Explaining the Covariance Between Attention-Deficit Hyperactivity Disorder Symptoms and Depressive Symptoms: The Role of Hedonic Responsivity

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Objective: The aim of this study was to examine low hedonic responsivity, a facet of hedonic capacity, as a potential explanatory variable in the relationship between attention deficit/hyperactivity disorder (ADHD) symptoms and depressive symptoms. **Method:** One hundred ninety-eight undergraduate students (mean age = 21.3, standard deviation = 4.6; 59.6% women) from a large, public university completed self-report measures for this cross-sectional study. **Results:** Results indicated that ADHD symptoms were significantly associated with depressive symptoms, and that low hedonic responsivity partially accounted for this association. This effect was statistically significant for total ADHD symptoms and inattentive symptoms, but not for hyperactive-impulsive symptoms. **Conclusions:** Findings are consistent with the possibility that impaired hedonic responsiveness may be a common endophenotype for depression and the inattentive symptoms of ADHD. Implications for future research and clinical work are discussed. © 2012 Wiley Periodicals, Inc. J. Clin. Psychol. 68:1111–1121, 2012.

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Attention-deficit hyperactivity disorder (ADHD) is one of the most commonly occurring childhood disorders, affecting up to 9% of children (Centers for Disease Control, 2010). Though once considered solely a childhood disorder, ADHD has been found to persist into adolescence in 50%-80% of cases and into adulthood in 10%-50% of cases (Barkley, Murphy & Kwasnik, 1996; Klassen, Katzman, & Chokka, 2010). Recent data indicate a 6-month prevalence rate of 3.4% among adults (McIntosh et al., 2009). ADHD is associated with numerous detrimental outcomes in adolescence and early adulthood, including academic failure, delinquency, substance abuse, automobile accidents, and risky sexual behavior (Barkley et al., 1996; Barkley, 2006; Klassen et al., 2010). Additionally, college students with ADHD have been shown to have lower levels of college adjustment, academic achievement, self esteem, and social skills (Shaw-Zirt, Popali-Lehane, Chaplin, & Bergman, 2005; Frazier, Youngstrom, Glutting, & Watkins, 2007) compared with college students without ADHD.

Individuals with ADHD also show higher rates of comorbid externalizing, anxiety, learning, and depressive disorders than individuals without a diagnosis of ADHD (Bagwell, Molina, Pelham, & Hoza, 2001; Biederman, Faraone, Mick, & Lelon, 1995; Flory, Molina, Pelham, Gnagy, & Smith, 2006; Hoza, Pelham, Waschbusch, Kipp, & Owens, 2001; Jensen et al., 2001; Meinzer et al., 2012; Milberger, Biederman, Faraone, & Murphy, 1995). For example, a recent report estimated that the prevalence of major depressive disorder (MDD) in the past year was 18.6% in adults with ADHD compared with 7.8% in non-ADHD adults (McIntosh et al., 2009). Similarly, the prevalence of dysthymia in the past year was 12.8% in adults with ADHD compared with 1.9% of non-ADHD adults (McIntosh et al., 2009). Research also indicates that

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individuals with comorbid ADHD-MDD experience higher levels of psychosocial impairment than individuals with either disorder in isolation (Biederman et al., 2008; Biederman, Mick, & Faraone, 1998; Biederman, Newcorn, & Sprich, 1991; Chronis-Tuscano et al., 2010).

The high rates of comorbidity between ADHD and unipolar depressive disorders and the high level of impairment associated with their co-occurrence highlight the need to develop an understanding of the mechanisms underlying their relationship. One potential explanation for the covariance of depressive symptoms and ADHD symptoms is shared endophenotypes. Endophenotypes are constructs that underlie psychopathological symptoms and are more directly influenced by genes than the manifest symptoms (Rende & Waldman, 2006; Turetsky et al., 2008). Recent work from genetic, neurological, and behavior paradigms has drawn attention to the possible role of altered reward system functioning in both ADHD and major depressive disorder (MDD). For example, molecular genetic studies have linked several dopaminergic and serotonergic genes to both ADHD (Wood & Neale, 2010) and MDD (Kato, 2007), and evidence indicates that neural activity in dopamine-mediated reward circuitry is linked to negative affect, low motivation, inattention, and depression (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008; Durston, 2003; Epstein et al., 2006; Pizzagalli, Iosifescu, Hallet, Ratner, & Fava, 2009; Scheres, Milham, Knutson, & Castellanos, 2007).

