique harms of substance use in early adolescence, it is important to understand factors that predispose adolescents to such behaviors. Externalizing pathology is known to increase substance use risk, yet research on brain-based factors underlying both externalizing and substance use behaviors is limited. This investigation evaluated the role of brain function in externalizing pathology and substance use among adolescents. Participants included 54 adolescents (ages 12-14) and their parents/guardians. In this home-based study, adolescents completed a resting-state electroencephalography focused on alpha power and clinical measures assessing substance use and externalizing pathology. Parents/guardians completed clinical measures of their adolescents' behavior. Externalizing pathology was most consistently associated with substance use on the behavioral level, yet less frequently on the diagnostic level. Frontal and central alpha power were also most consistently related to behavior-level externalizing pathology, yet less often on the diagnostic level. While frontal alpha power was associated with substance use, central alpha power was associated with alcohol craving. Our findings indicate that alpha power may relate to externalizing pathology, as well as substance use behaviors and urges. The results highlight the importance of behavior-level assessment of externalizing pathology among adolescents, in addition to providing direction for theoretical and clinical work.

Keywords: Adolescent; Substance Use; Electroencephalography; Functional Neuroimaging; Attention Deficit Hyperactivity Disorder; Disruptive; Impulse Control; Conduct Disorders

Abbreviations

ADHD: Attention Deficit-Hyperactivity Disorder; ODD: Oppositional Defiant Disorder; CD: Conduct Disorder; CNS: Central Nervous System; EEG: Electroencephalography; KSADS-COMP: Kiddie Schedule for Affective Disorders and Schizophrenia - Computerized Version; DSM-5: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition; CBCL: Child Behavior Checklist; AADIS: Adolescent Alcohol and Drug Involvement Scale; ACQ-

SF-R: Alcohol Craving Questionnaire - Short Form - Revised; IV: Independent Variable; DV: Dependent Variable; DBD: Disruptive Behavior Disorder

Introduction

The purpose of the present work was to evaluate connections between brain function, externalizing pathology, and substance use among adolescents. The literature indicates that externalizing pathology (operationalized as externalizing diagnoses and/

or behaviors) and substance use behaviors often co-occur [1-9], yet there is limited research on brain-based factors that may underlie both facets of behavior among adolescents. Thus, the current investigation aimed to fill this gap in the literature by assessing substance use, alcohol craving, and behavior-level and diagnostically significant externalizing pathology in an at-risk population of adolescents (ages 12-14). The goal of the study was to understand if brain function characteristics are related to substance use and externalizing pathology. Such knowledge serves to inform theory around the underlying causes of these risky behaviors, in addition to informing treatment and prevention efforts for adolescents who may be struggling with substance use and externalizing pathology.

Adolescence is a critical developmental period in which young people are growing rapidly. This stage of development is characterized by not only physical but also social and emotional changes that lay the foundation for adulthood [10-12]. Substance use rates are noteworthy among adolescents, with an estimated 2 million adolescents between the ages of 12 and 17 (comprising 7% of individuals in this age range) consuming alcohol within a one-month period [13]. Additionally, adolescents exhibit a faster transition from substance use to substance use disorder than is seen among adults [14]. Given the prevalence rates and unique harms associated with substance use during adolescence, it is important to understand factors that may predispose adolescents to substance use urges and behaviors.

Craving, defined as “a strong desire or urge” to use a substance or engage in an-other addictive behavior [15] (p. 491), is also a key component of substance use [16,17]. Craving is important to attend to as it is a strong predictor of future substance use among adolescents [18,19]. Furthermore, craving is especially of interest in the present investigation as it serves to gauge desire or urges to engage in substance use, even if the adolescent does not follow through with their desire or urge. Thus, it is useful to assess craving within early adolescent samples as it may indicate intent to engage in substance use before it begins. By assessing substance use from urges to behavior, the field may better understand risk factors for substance use among adolescents.

A group of disorders - termed externalizing disorders - have been identified in the literature as conferring increased risk for substance use among adolescents [6,9,20,21]. Externalizing

disorders are characterized by under-controlled behavior, such as impulsivity, rule-breaking, hyperactivity, and behavioral disinhibition [15]. The most widely prevalent and commonly discussed externalizing disorders among adolescents include attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD; [15]). Although the connections in the literature vary in strength based on the type of disorder, evidence suggests that externalizing pathology may increase risk of substance use among adolescents [6,9,20,21].

Given the comorbidity between substance use and externalizing pathology, it is not surprising that theories behind these experiences converge. A primary theory of externalizing pathology (particularly for ADHD and antisocial behavior), which proposes a biological basis of such pathology, is entitled the hypoarousal model [22-25]. This model maintains that individuals with externalizing pathology are in a chronic state of central nervous system (CNS) hypoarousal, which contributes to engaging in externalizing behavior as a form of self-stimulation to counter this chronic state of low arousal. Similar hypoarousal mechanisms have also been suspected to underlie substance use, with some researchers proposing that CNS hypoarousal may be associated with increased risk of substance use [26-29]. Due to the hypothesized CNS mechanisms underlying both substance use and externalizing pathology, brain-based factors have been studied in relation to these experiences.

Traditionally, externalizing behavior has been associated with decreased fast-wave (including beta and gamma waves) and increased slow-wave (including delta and theta) patterns in electrical brain activity [15]. For example, comparison of theta and beta waves (entitled the theta/beta ratio) has been studied extensively in relation to ADHD as it was proposed to reflect levels of arousal and attention [30]. In comparison to such slow-to-fast-wave ratios, alpha power has been studied less frequently in connection to externalizing pathology, although research indicates its potential utility. As such, alpha power may represent a relatively newer biomarker that could both complement and extend existing hypoarousal theories.

Alpha power, a brain-based factor assessed via electroencephalography (EEG), has been linked to CNS hypoarousal. Alpha power reflects the activity of alpha waves (i.e., moderate brain waves between 8-13 Hz) in the brain [31], displaying an inverse relationship with CNS arousal in some studies [32-35].

More specifically, research suggests that high arousal is associated with low alpha power, and low arousal is associated with high alpha power [32-35]. Thus, if the hypoarousal models are accurate, one would expect to see higher alpha power (and, subsequently, lower arousal) among individuals with externalizing pathology and/or substance use. However, the research on these connections remains limited, particularly with respect to adolescent samples.

In the few adolescent studies available, the findings regarding alpha power and externalizing pathology are unclear. Some studies have found increases in alpha power among adolescents with externalizing pathology (including ADHD, ODD, and CD pathology; [36-39]), other studies have found decreases in alpha power [40,41], and yet other studies have found no significant difference between adolescents with and without externalizing pathology [42,43]. Some research has highlighted methodological and sampling problems that may contribute to the divergent result seen [44]. For example, many studies consist of predominantly or all male samples of children ages 12 and under [44]. Additionally, there is variability in the conditions in which re-searchers choose to collect EEG data - ranging from eyes-open (EO) to eyes-closed (EC), to an average of the two or not reporting recording conditions at all - which can introduce unsystematic variance into the association aiming to be studied [44]. Thus, while there is theoretical reasoning to believe alpha power and externalizing pathology are connected and related to hypoarousal, empirical findings among adolescents are mixed and relatively sparse.

What also appears understudied in the literature is the relationship between alpha power and substance use, particularly among adolescents. In adult samples, the literature indicates a pattern in which individuals actively using substances, namely alcohol and marijuana (commonly used substances among adolescents; [45]), display decreased alpha power in comparison to individuals who do not use substances [46]. Similar results have been found for individuals using opioids [47]. Notably, during periods of abstinence from alcohol, alpha power tends to increase [46]. This suggests a potential pattern in which those who use substances exhibit higher baseline alpha power, that then decreases with substance use and increases with abstinence. However, as with much of the literature on EEG components, the samples consist largely of adults.

Given the potential link between alpha power and hypoarousal [32-35], and externalizing pathology and substance use [6,9,20,21], it is possible that alpha power may be related to externalizing pathology and substance use, serving as a brain-based factor that confers risk for these experiences. To fill this gap in the literature, the current study examined the relationships among alpha power, externalizing pathology, and substance use in a sample of at-risk adolescents. Externalizing pathology was assessed on both the diagnostic and behavioral levels, as including behavior-level assessment that is more continuous can reveal critical information that might have otherwise been missed through diagnostic assessment alone [48]. Substance use was assessed in terms of behavior (via self-reported use) and urges (via self-reported craving).

Aims

In keeping with the literature [6,9,20,21], it was hypothesized that externalizing pathology (including ADHD, ODD, CD diagnoses and behaviors) would be associated with increased substance use risk (assessed via use and craving self-reports) among adolescents. Drawing from hypoarousal models of externalizing pathology [32-35], the present study predicted that adolescents displaying greater alpha power (and, thus, lower arousal) would display greater externalizing pathology, similarly gauged via behavior-level and diagnostic assessments. Regarding substance use, adult research indicates a potential pattern in which those who use substances exhibit higher baseline alpha power, that then decreases with substance use and increases with abstinence [46,47]. As lower alpha power serves as an indicator of current substance use among adults, it was hypothesized that lower alpha power would be associated with the presence of substance use behavior among adolescents. As higher baseline alpha power may serve as a risk factor for substance use [46], it was hypothesized that higher alpha power would be associated with more urges to engage in substance use (i.e., greater craving) among adolescents.

Materials and Methods

Consent and funding

The procedures of this study were approved by the university's Institutional Review Board (IRB #17-0033). Written consent to participate in the study was obtained from the parent/guardian

of each adolescent, and written assent was obtained from each of the adolescents themselves. Funding was provided by the U.S. National Institute of Health - National Institute of Alcohol Abuse and Alcoholism (NIH-NIAAA).

Participants

Participants included adolescents between the ages of 12 and 14 years of age (total of participants (N) = 54; mean (M) age = 12.96; standard deviation (SD) of age = 0.85; 25 female, 29 male). For detailed demographic information of the adolescents (reported by parents/guardians), see Table 1. Participants were recruited through the local Department of Health and Human Services. The

participants in the present study were part of a broader NIH-NIAAA funded study and recruitment was established for the needs of that broader study. Exclusion criteria, assessed via a screening phone call, included the following: contraindications to EEG (e.g., seizures, epilepsy, other neurological problems), significant hearing impairment, significant visual impairment that is not corrected by eyeglasses or contacts, diagnosed intellectual disability that may prohibit completion of measures/tasks, or history of significant head injury (e.g., loss of consciousness over 15 minutes).

Characteristics	n	%
Sex		
Male	25	46.30%
Female	29	53.70%
Race and Ethnicity		
African American/Black	33	61.11%
Native American	0	--
Asian	0	--
Native Hawaiian/Pacific Islander	0	--
White Hispanic	9	16.67%
White Non-Hispanic	10	18.52%
Multiracial	2	3.70%
Annual Income		
< \$10,000	16	29.63%
\$10,000 - \$14,999	9	16.67%
\$15,000 - \$24,999	7	12.96%
\$25,000 - \$34,999	9	16.67%
\$35,000 - \$49,000	7	12.96%
\$50,000 - \$74,000	4	7.41%
\$75,000 - \$99,999	1	1.9%
< \$100,000	1	1.9%

Table 1: Demographic information of participants (N = 54).

n = number of participants included in the associated subgroup. % = percent of overall sample included in the associated subgroup.

Procedures

All procedures (including EEG and clinical measures) were conducted in the participants’ homes. The semi-structured

diagnostic interview was completed via the measure’s online portal. The remaining clinical measures were completed on Collaborative Informatics and Neuroimaging Suite [49], an encrypted, firewall-

protected online neuroimaging database. The parent/guardian and adolescent were asked to complete the measures in quiet, separate rooms to minimize distractions and increase privacy. Following completion of the measures, adolescents participated in an EEG assessment. Compensation was provided to both the parent/guardian (\$20) and the adolescent (\$40).

In keeping with the Health Insurance Portability and Accountability Act, all data were kept confidential outside of situations requiring mandated reporting. If child abuse or neglect was endorsed by a parent or adolescent, the principal investigator followed all mandated reporting laws. Names or other common identifiers (e.g., date-of-birth) were not stored with the data.

Measures

Kiddie Schedule for Affective Disorders and Schizophrenia - Computerized Version (KSADS-COMP; [50])

The KSADS-COMP is a semi-structured diagnostic interview for children and adolescents that assesses a wide variety of mental health diagnoses [50,51]. The computerized interview directly maps onto diagnostic criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), assessing parent and adolescent for the adolescent's current and historical psychopathology. Available data indicate that the KSADS-COMP displays good convergent validity when compared to established measures (e.g., Patient Health Questionnaire - 9; [52]), indicated by significant Wilcoxon signed rank tests ($p < .01$), and good to excellent concordance (Gwet's AC1 = .76-.94; [50]). In the current study, the KSADS-COMP [50] served as a diagnostic measure of externalizing pathology.

Child behavior checklist (CBCL; [53])

The CBCL is one of the most widely used parent-report measures of internalizing and externalizing behavior among children and adolescents [53-55]. The 113-item measure was completed by parents/guardians of the adolescents, with respondents rating their adolescent's emotional and behavioral problems on a three-point Likert-type scale (0 = "Not True," 1 = "Somewhat or Sometimes True," 2 = "Very True or Often True"). The CBCL demonstrates proficient psychometric, including excellent test re-test reliability (ICC = .95) and acceptable-to-excellent internal consistency ($\alpha = .78-.97$; [50]). The current study focused on externalizing behavior including the Externalizing Problems, ADHD, Oppositional Defiant Problems, and Conduct Problems subscales of the CBCL.

In the current study, the CBCL [53] served as a behavior-level measure of externalizing pathology without a specific cut-off point. Unlike the KSADS-COMP [50], the CBCL [53] is not a diagnostic measure. Although the CBCL reflects many of the criteria indicated in the DSM-5 [15], the measure assesses externalizing behavior on a continuum and does not have the requirement of endorsed impairment in life domains. As such, the CBCL may be more sensitive to detecting associations that binary diagnostic assessments might miss.

Alcohol and drug involvement scale (AADIS; [56])

The AADIS is a well-established self-report measure of substance use among adolescents [56]. The scale is face-valid, consisting of 14 items that screen for substance use behaviors (e.g., frequency, amount). Scores on the AADIS can range from 0-80, with higher scores indicating a higher level of alcohol and/or drug involvement. Since its creation and initial validation, the AADIS has been used in community-based samples, as was the case in the current investigation [57-59]. With respect to the psychometrics of the scale, the AADIS exhibits excellent internal consistency ($\alpha = .94$; [56]). Scores on the AADIS correlate with clinical assessment ($r = .75$), adolescents' reported level of substance use ($r = .72$), and adolescents' perceived use severity ($r = 0.79$; [56,60]).

Alcohol craving questionnaire - Short form - revised (ACQ-SF-R; [61])

The ACQ-SF-R is a 12-item self-report measure designed to assess alcohol craving [61]. The face-valid measure gauges different facets of alcohol craving (including compulsivity, expectancy, purposefulness, and emotionality). Responses result in a total raw score ranging from 12 to 84, with higher scores indicating greater craving for alcohol. Of note, the ACQ-SF-F was developed for adults. While the measure has been used with late teens and adolescents (e.g., [62]), it has not been used in available literature with early adolescents such as the current sample. This highlights an area for caution when generalizing the current findings.

With respect to the psychometrics of the scale, the ACQ-SF-R exhibits acceptable to good internal consistency (α of factors = .77-.86; [61]). Scores on the ACQ-SF-R strongly correlate with other well-known measures assessing alcohol craving (e.g., Penn Alcohol Craving Scale, $r = .65$; [63,64]) and alcohol use (Alcohol Use Disorders Identification Test, $r = .65$; [65-67]). Note, the ACQ-SF-R is not intended as a diagnostic tool, although it may detect

symptoms (e.g., craving) of alcohol use disorder [61]. The ACQ-SF-R was used to supplement the substance use behavior data (AADIS; [56]) collected, serving to assess desire or intent to engage in substance use behaviors even if the adolescent does not follow through with their craving.

EEG data collection and analysis

To collect EEG data at resting-state, participants were asked to sit still and quietly to complete a 3-minute eyes-open period and a 3-minute eyes-closed period of recorded data. The study team ensured that EEG was conducted in a quiet, calm space (with only the team member and parent/guardian [if requested]) and led participants through a demonstration of movement artifacts (e.g., eye blinking, muscle tension) to limit external interference. EEG data were collected through an 8-channel g.Nautilus g.Ladybird System (g.tec® medical engineering GmbH, Austria). Electrodes were placed in keeping with the International 10-20 system, with the right earlobe serving as the reference. Both g.Nautilus software (g.tec® medical engineering GmbH, Austria) and MATLAB Simulink library were used to record incoming data. The sampling rate was 250 Hz, with electrode impedances remaining below 100 k Ω .

Using MATLAB and EEGLAB [68], EEG data were processed after the study appointment. The low-pass filter remained at 50 Hz and the high-pass filter remained at 0.5 Hz. EEG data were separated into 2 second epochs, with a 1 second overlap between epochs. Epochs that contained significant artifact (e.g., strong eye blinks, jaw muscle movements; $\pm 150\mu V$) were excluded. An average of 85% of collected data were acceptable among participants. EEG data were then averaged among all channels. Recording periods with under 30 clean epochs were excluded to ensure reliability and validity of collected EEG data. Over 90% of total possible epochs were retained after artifact rejection for the midline electrodes (range 91-93%).

Using a Fast Fourier Transformation, EEG data were analyzed with a 2 second Hanning window. Absolute power was calculated for both the entire frequency band (1-40 Hz) and the alpha frequency band (8-13 Hz). To calculate alpha power, the highest alpha Hz was extracted from each 2 second epoch and averaged across all included epochs for each participant. Alpha power was assessed at three electrode sites - frontal midline sagittal (Fz), central midline sagittal (Cz), and parietal midline sagittal (Pz). Note, alpha power

has an inverse relationship with electrical activity as assessed via EEG [69].

Analytic strategy and covariate considerations

To test the hypotheses of the present study, linear and logistic regression were conducted to investigate relationships among substance use (including urges and behavior), externalizing pathology (including diagnostically significant and behavior-level pathology), and alpha power (across frontal, central, and parietal cortical brain regions). Throughout the investigation, decisions to transform were data-driven and applied primarily during logistic regression analyses to satisfy critical assumptions (e.g., linearity of the logit). For greater ease in comparability across models, exponentiated coefficients (labelled as odds ratios [OR]) are included in Tables 3-6. In logistic regression models, the exponentiated coefficient represents the change in the odds of the outcome for a one-unit increase in the predictor. In log-transformed linear regression models, the exponentiated coefficient represents the multiplicative change in the outcome, with numbers greater than 1 indicating an increase and numbers less than one indicating a decrease. In non-transformed linear regression, the exponentiated coefficient represents a scaled effect estimate. Although not all exponentiated coefficients are ORs, exponentiating the coefficients allows for interpretation on a more comparable multiplicative scale, as ORs indicate in logistic regression.

With a small sample, covariates were carefully selected to avoid muddying the regression results. Each potential covariate - including age, sex, race, ethnicity, and socioeconomic status - was tested for independent relationships with the independent variables (IVs) and dependent variables (DVs) to justify their inclusion through empirical reasoning. To gauge empirical reasoning for including each control variable, statistical analyses (e.g., biserual correlations, chi-square tests) were conducted between each IV, DV, and potential control variable. When no significant relationships were evidenced between the IV/ DV and any of the control variables, this indicated that the covariates did not have significant independent impacts on the variables of interest. Thus, no control variables were included in those analyses.