Given the moderate to high heritability estimates for ADHD and MDD and the overlapping neurotransmitter systems and neural circuitry involved in ADHD and MDD, it is possible that altered reward system functioning (i.e., hedonic capacity; Meehl, 1975) represents a common endophenotype for symptoms seen in both depression and ADHD. The purpose of the present study was to examine that possibility among college students who completed self-report measures of ADHD symptoms, depressive symptoms, and hedonic responsiveness.

Hedonic responsivity, a feature of hedonic capacity, is a heritable individual difference in reactivity to pleasurable stimuli and reward (Bogdan & Pizzagalli, 2009). Hedonic responsiveness has often been studied in the context of the mesocortical dopamine system, where the role of dopamine has received the most focus as a neural substrate of reward processing (Naranjo, Tremblay, & Busto, 2001). Research has established elements of poor hedonic responsiveness as a feature of both depression and ADHD (Bogdan & Pizzagalli, 2006; Forbes & Dahl, 2005; Luman, Oosterlaan, & Sergeant, 2005; Pizzagalli, Jahn, & O'Shea, 2005; Pizzagalli et al., 2009). A substantial body of research has documented impaired hedonic responsivity in depression and has demonstrated that the failure to respond to rewarding stimuli maps onto the severity of anhedonic symptoms (Bogdan & Pizzagalli, 2006; Forbes, 2009; Pizzagalli et al., 2005; Pizzagalli et al., 2009; Shankman, Klein, Tenke, & Bruder, 2007).

Furthermore, comparative research has replicated this general deficit in hedonic responsivity, as well as a blunted dopaminergic reward system, among animals bred to display depressive behaviors (Naranjo et al., 2001). Neurological studies have revealed that depressed individuals display decreased activity in the striatum in response to rewarding stimuli, specifically in the region associated with the detection of rewards and the representation of reward-related goals (Forbes, 2009; Forbes & Dahl, 2005). Additionally, Heller and colleagues (2009) reported that compared with nondepressed controls, individuals with MDD displayed a decrease in activation over time in the nucleus accumbens, a region associated with motivation and reward processing. This may reflect difficulty sustaining positive affect following reward (Heller et al., 2009).

Research also supports impaired hedonic responsivity in ADHD. Scheres and colleagues (2007) reported reduced ventral striatal activation during anticipation of reward among adolescents with ADHD as compared with healthy controls. Similarly, neural activation during dopaminergic-driven reward tasks was negatively correlated with severity of ADHD symptoms among individuals with ADHD (Stark et al., 2011). In behavioral paradigms, children with ADHD show less response to both positive and negative reinforcement as well as reward and extinction conditions (Luman et al., 2005).

Furthermore, research has suggested that children with ADHD have a tendency to choose smaller rewards sooner rather than delay gratification for a larger reward, which may represent an impulsive drive for immediate rewards and a delay aversion (Marco et al., 2009). However, research regarding responsiveness to reward incentives has received far less attention and it

remains unclear whether children with ADHD show hyper- or hypo-responsiveness to rewarding incentives (Scheres et al., 2007).

Additionally, prior research has indicated that reward preferences and inhibitory deficits were not significantly correlated (Solanto et al., 2001), which suggests that the poor impulse inhibition often seen in individuals with ADHD is distinct from their responsiveness to rewards. Though little research has examined reward processing across ADHD subtypes, the inattentive subtype of ADHD may relate to dysfunctional reward patterns through its association with sluggish cognitive tempo, or marked drowsiness, lethargy, passivity, and forgetfulness, a key feature in ADHD-Inattentive subtype (Derefinko et al., 2008). Given that a tendency to automatically attend to reward-related stimuli is an important aspect of appraising the incentive salience of cues (Berridge, Robinson, & Aldrige, 2009), it is also possible that inattention may disrupt one's ability to process the incentive properties of reward-related stimuli, which could in turn result in impaired reward responsiveness.