Results and Discussion

Covariate outcomes and sample characteristics

Only ethnicity showed a significant independent relationship with alcohol craving in Hypothesis 1, leading to its inclusion as

a covariate in that instance. As no other significant relationships were evidenced between potential covariates and the variables of interest, the remaining analyses did not include covariates. Mean and standard deviation for each measure completed by participants are presented in Table 2. While the overall sample consisted of 54 adolescents, half of the participants (n = 27) were missing EEG

data. Missing EEG data was attributable primarily (~90%) to structural reasons that prevented EEG from being conducted (e.g., hairstyles preventing electrode connection with the scalp) and, to a much lesser extent, other factors (e.g., insufficient artifact-free data due to at-home data collection).

Variables	<i>M</i>	<i>SD</i>
CBCL [53]		
Externalizing Problems Scale	56.38	11.93
ADHD Scale	59.29	8.75
Oppositional Defiant Problems Scale	58.76	7.71
Conduct Problems Scale	59.10	7.86
ACQ-SF-R [61]	21.88	8.14
Alpha Power		
Frontal (EO)	1.25	0.85
Frontal (EC)	4.39	4.17
Central (EO)	0.81	0.56
Central (EC)	3.03	3.04
Parietal (EO)	0.67	0.82
Parietal (EC)	2.40	2.59
	C	NC
AADIS - Substance Use [56]	16	37
KSADS-COMP [50]		
Externalizing Diagnosis	27	27
ADHD Diagnosis	21	33
ODD Diagnosis	17	37
CD Diagnosis	1	53

Table 2: Variable statistics.

M = mean score of the associated clinical or EEG measure. *SD* = standard deviation of the associated clinical or EEG measure. EO = eyes-open. EC = eyes-closed. C = number of cases that met diagnostic criteria in the given analysis. NC = number of cases that did not meet diagnostic criteria in the given analysis. One AADIS [56] and three ACQ-SF-R [61] scores were excluded due to missing data.

As assessed via the KSADS-COMP [50] in the current sample, 27 participants (50% of the total sample) met criteria for any of the assessed externalizing disorders. A total of 21 participants (38.9% of the sample) met criteria for ADHD, 17 participants (31.5% of the sample) met criteria for ODD, and 1 participant (1.9% of the

sample) met criteria for CD. Of the 21 participants that met criteria for ADHD, over half (11 participants) met criteria for co-occurring ODD. The 1 participant that met criteria for CD also met criteria for ADHD and ODD. Because only one participant met the criteria for CD, analyses could not be conducted with this diagnosis as such limited

variability in the predictor would compromise model estimates and validity (see Limitations). One substance use report (AADIS [56]) and three alcohol craving reports (ACQ-SF-R [61]) were excluded due to missing data. While all substances were assessed, alcohol and marijuana were the only reported substances, which is in keeping with general patterns of early adolescent substance use [45]. For detailed information on the study's variables, see Table 2. Note, exponentiated coefficients (labelled as ORs) are reported to increase comparability across linear and logistic regression models (see Methods for elaboration).

Externalizing pathology, substance use, and craving

Diagnostic pathology and substance-related variables

Three logistic regressions were conducted with the presence of an externalizing diagnosis (ADHD, ODD, or overall externalizing diagnosis) as the IVs and the presence of substance use as the DV in each analysis. No significant results emerged among the models. Neither ADHD diagnosis ($\chi^2[1] = 0.35, p = .55$), nor ODD diagnosis ($\chi^2[1] = 1.98, p = .16$), nor overall externalizing diagnosis ($\chi^2[1] = 0.05, p = .83$) were associated with the presence of substance use among adolescents.

Three linear regressions were conducted with the presence of an externalizing diagnosis (ADHD, ODD, or overall externalizing diagnosis) as the IVs and degree of alcohol craving as the DV in each analysis. No significant results emerged among the models. Neither

ADHD diagnosis ($F[2, 48] = 1.72, p = .20$), nor ODD diagnosis ($F[2, 48] = 3.37, p = .07$), nor overall externalizing diagnosis ($F[2, 48] = 2.9, p = .09$) were associated with degree of alcohol craving among adolescents.

Behavior-level pathology and substance-related variables

Four logistic regressions were conducted with externalizing behaviors (ADHD behavior, ODD behavior, CD behavior, or overall externalizing behavior) as the IVs and the presence of substance use as the DV in each analysis. ODD behavior ($\chi^2[1] = 7.25, p < .01$), CD behavior ($\chi^2[3] = 17.76, p < .001$), and overall externalizing behavior ($\chi^2[1] = 14.08, p < .01$) were associated with the presence of substance use among adolescents. However, ADHD behavior was not related to substance use endorsement ($\chi^2[1] = 1.17, p = .28$).

Four linear regressions were conducted with externalizing behaviors (ADHD behavior, ODD behavior, CD behavior, or overall externalizing behavior) as the IVs and degree of alcohol craving as the DV in each analysis. Two of the models were significant, in which overall externalizing behavior ($F[1, 39] = 4.42, p < .05$) and CD behavior ($F[1, 39] = 5.86, p < .05$) were associated with increased alcohol craving among adolescents. Neither ADHD behavior ($F[1, 39] = 0.0007, p = .98$) nor ODD behavior ($F[1, 39] = 0.35, p = .56$) were related to degree of alcohol craving. See Table 3 for detailed statistics on the conducted analyses.

	C	NC	Substance Use				C	NC	Alcohol Craving			
			β	SE	OR	p			β	SE	OR	p
Externalizing Diagnosis	27	26	0.13	0.61	1.14	0.83	27	26	3.85	2.26	4.70	0.09
Externalizing Bx.			0.18	0.07	1.20	0.009**			0.21	0.10	1.23	0.04*
ADHD Diagnosis	21	32	-0.37	0.64	0.69	0.56	20	31	3.07	2.34	2.16	0.20
ADHD Bx.			0.04	0.04	1.04	0.28			0.004	0.14	1.00	0.98
ODD Diagnosis	17	36	0.90	0.64	2.45	0.16	16	35	4.45	2.42	8.58	0.07
ODD Bx.			0.13	0.05	1.14	0.01**			0.09	0.16	1.10	0.56
CD Diagnosis	1	52	--	--	--	--	1	50	--	--	--	--
CD Bx.			0.46	0.16	1.58	0.004**			0.35	0.15	1.42	0.02*

Table 3: Externalizing pathology, substance use, and craving statistics.

Bx. = Behavior. C = number of cases that met diagnostic criteria in the given analysis. NC = number of cases that did not meet diagnostic criteria in the given analysis. For continuous predictors, C and NC are not applicable. Substance use analyses included 53 participants (15 reported use; 38 denied use), and alcohol craving analyses included 51 participants due to missing data. * $p \leq .05$ ** $p \leq .01$ *** $p \leq .001$.

Alpha power and externalizing pathology

Frontal alpha power and externalizing pathology

Six logistic regressions were conducted with frontal alpha power (during eyes-open and eyes-closed conditions) as the IVs and the presence of an externalizing diagnosis (ADHD, ODD, or overall externalizing diagnosis) as the DVs. Frontal alpha power during eyes-open ($\chi^2[1] = 4.83, p < .05$) and eyes-closed ($\chi^2[1] = 5.29, p < .05$) conditions was associated with overall externalizing diagnosis. Frontal alpha power during the eyes-closed condition was associated with ODD diagnosis ($\chi^2[1] = 4.71, p < .05$), but not during the eyes-open condition ($\chi^2[1] = 3.24, p = .07$). Neither frontal alpha power during eyes-open ($\chi^2[1] = 1.34, p = .25$) nor eyes-closed ($\chi^2[1] = 2.24, p = .13$) conditions were related to ADHD diagnosis.

Eight linear regressions were conducted with frontal alpha power (during eyes-open and eyes-closed conditions) as the IVs and externalizing behaviors (ADHD behavior, ODD behavior, CD behavior, or overall externalizing behavior) as the DVs. Frontal alpha power during both conditions was associated with ADHD behavior (eyes-open: $F[1, 18] = 15.8, p < .001$; eyes-closed: $F[1, 18] = 7.78, p < .05$), CD behavior (eyes-open: $F[1, 18] = 6.53, p < .05$; eyes-closed: $F[1, 18] = 6.48, p < .05$), and overall externalizing behavior (eyes-open: $F[1, 18] = 8.88, p < .01$; eyes-closed: $F[1, 18] = 6.68, p < .05$). Frontal alpha power was not related to ODD behavior under eyes-open ($F[1, 18] = 2.63, p = .12$) and eyes-closed ($F[1, 18] = 3.29, p = .09$) conditions.

Central alpha power and externalizing pathology

Six logistic regressions were conducted with central alpha power (during eyes-open and eyes-closed conditions) as the IVs and the presence of an externalizing diagnosis (ADHD, ODD, or overall externalizing diagnosis) as the DVs. Central alpha power during eyes-open ($\chi^2[2] = 6.61, p < .05$) and eyes-closed ($\chi^2[1] = 4.79, p < .05$) conditions was associated with overall externalizing diagnosis. Central alpha power during the eyes-open condition was associated with ODD diagnosis ($\chi^2[3] = 9.05, p < .05$), but not during the eyes-closed condition ($\chi^2[1] = 2.23, p = .14$). Neither central alpha power during eyes-open ($\chi^2[1] = 0.15, p = .70$) nor eyes-closed ($\chi^2[1] = 2.71, p = .10$) conditions were related to ADHD diagnosis.