Even though evidence suggests dysregulated hedonic responsiveness in both depression and ADHD, prior research has not examined it as an explanation for the covariation between depression and ADHD symptoms. Given the overlap in neural circuitry affected in regards to reward processing in ADHD and MDD as well as similar findings of irregular reward processing, the current cross-sectional study examined the role of hedonic responsiveness in accounting for the relationship between ADHD symptoms and depressive symptoms. In this initial investigation, these associations were studied in a sample of college students. Examining factors that explain depression-ADHD relationships among college students is important because this is a high-risk period for depression, ADHD symptoms, and their co-occurrence (Kisch, Leino, & Silverman, 2005; Weyendat & Dupal, 2006). Based on prior research and theory, we expected that ADHD symptoms would be significantly and positively associated with depressive symptoms, and that ADHD symptoms and depressive symptoms each would be significantly and negatively associated with hedonic responsivity. Finally, we hypothesized that hedonic responsivity would significantly account for the covariation between ADHD symptoms and depressive symptoms, such that there would be a significant indirect effect of ADHD symptoms on depressive symptoms via hedonic responsiveness.

Method

Participants and Procedures

Participants were recruited through announcements made in undergraduate psychology classes and through flyers posted for the general student body at a large public university in an urban setting that announced the opportunity to participate in a study on mood, emotions, and sexual orientation. Participants drawn from psychology classes (80.8% of the total sample) received extra course credit for their participation. The remainder (19.2%) of undergraduate students received $10 as compensation for their time. No mean score on measured variables significantly differed across the two recruitment methods (all $ps > .05$). We therefore did not covary recruitment method in analyses.

The sample comprised 198 undergraduate students. Participants (59.6% women) ranged from 18–46 years old (mean = 21.3, standard deviation [*SD*] = 4.6). The majority of participants endorsed Hispanic/Latino ethnicity (74.2%). The racial composition was as follows: 67.1% White, 13.6% Black, 5.5% Asian, 1.5% Native American, and 0.5% Pacific Islander. Twenty-five participants (12.6%) selected an "Other" category for race. Percentages sum to over 100% due to a small number of participants selecting more than one racial category. Thirty-nine percent of the sample was born outside of the United States. Among those, the mean number of years residing in the United States was 11.34 ($SD = 6.09$). Given the mean age of the sample (21.28 years), this indicates that a large portion of those who were born outside the United States immigrated here during childhood or early adolescence. The racial and ethnic composition of this sample is representative of the university from which the sample was drawn and the community in which the university is located.

After providing written consent to take part in the study, participants completed paper and pencil measures. The study was conducted as approved by the institutional review board.

Measures

Adult ADHD Self-Report Scale (ASRS; Kessler et al., 2005). The ASRS is an 18-item self-report measure that is based on the 18 Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994) Criteria A symptoms of adult ADHD. Each question asks individuals to rate how often a symptom has occurred in the last 6 months (e.g., "How often do you make careless mistakes when you have to work on a boring or difficult project?"). Response options range from 0 (*never*) to 4 (*very often*). Scores on each item are dichotomized (see Kessler et al., 2005), with total scores ranging from 0–18 and higher scores indicating more severe symptoms. A score of nine or higher has been recommended as a clinical cut-off for being at high risk for ADHD (Kessler et al., 2005). Previous research has supported the measure's internal reliability ($\alpha = 0.88$), sensitivity, and specificity (Kessler et al., 2005), and convergent validity via associations with clinician-rated ADHD symptoms (Adler et al., 2006). As has been done in past research among college students (e.g., Arria et al., 2011), we computed a total ADHD score and separate scores for inattention and hyperactivity-impulsivity. Internal consistency in the present study was acceptable for the total scale ($\alpha = .86$) and for the inattentive and hyperactive/impulsive subscales ($\alpha = .82$ and $\alpha = .77$, respectively).

Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). Depressive symptoms were measured with the CES-D. It is a 20-item, self-report scale on which participants rate the frequency of different depressive symptoms experienced in the past week. Items are rated on a 4-point Likert scale ranging from 0 (*rarely or none of the time [less than 1 day]*) to 3 (*most or all of the time [5–7] days*). Scores range from zero to sixty. Recommended clinical cut-off scores range from 16–24 (Radloff, 1977; Roberts, Lewinsohn, & Seeley, 1991). It has demonstrated excellent reliability and validity in many samples, including young adults (Joiner, Walker, Pettit, Perez, & Cukrowicz, 2005). Internal consistency in the present study was acceptable ($\alpha = .86$).