Eight linear regressions were conducted with central alpha power (during eyes-open and eyes-closed conditions) as the IVs and externalizing behaviors (ADHD behavior, ODD behavior, CD behavior, or overall externalizing behavior) as the DVs. Central alpha power during both conditions was associated with ADHD behavior (eyes-open: $F[1, 18] = 9.76, p < .01$; eyes-closed: $F[1, 18] = 6.91, p < .01$), CD behavior (eyes-open: $F[3, 16] = 4.93, p < .05$; eyes-closed: $F[1, 18] = 5.47, p < .05$), and overall externalizing behavior (eyes-open: $F[1, 18] = 5.91, p < .05$; eyes-closed: $F[1, 18] = 4.96, p < .05$). Central alpha power was not related to ODD behavior under eyes-open ($F[1, 18] = 3.07, p = .10$) and eyes-closed ($F[1, 18] = 1.26, p = .27$) conditions.

Parietal Alpha Power and Externalizing Pathology

Six logistic regressions were conducted with parietal alpha power (during eyes-open and eyes-closed conditions) as the IVs and the presence of an externalizing diagnosis (ADHD, ODD, or overall externalizing diagnosis) as the DVs. Only one significant result emerged between parietal alpha power during the eyes-open condition and overall externalizing diagnosis ($\chi^2[2] = 6.92, p < .05$). Eyes-closed parietal power was not related to overall externalizing diagnosis ($\chi^2[1] = 2.74, p = .10$). Neither parietal alpha power during eyes-open nor eyes-closed conditions were related to ADHD diagnosis (eyes-open: $\chi^2[1] = 0.06, p = .81$; eyes-closed: $\chi^2[1] = 0.07, p = .79$) or ODD diagnosis (eyes-open: $\chi^2[1] = 0.31, p = .58$; eyes-closed: $\chi^2[1] = 0.34, p = .56$).

Eight linear regressions were conducted with parietal alpha power (during eyes-open and eyes-closed conditions) as the IVs and externalizing behaviors (ADHD behavior, ODD behavior, CD behavior, or overall externalizing behavior) as the DVs. No significant results emerged among the models. Parietal power during both conditions was not related to ADHD behavior (eyes-open: $F[1, 18] = 1.25, p = .24$; eyes-closed: $F[1, 18] = 2.05, p = .17$), ODD behavior (eyes-open: $F[1, 18] = 1.14, p = .30$; eyes-closed: $F[1, 18] = 0.65, p = .43$), CD behavior (eyes-open: $F[1, 18] = 1.02, p = .33$; eyes-closed: $F[1, 18] = 2.71, p = .12$), or overall externalizing behavior (eyes-open: $F[1, 18] = 1.64, p = .22$; eyes-closed: $F[1, 18] = 1.64, p = .22$). See Table 4 for detailed statistics on the conducted analyses.

	C	NC	Frontal (EO)				C	NC	Frontal (EC)			
			β	SE	OR	p			β	SE	OR	p
Externalizing Diagnosis	15	12	1.18	0.62	3.26	0.056	15	12	0.28	0.15	1.33	0.06
Externalizing Bx.			8.25	2.77	3.84	0.008**			1.57	0.61	4.78	0.02*
ADHD Diagnosis	12	15	0.54	0.48	1.71	0.26	12	15	0.15	0.11	1.16	0.17
ADHD Bx.			6.35	1.60	5.75	0.0009***			1.07	0.38	2.92	0.01**
ODD Diagnosis	10	17	0.86	0.52	2.39	0.09	10	17	0.23	0.12	1.26	0.058
ODD Bx.			3.24	2.00	2.55	0.12			0.75	0.41	2.11	0.086
CD Diagnosis	1	26	--	--	--	--	1	26	--	--	--	--
CD Bx.			4.52	1.77	9.20	0.02*			0.95	0.37	2.57	0.02*
	C	NC	Central (EO)				C	NC	Central (EC)			
			β	SE	OR	p			β	SE	OR	p
Externalizing Diagnosis^a	15	12	2.23	1.19	0.11	0.06	15	12	0.39	0.23	1.48	0.088
Externalizing Bx.			11.00	4.52	5.97	0.03*			1.89	0.85	6.61	0.04*
ADHD Diagnosis	12	15	0.26	0.69	1.30	0.70	12	15	0.23	0.16	1.26	0.15
ADHD Bx.			8.49	2.72	4.86	0.006**			1.60	0.49	4.97	0.004**
ODD Diagnosis^b	10	17	-4.96	2.61	0.007	0.057	10	17	0.20	0.15	1.22	0.17
ODD Bx.			5.33	3.04	2.06	0.10			0.66	0.58	1.93	0.28
CD Diagnosis	1	26	--	--	--	--	1	26	--	--	--	--
CD Bx. ^b			17.45	5.88	0.003	0.009**			1.20	0.51	3.31	0.03*
	C	NC	Parietal (EO)				C	NC	Parietal (EC)			
			β	SE	OR	p			β	SE	OR	p
Externalizing Diagnosis^a	15	12	1.72	0.79	0.20	0.04*	15	12	0.30	0.21	1.35	0.15
Externalizing Bx.			5.11	4.00	1.66	0.22			1.34	1.05	3.83	0.22
ADHD Diagnosis	12	15	0.11	0.47	1.12	0.81	12	15	0.04	0.15	1.04	0.79
ADHD Bx.			2.92	2.61	1.85	0.28			0.96	0.67	2.62	0.17
ODD Diagnosis	10	17	-0.29	0.54	0.75	0.60	10	17	0.09	0.15	1.09	0.56
ODD Bx.			2.72	2.55	1.52	0.30			0.55	0.68	1.73	0.43
CD Diagnosis	1	26	--	--	--	--	1	26	--	--	--	--
CD Bx.			2.50	2.48	1.22	0.33			1.03	0.62	2.79	0.12

Table 4: Alpha power and externalizing pathology statistics.

C = number of cases that met diagnostic criteria in the given analysis. NC = number of cases that did not meet diagnostic criteria in the given analysis. For continuous predictors, C and NC are not applicable. EO = eyes-open. EC = eyes-closed. Bx. = behavior. ^a = quadratic-transformed predictor. ^b = cubic-transformed predictor. Analyses included 27 participants due to missing EEG data. * $p \leq .05$. ** $p \leq .01$.

*** $p \leq .00$.

Alpha power and substance use

Frontal alpha power and substance use

Two logistic regressions were conducted with frontal alpha power (during eyes-open and eyes-closed conditions) as the IVs and the presence of substance use as the DV in each analysis. While frontal alpha power during the eyes-open condition was associated with substance use endorsement among adolescents ($\chi^2[3] = 13.34, p < .01$), eyes-closed frontal alpha power was not ($\chi^2[2] = 4.08, p = .13$).

Central alpha power and substance use

Two logistic regressions were conducted with central alpha power (during eyes-open and eyes-closed conditions) as the IVs and the presence of substance use as the DV in each analysis.

Neither eyes-open ($\chi^2[1] = 0.30, p = .58$) nor eyes-closed ($\chi^2[1] = 0.07, p = .79$) central alpha power were related to substance use endorsement.

Parietal alpha power and substance use

Two logistic regressions were conducted with parietal alpha power (during eyes-open and eyes-closed conditions) as the IVs and the presence of substance use as the DV in each analysis. While parietal alpha power during the eyes-closed condition was associated with substance use endorsement among adolescents ($\chi^2[2] = 6.79, p < .05$), eyes-open parietal alpha power was not ($\chi^2[3] = 7.37, p = .06$). See Table 5 for detailed statistics on the conducted analyses.

			Substance Use			
	C	NC	β	SE	OR	p
Frontal	9	18				
EO ^b			-3.53	1.36	0.03	0.01**
EC ^a			0.04	0.03	1.04	0.11
Central	9	18				
EO			0.39	0.72	1.48	0.58
EC			-0.04	0.14	0.96	0.79
Parietal	9	18				
EO ^b			-2.34	1.15	0.10	0.04*
EC ^a			0.23	0.13	1.25	0.07

Table 5: Alpha power and substance use statistics.

C = number of cases that met diagnostic criteria in the given analysis. NC = number of cases that did not meet diagnostic criteria in the given analysis. For continuous predictors, C and NC are not applicable. EO = eyes-open. EC = eyes-closed. ^a = quadratic-transformed predictor. ^b = cubic-transformed predictor. Analyses included 27 participants (9 reported substance use; 18 denied substance use) due to missing EEG data. * p

$\leq .05$. ** $p \leq .01$. *** $p \leq .001$.

Alpha power and alcohol craving

Frontal alpha power and craving

Two linear regressions were conducted with frontal alpha power (during eyes-open and eyes-closed conditions) as the IVs and degree of alcohol craving as the DV in each analysis. Neither eyes-open ($F[1, 24] = 0.02, p = .89$) nor eyes-closed ($F[1, 24] = 0.51, p = .48$) frontal alpha power was related to alcohol craving.

Central alpha power and craving

Two linear regressions were conducted with central alpha power (during eyes-open and eyes-closed conditions) as the IVs and degree of alcohol craving as the DV in each analysis. While central alpha power during the eyes-open condition was associated with alcohol craving among adolescents ($F[3, 22] = 3.58, p < .05$), eyes-closed central alpha power was not ($F[1, 24] = 0.009, p = .92$).

Parietal alpha power and craving

Two linear regressions were conducted with parietal alpha power (during eyes-open and eyes-closed conditions) as the IVs and degree of alcohol craving as the DV in each analysis. Neither

eyes-open ($F[1, 24] = 1.09, p = .31$) nor eyes-closed ($F[1, 24] = 0.86, p = .36$) parietal alpha power was related to alcohol craving. See Table 6 for detailed statistics on the conducted analyses.

	Alcohol Craving			
	β	<i>SE</i>	OR	<i>p</i>
Frontal				
EO	0.29	2.12	1.34	0.89
EC	0.30	0.43	1.36	0.48
Central	-20.52	6.55	1.23	0.005**
EO ^b	0.06	0.59	1.06	0.92
EC				
Parietal				
EO	-2.25	2.16	0.11	0.31
EC	-0.64	0.69	5.29	0.36

Table 6: Alpha power and alcohol craving statistics.

EO = eyes-open. EC = eyes-closed. ^a = quadratic-transformed predictor. ^b = cubic-transformed predictor. Analyses included 27 participants due to missing EEG data. * $p \leq .05$. ** $p \leq .01$. *** $p \leq .001$.

Conclusion

Summary of findings and interpretations

The aim of the current study was to examine the relationships among externalizing pathology, substance use, and alpha power in a sample of at-risk adolescents. Externalizing behavior (particularly ODD and CD behavior) was associated with substance use behavior and urges. These findings introduced a pattern evidenced across externalizing pathology, in which behaviors, rather than diagnostic categories, were more strongly linked to potential risk factors (e.g., substance use risk, brain-based factors) among the current sample of adolescents. Findings on alpha power and externalizing pathology extend this pattern, with frontal and central alpha power more consistently relating to behavior-level externalizing pathology (namely ADHD and CD behavior) but not those diagnoses. One of the only findings to diverge from this pattern - alpha power and ODD diagnosis - may reflect the mood component involved in assessing ODD among adolescents, which is not present in the other externalizing disorders (e.g., ADHD and CD).

Similar to externalizing pathology, frontal and central alpha power were also related to substance use risk. While frontal alpha power was implicated in substance use endorsement among adolescents, central alpha power was implicated in alcohol craving. As such, different mechanisms may underlie the relationships between alpha power and substance use actions versus urges in adolescents. In keeping with available research [46,47], early adolescents initiating substance use seem to display similar brain characteristics (i.e., high alpha power) to what is seen among adults identified as being at increased risk of substance use in the literature.

Externalizing pathology and substance risk

Given the body of literature indicating that externalizing pathology (including ODD and CD, with ADHD to a lesser extent) may confer risk of substance use [6,9,20,21], we hypothesized that such pathology would be associated with substance use behavior and urges. However, the findings suggest that a more complex relationship exists among our early adolescent sample. Namely,

two patterns emerged: 1) ODD and CD pathology (but not ADHD pathology) were related to substance use behavior and/or urges, and 2) significant results emerged only on the behavioral (but not diagnostic) level.

Adolescents who displayed increased ODD, CD, and overall externalizing behaviors were more likely to endorse substance use. Similarly, adolescents who displayed increased CD and overall externalizing behaviors reported greater alcohol craving. Contrary to predictions, ADHD pathology was not associated with either sub-stance-related variable among adolescents. This divergence of ADHD from the rest of the assessed externalizing pathology is not wholly surprising, as evidence supporting the connection between ADHD pathology and substance use tends to be less consistent in comparison to ODD and CD pathology [20,70-72]. Some research has indicated that ADHD behavior is associated with substance use [20,21,71,73], while other findings have suggested that ADHD behavior may not be related to substance use independently (i.e., without comorbid ODD or CD pathology; [70,72,74]).

When considering why differences in the substance-related variables emerged among adolescents with ADHD pathology in comparison to ODD and CD pathology, the varying behavioral characteristics of these externalizing disorders should be noted. While ADHD is categorized as a neurodevelopmental condition within the DSM-5, both ODD and CD fall under the Disruptive, Impulse-Control, and Conduct Disorders category [15]. Disruptive behavior disorders (DBDs; e.g., ODD, CD) are characterized by defiant, rule-breaking, and sometimes illegal behaviors. On the other hand, ADHD behavior often consists of milder behavioral displays, such as distractibility or difficulty waiting turns [15]. Thus, it is not necessarily surprising that substance use - a risky and illegal behavior for minors, often with significant consequences - was associated with DBD pathology, and not ADHD pathology, in the current sample.

While ADHD pathology was not related to either of the substance-related variables, DBD pathology was related at the behavioral level but not the diagnostic level. This pattern may reflect the greater sensitivity inherent to behavior-level assessment, as assessment on a continuum can capture subthreshold that binary diagnostic assessment might miss. No associations emerged among assessed externalizing diagnoses (excluding CD due to an insufficient

number of diagnoses) and substance use behaviors or urges. When considering why the substance-related variables were associated with DBD behaviors, but not DBD diagnoses, an interesting trend emerges that remains largely consistent throughout the current study's findings - perhaps it is not as much about the diagnosis of interest as it is about the behaviors of interest when investigating risk factors (e.g., substance use behavior or urges) among adolescents. In the case of externalizing pathology and substance use, it appears that DBD behaviors are connected to substance use, whether DSM-5 criteria are fully met or not. In the present investigation, adolescents and their parents/guardians needed to endorse specific DSM domain dysfunction and duration criteria to receive an externalizing disorder diagnosis [15]. The current results indicate that such criteria, which result in a diagnosis, may not be as helpful when identifying early adolescents at risk of substance use. The finding that the substance-related variables were associated with DBD behaviors, and not DBD diagnoses, suggests that in developing prevention programs for teens, externalizing behavior may be a better indicator of substance use risk among adolescents.

Alpha power and externalizing pathology

Several effects were found among alpha power and externalizing pathology, most widely and consistently on the behavioral level. Of all cortical brain regions studied (including frontal, central, and parietal regions), frontal and central alpha power were consistent indicators of externalizing behavior. Overall externalizing behavior, ADHD behavior, and CD behavior were associated with increased frontal and central alpha power under both eyes-open and eyes-closed conditions. On the diagnostic level, overall externalizing diagnosis was also associated with increased frontal and central alpha power under both conditions. These findings reflect predictions of the current study, as well as predictions made in the literature regarding the relationship between alpha power and externalizing pathology [22,24,25,33-35,75,76].

Albeit from a small pool of literature, researchers have proposed that alpha power may be related to externalizing pathology due to hypoarousal underlying ADHD and antisocial behaviors [22,32,75,76]. These hypoarousal models [24,25,77,78] propose that individuals with such experiences are in a chronic state of CNS hypoarousal (i.e., low arousal), which contributes to engaging in externalizing behavior as a form of self-stimulation to

counter this chronic state of low arousal. Given that low arousal is associated with high alpha power, one would expect to see higher alpha power (and, subsequently, lower arousal) among individuals engaging in externalizing behavior. While the literature appears mixed as to whether such an association exists between alpha power and externalizing behavior through hypoarousal mechanisms [33,75,79,80], the results of the present investigation offer potential support for these hypoarousal models. Given that the current study did not directly assess arousal, future work should incorporate measures of arousal (e.g., skin conductance level; [33,35,75]) to clarify connections among arousal level, alpha power, and externalizing pathology among adolescents.

In some cases, ODD diagnosis was also associated with frontal (eyes-open) and central (eyes-closed) alpha power. While this finding is in keeping with predictions, replication is warranted because significant associations were only evidenced during one EEG recording condition for the frontal and central regions (eyes-open or eyes-closed, but not both). In exploring why alpha power was related to ODD diagnosis, but not ODD behavior, it is important to consider how informant reporting may have contributed to the differences seen. The presence of an ODD diagnosis was gauged through both the adolescent and parent report. ODD behavior, on the other hand, was gauged through only parent report. Note, a significant effect between alpha power and ODD pathology only emerged when the adolescent had input into the measure. The divergence in findings between differing informant reports is further understood as ODD includes an internal mood-related component (e.g., being resentful; [15]), which is not always apparent to an outside observer. Thus, it is possible that the behavior-level ODD information differs from the diagnostic ODD information because the latter includes adolescents' report of their internal mood experiences. Including adolescent report, in addition to parent report, in future studies could be useful for fully representing the adolescents' experience with ODD and likely other pathology as well.

Contrary to frontal and central alpha power, parietal alpha power evidenced minimal associations with externalizing pathology among adolescents. More specifically, no associations emerged on the behavioral level and only one association emerged on the diagnostic level with overall externalizing diagnosis during the eyes-open condition. In exploring why parietal alpha power

diverges from the other cortical brain regions in its relation to externalizing pathology, gaps in the literature become apparent. Much of the literature on alpha power has centered around the frontal cortex, interhemispheric asymmetry (i.e. EEG asymmetry; [81,82]), and internalizing or psychotic pathology [44,83,84]. The present investigation and literature review indicate that additional study of alpha power across cortical brain regions is needed to better understand relationships between externalizing pathology and alpha power, particularly among adolescents. Future EEG research could consider analyzing midline alpha power to help fill these gaps in the literature as such data is often already collected as a part of the EEG recording process.

Alpha power, substance use behavior, and urges

Similar to externalizing pathology, it has been hypothesized that individuals with CNS hypoarousal (and, coincidentally, increased alpha power) use substances as a form of self-stimulation [26-29]. Drawing from patterns evidenced in adult research, it was hypothesized that decreased alpha power would be associated with endorsement of substance use among adolescents, while increased alpha power would be associated with greater alcohol craving (intended to gauge substance use urges; [46,47]). In keeping with the latter prediction, alcohol craving was associated with increased central alpha power (eyes-open) among the current sample of adolescents. Given the young age of our sample, the craving scores were likely indicative of desire or curiosity around substances, rather than craving in the physiological sense as is primarily seen among adults. These findings are in keeping with currently available adult research, suggesting that increased alpha power may confer risk for substance use among adolescents [46].

Contrary to the former prediction, substance use was associated with increased (rather than decreased, as hypothesized) alpha power at the frontal (eyes-open) cortical region. Note, this effect was also seen in the parietal (eyes-closed) region, but the low power of this result suggests significant caution in interpreting. Low rates of substance use among early adolescents may help to explain this divergence from predictions. While adult literature primarily includes people who are regularly using their drug of choice or meet substance use disorder criteria [46,47], epidemiological data on substance use among young adolescents indicates relatively low rates of use in this population (12-to-14-year-olds; [13]). This distinction may help to explain why the

current findings with adolescents do not follow the same pattern evidenced in adults. It is possible (and likely, given epidemiological data; [13]) that substances were not being used by the adolescents at a rate that might have reflected a reduction in alpha power, as is seen among adult samples. Future work should assess chronicity of substance use among adolescents, potentially including older adolescents who use substances at higher rates [85], to see if the pattern of alpha power evidenced among substance using adults is also present for older adolescents.

The current work offers preliminary evidence that both substance use and alcohol craving are related to alpha power during adolescence, albeit in different cortical brain regions. While substance use was associated with frontal (and potentially parietal) alpha power, alcohol craving was associated with central alpha power. It is important to note that the substance use and alcohol craving measures are getting at different constructs (behavior versus urges) as well as different timelines (one year versus present moment), which may help to explain why different cortical regions are implicated. For example, substance use was assessed over a longer period, reflecting a state (i.e., stable) experience. On the other hand, craving was assessed in the present moment, reflecting a trait (i.e., temporary) experience. As resting-state alpha power was conceptualized as a trait risk factor for substance use risk, this distinction between substance use and alcohol craving may help to explain why the findings diverge. In sum, a primary takeaway from the separately implicated cortical regions is that different mechanisms may underlie the relationships between alpha power and substance use versus urges in adolescents. Further research is warranted to explore whether such findings are upheld and, if so, why such connections may exist (e.g., arousal mechanisms).

Limitations

Sample size and low prevalence of variables

Despite the strengths of this home-based study on at-risk adolescents, there are limitations to note. Additionally, the current investigation's small sample size ($N = 54$) led to some challenges, including limited statistical power in a few analyses, which increases the likelihood of Type II errors [86]. The small sample size and the expected low base rates of certain variables (e.g., CD diagnosis, substance use) exacerbated some challenges with statistical analyses. For example, although the low prevalence of CD in the current sample mirrors epidemiological data (1.9%;

[87,88]), statistical analyses with this diagnosis could not be run as only one participant met DSM-5 criteria for CD. Thus, there is an unavoidable gap in information on how CD as a diagnosis relates to the other variables of interest, including substance use and alpha power. Similarly, despite the expected low rates of substance use and alcohol craving among this sample of 12-to-14-year-olds [13], low prevalence and variability in the substance-related variables likely reduced statistical power [87]. Had the sample been larger and the prevalence of such variables been subsequently higher, all of the present research questions could have been fully explored.

Home-based EEG

Under ideal circumstances, EEG would have been conducted in a lab-controlled environment to greater reduce external interference. However, requiring the population of interest to do so was not feasible due to the significant financial barriers (e.g., transportation, childcare; see Table 1) faced by most of the sample. The study team took proactive steps to minimize external interference (e.g., removing distracting stimuli from the recording space, demonstrating movement artifacts to participants). While the recording environment was less favorable, the home-based nature of the current study allowed for inclusion of a population that has historically been underrepresented in research. Replication in lab-controlled environments is warranted to see if findings remain consistent across recording conditions.

Alpha correction

Traditionally, alpha correction (e.g., Bonferroni correction) is implemented in psychological research to reduce risk of Type I errors [89]. However, given the hypothesis-driven nature of the current investigation, such corrections were not necessarily required and would have resulted in an overly restrictive alpha value (e.g., $p < .005$). Much of the present work, including the EEG-related hypotheses, informs clinical and neuropsychological theory. For this reason, an alpha correction was not applied so that we could evaluate potential associations to inform future work. However, we understand that this study is a first step, and future studies with larger samples that include alpha corrections are needed to validate the current findings.

Marginal significance of exploratory predictors

In a few regression models, the overall model was significant, while the predictor was marginally significant (i.e., $p = .05-.09$).

Findings like this can be due to various factors [90,91]. Within the context of the current work, it is likely that the small sample size (and, subsequently, lower power) contributed to variability in significance between the overall model and individual predictors. Thus, it is possible that the marginal significance of some predictors may reflect low power rather than a lack of true effects.

Despite the reasons for variability in significance, the fact that some predictors were marginally significant on their own indicates that such findings should be interpreted cautiously. Given the potential theoretical utility of the current work for the areas of clinical and neuropsychology, further investigation is needed to see if such effects among alpha power, externalizing pathology, and substance use are present among other samples of early adolescents. As with any research, replication (especially work with larger samples) is warranted.

Implications

Theory

The current work has theoretical implications, particularly for hypoarousal models of externalizing pathology and substance use [24,25,27-29,77]. In this sample of early adolescents, increased alpha power was associated with externalizing pathology. Given the connections made in the literature between increased alpha power and low CNS arousal [33-35,75], the present work offers provisional support for the hypothesis that low arousal may underlie externalizing pathology among early adolescents [38,43,92,93]. Additionally, early adolescents endorsing substance use evidenced a similar pattern of increased alpha power that has been reflected among adults identified in the literature as being at heightened risk of substance use [46]. Thus, multiple data points indicate provisional support for these hypoarousal models and, as such, continued research in this area is warranted. Gaining a better understanding of alpha power may help the field better understand if (and how) low arousal may underlie externalizing pathology and substance use risk. As the current alpha power findings have been found in more economically diverse samples (e.g., [46,94]), the current study may be more broadly applicable.

Research

In addition to theoretical implications, the current findings highlight multiple areas where continued research is needed. Given the bidirectional nature of the brain and behavior, our

results highlight the need to examine whether increased alpha power precedes externalizing pathology and substance use, or arises as a result. Although the current findings suggest hypoarousal mechanisms, it is also possible that the alpha power differences observed could reflect influences of the psychosocial stressors experienced by our sample (e.g., socioeconomic stress). Additionally, since alpha abnormalities have also been observed in internalizing pathology, increased alpha power may be indicative of a more general neural dysregulation. Future research, including longitudinal work with more diverse samples (in terms of pathology and demographics), is warranted to clarify these considerations.

Broader research implications also arise from the present work. As evidenced by the results, alpha power at frontal and central cortical brain regions was implicated in both externalizing pathology and substance use behaviors and urges. As the frontal region of the cortex is associated with executive functioning and the mesolimbic reward pathway [95-97], it makes sense that abnormal electrical brain activity at this location may relate to externalizing pathology and substance use. It may be helpful for future research to explore connections between underlying brain structures, such as those included in the mesolimbic pathway, and alpha power as it relates to hypoarousal models. Note that the field still has many areas to grow regarding hypoarousal and alpha power in the cortex before tying in the study of subcortical brain structures. Such work would involve neuroimaging techniques outside of EEG that can observe subcortical structures, expanding the expertise needed for such research.

The present investigation also highlights areas to grow with regard to standardization of EEG practices in the realm of alpha power research and EEG research as a whole. The present literature review and previous meta-analytic research [82] show significant variability in EEG recording conditions (i.e., eyes-open, eyes-closed, both, or an average of the two) across studies. This distinction is important within the context of the current findings, as results occasionally vary depending on the EEG recording condition (i.e., eyes-open or eyes-closed). Transparent and consistent reporting on EEG recording conditions, including differences that emerge among conditions, is important for establishing potential relationships between electrical brain activity and risky behaviors (e.g., externalizing pathology, substance use) as alpha power research continues to grow.

Clinical

Clinical implications also arise as a result of the current findings. A trend emerged among several of the analyses in which associations were evident on the behavioral level, yet not on the diagnostic level, with regard to externalizing pathology in the adolescent sample. This pattern suggests that it may not be as much about the diagnosis of interest as it is about the behaviors of interest when studying and identifying risky behaviors (e.g., substance use) and risk factors (e.g., alpha power) among early adolescents. While this pattern serves to guide future research, it also has implications for treatment as it encourages a continued focus on behavior-based psychotherapy treatments for adolescents experiencing DBD behaviors [98-100]. Further, the results highlight the importance of assessing and treating DBDs on the behavioral or symptom level, even if the clinical findings are not diagnostically significant. Should these findings hold true across replications, it would be important for regularly used screening tools (e.g., in schools, doctor's offices) to implement behavior-level screenings of externalizing pathology. As evidenced by the current results, heightened risk (e.g., greater likelihood of substance use) is associated with subclinical DBD presentations among early adolescents.

Conclusion

Taken together, the results of the current investigation implicate alpha power in externalizing pathology and substance use (behavior and urges) among early adolescents. These findings offer provisional support for hypoarousal hypotheses, which suggest that low CNS arousal may underlie such behaviors [24,25,27-29,77]. This work also highlights the overall need for continued research and replication in this area of study. The literature regarding alpha power, externalizing pathology, and substance use is relatively limited, particularly among adolescent samples. The present findings on alpha power highlight the potential value and utility of alpha power in research on adolescent mental and behavioral health.

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Conflict of Interest

The authors declare no known conflicts of interest.

Bibliography

1. Armstrong T and E Costello. "Community Studies on Adolescent Substance Use, Abuse, or Dependence and Psychiatric Comorbidity". *Journal of Consulting and Clinical Psychology* 70.6 (2003): 1224-1239.
2. Bukstein O G. "Disruptive Behavior Disorders and Substance Use Disorders in Adolescents". *Journal of Psychoactive Drugs* 32.1 (2000): 67-79.
3. Disney E R., et al. "Effects of ADHD, Conduct Disorder, and Gender on Substance Use and Abuse in Adolescence". *American Journal of Psychiatry* 156.10 (1999): 1515-1521.
4. Hopfer C., et al. "Conduct Disorder and Initiation of Substance Use: A Prospective Longitudinal Study". *Journal of the American Academy of Child and Adolescent Psychiatry* 52.5 (2013): 511-518.e4.
5. King KM and L Chassin. "A Prospective Study of the Effects of Age of Initiation of Alcohol and Drug Use on Young Adult Substance Dependence". *Journal of Studies on Alcohol and Drugs* 68.2 (2007): 256-265.
6. Masroor A., et al. "Conduct Disorder-Related Hospitalization and Substance Use Disorders in American Teens". *Behavioral Sciences* 9.7 (2019): 1-13.
7. Nock M K., et al. "Cross-National Prevalence and Risk Factors for Suicidal Ideation, Plans, and Attempts". *British Journal of Psychiatry* 192.2 (2008): 98-105.
8. Reebye P., et al. "Conduct Disorder and Substance Use Disorder: Comorbidity in a Clinical Sample of Preadolescents and Adolescents". *Canadian Journal of Psychiatry* 40.6 (1995): 313-319.
9. Köck P., et al. "Co-Occurring Mental Disorders in Transitional Aged Youth with Substance Use Disorders - A Narrative Review". *Frontiers in Psychiatry* 13 (2022): 1-9.
10. Blakemore S J. "The Social Brain in Adolescence". *Nature Reviews Neuroscience* 9.4 (2008): 267-277.
11. Kapur S. "Adolescence: The Stage of Transition". *Horizons of Holistic Education* 2 (2015): 233-250.
12. Yurgelun-Todd D. "Emotional and Cognitive Changes during Adolescence". *Current Opinion in Neurobiology* 17 (2007): 251-257.

13. Substance Abuse and Mental Health Services Administration. "2021 NSDUH Annual National Report". Center for Behavioral Health Statistics and Quality, Substance Abuse and Mental Health Services Administration, Dec. 2022. Web. 14 Nov. (2023).
14. Volkow N D., *et al.* "Prevalence of Substance Use Disorders by Time since First Substance Use among Young People in the US". *JAMA Pediatrics* 175.6 (2021): 640-643.
15. American Psychiatric Association. "Diagnostic and Statistical Manual of Mental Disorders". 5th ed., Text Revision, American Psychiatric Publishing, (2022).
16. Sayette M A. "The Role of Craving in Substance Use Disorders: Theoretical and Methodological Issues". *Annual Review of Clinical Psychology* 12 (2016): 407-433.
17. Vafaie N and H Kober. "Association of Drug Cues and Craving with Drug Use and Relapse: A Systematic Review and Meta-analysis". *JAMA Psychiatry* 79.6 (2022): 641-650.
18. Van Zundert R M., *et al.* "Dynamic Effects of Craving and Negative Affect on Adolescent Smoking Relapse". *Health Psychology* 31.2 (2012): 226-234.
19. Ramirez J and R Miranda. "Alcohol Craving in Adolescents: Bridging the Laboratory and Natural Environment". *Psychopharmacology* 231.9 (2014): 1841-1851.
20. Groenman A P., *et al.* "Childhood Psychiatric Disorders as Risk Factor for Subsequent Substance Abuse: A Meta-analysis". *Journal of the American Academy of Child and Adolescent Psychiatry* 56.7 (2017): 556-569.
21. Wilens TE., *et al.* "Does ADHD Predict Substance Use Disorders? A 10-Year Follow-Up Study of Young Adults with ADHD". *Journal of the American Academy of Child and Adolescent Psychiatry* 50.6 (2011): 543-553.
22. Bellato A., *et al.* "Is Autonomic Nervous System Function Atypical in Attention Deficit Hyperactivity Disorder (ADHD)? A Systematic Review of the Evidence". *Neuroscience and Biobehavioral Reviews* 108 (2020): 182-206.
23. Howells F M., *et al.* "Synergistic Tonic and Phasic Activity of the Locus Coeruleus Norepinephrine (LC-NE) Arousal System Is Required for Optimal Attentional Performance". *Metabolic Brain Disease* 27.2 (2012): 267-274.
24. Satterfield JH and DP Cantwell. "Proceedings: CNS Function and Response to Methylphenidate in Hyperactive Children". *Psychopharmacology Bulletin* 10 (1974): 36-37.
25. Zentall S S and T R Zentall. "Optimal Stimulation: A Model of Disordered Activity and Performance in Normal and Deviant Children". *Psychological Bulletin* 94.3 (1983): 446-471.
26. Evans B E., *et al.* "Alcohol and Tobacco Use and Heart Rate Reactivity to a Psychosocial Stressor in an Adolescent Population". *Drug and Alcohol Dependence* 126.1-2 (2012): 296-303.
27. Evans B E., *et al.* "Stress Reactivity as a Prospective Predictor of Risky Substance Use during Adolescence". *Journal of Studies on Alcohol and Drugs* 77.2 (2016): 208-219.
28. Gallant C and D Good. "Alcohol Misuse and Traumatic Brain Injury: A Review of the Potential Roles of Dopaminergic Dysfunction and Physiological Underarousal Post-Injury". *Applied Neuropsychology: Adult* 28.5 (2021): 501-511.
29. Kliewer W., *et al.* "Emotion Dysregulation, Anticipatory Cortisol, and Substance Use in Urban Adolescents". *Personality and Individual Differences* 99 (2016): 200-205.
30. Monastra V., *et al.* "Assessing Attention Deficit Hyperactivity Disorder via Quantitative Electroencephalography: An Initial Validation Study". *Neuropsychology* 13.3 (1999): 424-433.
31. Nunez P L and R Srinivasan. "Electric Fields of the Brain: The Neurophysics of EEG". 2nd ed., Oxford University Press (2006).
32. Barry R J., *et al.* "A Review of Electrophysiology in Attention-Deficit/Hyperactivity Disorder: I. Qualitative and Quantitative Electroencephalography". *Clinical Neurophysiology* 114.2 (2003): 171-183.
33. Barry R J., *et al.* "EEG Differences between Eyes-Closed and Eyes-Open Resting Conditions". *Clinical Neurophysiology* 118.12 (2007): 2765-2773.
34. Lim C L., *et al.* "The Relationship between Quantified EEG and Skin Conductance Level". *International Journal of Psychophysiology* 21.2 (1996): 151-162.
35. Starita F., *et al.* "Theta and Alpha Power Track the Acquisition and Reversal of Threat Predictions and Correlate with Skin Conductance Response". *Psychophysiology* 60.3 (2023): e14247.

36. Lazzaro I., *et al.* "Simultaneous EEG and EDA Measures in Adolescent Attention Deficit Hyperactivity Disorder". *International Journal of Psychophysiology* 34.2 (1999): 123-134.
37. Baving L., *et al.* "Frontal EEG Correlates of Externalizing Spectrum Behaviors". *European Child and Adolescent Psychiatry* 12.1 (2003): 36-42.
38. Niv S., *et al.* "Childhood EEG Frontal Alpha Power as a Predictor of Adolescent Antisocial Behavior: A Twin Heritability Study". *Biological Psychology* 105 (2015): 72-76.
39. Niv S., *et al.* "Relationships of Alpha, Beta, and Theta EEG Spectra Properties with Aggressive and Nonaggressive Antisocial Behavior in Children and Adolescents". *American Journal of Psychology* 131.4 (2018): 277-284.
40. Sohn H., *et al.* "Linear and Non-Linear EEG Analysis of Adolescents with Attention-Deficit/Hyperactivity Disorder during a Cognitive Task". *Clinical Neurophysiology* 121.11 (2010): 1863-1870.
41. Knyazev G G., *et al.* "Impulsivity, Anxiety, and Individual Differences in Evoked and Induced Brain Oscillations". *International Journal of Psychophysiology* 68.3 (2008): 242-254.
42. Hobbs M J., *et al.* "EEG Abnormalities in Adolescent Males with AD/HD". *Clinical Neurophysiology* 118.2 (2007): 363-371.
43. Kitsune GL., *et al.* "A Matter of Time: The Influence of Recording Context on EEG Spectral Power in Adolescents and Young Adults with ADHD". *Brain Topography* 28.4 (2015): 580-590.
44. RudoHutt A S. "Electroencephalography and Externalizing Behavior: A MetaAnalysis". *Biological Psychology* 105 (2015): 1-19.
45. Monitoring the Future. "Monitoring the Future Annual Reports". Institute for Social Research, The University of Michigan, Jan. 2023. Web. 14 November (2023).
46. Liu Y., *et al.* "RestingState EEG, Substance Use, and Abstinence after Chronic Use: A Systematic Review". *Clinical EEG and Neuroscience* 53.4 (2022): 344-366.
47. Newson J J and T C Thiagarajan. "EEG Frequency Bands in Psychiatric Disorders: A Review of Resting State Studies". *Frontiers in Human Neuroscience* 12 (2019): 521.
48. TorresRosado L., *et al.* "Operational Definitions and Measurement of Externalizing Behavior Problems: An Integrative Review Including Research Models and Clinical Diagnostic Systems". *World Journal of Psychiatry* 13.6 (2023): 278-297.
49. Scott A., *et al.* "COINS: An Innovative Informatics and Neuroimaging Tool Suite Built for Large Heterogeneous Datasets". *Frontiers in Neuroinformatics* 5 (2011): 33.
50. Townsend L., *et al.* "Development of Three WebBased Computerized Versions of the Kiddie Schedule for Affective Disorders and Schizophrenia Child Psychiatric Diagnostic Interview: Preliminary Validity Data". *Journal of the American Academy of Child and Adolescent Psychiatry* 59.3 (2020): 309-325.
51. Kaufman J., *et al.* "Schedule for Affective Disorders and Schizophrenia for SchoolAge ChildrenPresent and Lifetime Version (KSADSPL): Initial Reliability and Validity Data". *Journal of the American Academy of Child and Adolescent Psychiatry* 36.7 (1997): 980-988.
52. Kroenke K., *et al.* "The PHQ9: Validity of a Brief Depression Severity Measure". *Journal of General Internal Medicine* 16.9 (2001): 606-613.
53. Achenbach T M and L. Rescorla. "Manual for the ASEBA SchoolAge Forms and Profiles: An Integrated System of MultiInformant Assessment". ASEBA, Burlington, VT, (2001).
54. Knepley M J., *et al.* "An Analysis of the Child Behavior Checklist Anxiety Problems Scale's Predictive Capabilities". *Journal of Psychopathology and Behavioral Assessment* 41.2 (2019): 249-256.
55. Nakamura B J., *et al.* "A Psychometric Analysis of the Child Behavior Checklist DSMOriented Scales". *Journal of Psychopathology and Behavioral Assessment* 31.2 (2009): 178-189.
56. Moberg D. "Screening for Alcohol and Other Drug Problems Using the Adolescent Alcohol and Drug Involvement Scale (AADIS)". Center for Health Policy and Program Evaluation, University of Wisconsin—Madison, (2003).
57. Blumenthal H., *et al.* "Social Anxiety and Motives for Alcohol Use among Adolescents". *Psychology of Addictive Behaviors* 24.3 (2010): 529-534.

58. Blumenthal H., *et al.* "A LaboratoryBased Test of the Relation between Adolescent Alcohol Use and PanicRelevant Responding". *Experimental and Clinical Psychopharmacology* 23.3 (2015): 303-313.
59. Nkyi A. "Substance Abuse among Senior High School Students in Ghana". *International Journal of Social Sciences Education* 4.3 (2014): 346-353.
60. Moberg D P and L Hahn. "The Adolescent Drug Involvement Scale". *Journal of Adolescent Chemical Dependence* 2.1 (1991): 75-88.
61. Singleton E G., *et al.* "Alcohol Craving Questionnaires (ACQNOW and ACQSFR): Background, Scoring, and Administration". Baltimore, MD, (1994).
62. You D., *et al.* "Emotion Regulation Strategies Moderate the Impact of Negative Affect Induction on Alcohol Craving in College Drinkers: An Experimental Paradigm". *Journal of American College Health* 71.4 (2021): 1538-1546.
63. Flannery B A., *et al.* "Psychometric Properties of the Penn Alcohol Craving Scale". *Alcoholism: Clinical and Experimental Research* 23.9 (1999): 1289-1295.
64. Rodrigues R., *et al.* "Portuguese Validation of the Alcohol Craving Questionnaire-Short Form-Revised". *PLoS ONE* 16 (2021): e0251733.
65. Augur IF., *et al.* "Distress Tolerance, Coping Motives, and Alcohol Craving and Consumption: Two Experiments Testing Momentary Responses to a Mood Induction". *Drug and Alcohol Dependence* 253 (2023): 111034.
66. Saunders J B., *et al.* "Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO Collaborative Project on Early Detection of Persons with Harmful Alcohol Consumption - II". *Addiction* 88.6 (1993): 791-804.
67. Frahm W G. "Cognitive Biases in Alcohol and Marijuana Users". Master's thesis, Central Washington University, Ellensburg, WA, (2017).
68. Delorme A and S Makeig. "EEGLAB: An Open Source Toolbox for Analysis of SingleTrial EEG Dynamics Including Independent Component Analysis". *Journal of Neuroscience Methods* 134.1 (2004): 9-21.
69. Allen J J B., *et al.* "Issues and Assumptions on the Road from Raw Signals to Metrics of Frontal EEG Asymmetry in Emotion". *Biological Psychology* 67.2 (2004): 183-218.
70. Brook DW., *et al.* "Association between AttentionDeficit/Hyperactivity Disorder in Adolescence and Substance Use Disorders in Adulthood". *Archives of Pediatrics and Adolescent Medicine* 164.10 (2010): 930-934.
71. Groenman A P., *et al.* "Substance Use Disorders in Adolescents with Attention Deficit Hyperactivity Disorder: A 4Year Follow-Up Study". *Addiction* 108.8 (2013): 1503-1511.
72. SerraPinheiro M A., *et al.* "Is ADHD a Risk Factor Independent of Conduct Disorder for Illicit Substance Use? A Meta-Analysis and Meta regression Investigation". *Journal of Attention Disorders* 17.5 (2013): 459-469.
73. Lee SS., *et al.* "Prospective Association of Childhood AttentionDeficit/Hyperactivity Disorder (ADHD) and Substance Use and Abuse/Dependence: A MetaAnalytic Review". *Clinical Psychology Review* 31.2 (2011): 328-341.
74. August G J., *et al.* "Prospective Study of Adolescent Drug Use among Community Samples of ADHD and NonADHD Participants". *Journal of the American Academy of Child and Adolescent Psychiatry* 45.7 (2006): 824-832.
75. Barry R J., *et al.* "EEG Differences in Children as a Function of RestingState Arousal Level". *Clinical Neurophysiology* 115.2 (2004): 402-408.
76. Kavish N., *et al.* "Physiological Arousal and Juvenile Psychopathy: Is Low Resting Heart Rate Associated with Affective Dimensions?" *Psychiatric Quarterly* 88.1 (2017): 103-114.
77. Hare R D and S A Thorvaldson. "Psychopathy and Response to Electrical Stimulation". *Journal of Abnormal Psychology* 76.3 (1970): 370-374.
78. Coren S. "Arousal Predisposition as a Predictor of Antisocial and Delinquent Behavior". *Personality and Individual Differences* 27.5 (1999): 815-820.
79. Clarke A R., *et al.* "Children with AttentionDeficit/Hyperactivity Disorder and Comorbid Oppositional Defiant Disorder: An EEG Analysis". *Psychiatry Research* 111.2 (2002): 181-190.
80. Clarke A R., *et al.* "EEG Activity in Girls with AttentionDeficit/Hyperactivity Disorder". *Clinical Neurophysiology* 114.2 (2003): 319-328.
81. Zhao Y., *et al.* "Alterations in Gray Matter Volumes and Intrinsic Activity in the Prefrontal Cortex Are Associated with Suicide Attempts in Patients with Bipolar Disorder". *Psychiatry Research: Neuroimaging* 307 (2021): 111229.

82. Butler L B and K B Nooner. "The Link between Suicidality and Electroencephalography Asymmetry: A Systematic Review and MetaAnalysis". *Clinical Psychopharmacology and Neuroscience* 21.4 (2023): 419-428.
83. Greco C., *et al.* "Discriminative Power of EEGBased Biomarkers in Major Depressive Disorder: A Systematic Review". *IEEE Access* 9 (2021): 112850.
84. Perrottelli A., *et al.* "Unveiling the Associations between EEG Indices and Cognitive Deficits in Schizophrenia Spectrum Disorders: A Systematic Review". *Diagnostics* 12.9 (2022): 2193.
85. Jordan C J and S L Andersen. "Sensitive Periods of Substance Abuse: Early Risk for the Transition to Dependence". *Developmental Cognitive Neuroscience* 25 (2017): 29-44.
86. Cohen J. "A Power Primer". *Psychological Bulletin* 112.1 (1992): 155-159.
87. Bachmann C J., *et al.* "Recognition and Management of Children and Adolescents with Conduct Disorder: A RealWorld Data Study from Four Western Countries". *Child and Adolescent Psychiatry and Mental Health* 18 (2024): 18.
88. Mohammadi M R., *et al.* "The Global Prevalence of Conduct Disorder: A Systematic Review and MetaAnalysis". *Iranian Journal of Psychiatry* 16.2 (2021): 205-225.
89. Holm S. "A Simple Sequentially Rejective Multiple Test Procedure". *Scandinavian Journal of Statistics* 6.1 (1979): 65-70.
90. Fisher R. "Statistical Methods, Experimental Design, and Scientific Inference: A ReIssue of Statistical Methods for Research Workers, The Design of Experiments and Statistical Methods and Scientific Inference". Oxford University Press, (1990).
91. Tabachnick B G and L S Fidell. "Using Multivariate Statistics". Pearson Education (2013).
92. Byeon J., *et al.* "A Novel Quantitative Electroencephalography Subtype with High Alpha Power in ADHD: ADHD or Misdiagnosed ADHD?" *PLoS One* 15. (2020): e0242566.
93. Kiiski H., *et al.* "EEG Spectral Power, but Not Theta/Beta Ratio, Is a Neuro marker for Adult ADHD". *European Journal of Neuroscience* 51.9 (2020): 2095-2109.
94. Baker L., *et al.* "The Southern California Twin Register at the University of Southern California: III". *Twin Research and Human Genetics* 16.3 (2013): 336-343.
95. Berridge K C and M L Kringelbach. "Pleasure Systems in the Brain". *Neuron* 86.3 (2015): 646-664.
96. Haber SN and B Knutson. "The Reward Circuit: Linking Primate Anatomy and Human Imaging". *Neuropsychopharmacology* 35.1 (2010): 4-26.
97. Stuss D T and R T Knight. "Principles of Frontal Lobe Function". 2nd ed., Oxford University Press, (2012).
98. Aldabbagh R., *et al.* "Systematic Review and MetaAnalysis of the Effectiveness of TeacherDelivered Interventions for Externalizing Behaviors". *Journal of Behavioral Education* 33.2 (2024): 233-274.
99. Correll C U., *et al.* "Efficacy and Acceptability of Pharmacological, Psychosocial, and Brain Stimulation Interventions in Children and Adolescents with Mental Disorders: An Umbrella Review". *World Psychiatry* 20.2 (2021): 244-247.
100. Valero Aguayo L., *et al.* "MetaAnalysis of the Efficacy and Effectiveness of ParentChild Interaction Therapy (PCIT) for Child Behaviour Problems". *Psicothema* 33.4 (2021): 544-555.