Tripartite Pleasure Inventory (TPI; Leventhal, 2012). The TPI describes 12 types of experiences that span interest/pastimes, social interaction, sensory, and goals/mastery (e.g., learning new information or skills, romantic or sexual activities). For each experience, participants are asked to rate how much pleasure/happiness/enjoyment they usually feel in response to these experiences (hedonic responsivity), how often they usually engage in these experiences (hedonic engagement), and how strongly they usually want to engage in these experiences (hedonic desire). Individuals base their responses on their usual perceptions and behaviors. For the responsivity subscale, items are ranked on a 5-point Likert scale ranging from 0 (*no pleasure*) to 4 (*extreme pleasure*), with higher scores indicating greater pleasure. A mean score is then computed for the responsivity subscale, resulting in averages ranging from 0 to 4. Given the focus on hedonic responsivity as a common liability to depression and ADHD in present study, only the hedonic responsivity subscale was included in analyses. The TPI-responsivity subscale has illustrated adequate internal consistency (αs ranging .77 – .87) and good convergent validity with depressive symptoms and other measures of hedonic capacity in college student and adult general population samples (Leventhal, 2012; Leventhal et al., 2012). The internal consistency of the hedonic responsivity subscale in this sample was acceptable ($\alpha = .78$).

Results

Means of, standard deviations of, and intercorrelations between measures are presented in Table 1. Mean scores on measures of depressive symptoms and ADHD symptoms fell below clinical cut-offs, as expected in this sample of college students. As hypothesized, and consistent with prior research, depressive symptoms were significantly and positively correlated with total ADHD symptoms, inattentive ADHD symptoms, and hyperactive-impulsive ADHD

Table 1
Means of, Standard Deviations of, and Intercorrelations Between Measures

Variable	1	2	3	4	5	6	7
1. ASRS	–						
2. ASRS-HI	0.83**	–					
3. ASRS-IA	0.88**	0.47**	–				
4. TPI-R	−0.16*	−0.09	−0.18**	–			
5. CESD	0.40**	0.28**	0.39**	−0.34**	–		
6. Age	−0.19**	−0.13	−0.19**	0.11	−0.16*	–	
7. Sex	−0.15*	−0.11	−0.15**	−0.10	0.001	−0.04	–
M	6.13	2.55	3.59	2.93	13.12	21.28	–
SD	3.81	2.02	2.42	0.48	8.31	4.46	–

Note. ASRS = Total Score for Adult ADHD Self-Report Scale; ASRS-HI = Hyperactive/Impulsive Subscale Score of ASRS; ASRS-IA = Inattention Subscale Score of ASRS; TPI-R = Hedonic Responsivity Subscale of Tripartite Pleasure Inventory; CESD = Center for Epidemiological Studies Depression Scale; M = Mean; SD = Standard Deviation; For Sex, 0 = Female and 1 = Male (i.e., coefficients with a positive valence indicate higher scores among men and coefficients with a negative valence indicate higher scores among women).
N = 198. **p < .01. *p < .05.

symptoms. Also as predicted, hedonic responsivity was significantly and negatively correlated with depressive symptoms, total ADHD symptoms, and inattentive ADHD symptoms, but not with hyperactive-impulsive ADHD symptoms.

To test the hypothesis that the association between ADHD symptoms and depressive symptoms would be accounted for by low hedonic responsivity, we used a nonparametric resampling procedure with n = 5,000 bootstrap resamples to derive a point estimate and 95% confidence interval for the indirect effect. Confidence intervals that do not include zero indicate a statistically significant indirect effect. The INDIRECT program for mediation effects (Preacher & Hayes, 2008) was employed to estimate the indirect effect and 95% confidence interval. This program allows for the inclusion of covariates and adjusts paths accordingly. The INDIRECT program provided five relevant path coefficients in its output: a coefficient (a) for the path from the independent variable (ADHD symptoms) to the mediator (hedonic responsivity); a coefficient (b) for the path from the mediator (hedonic responsivity) to the dependent variable (depressive symptoms); a coefficient (a*b) for the indirect effect of ADHD symptoms on depressive symptoms through hedonic responsivity; a coefficient (c) for the total effect of ADHD symptoms on depressive symptoms; and a coefficient (c') for the direct effect of ADHD symptoms on depressive symptoms (i.e., the total effect minus the indirect effect). In the present study, participant age and gender were entered as covariates because of their significant correlations with ADHD symptoms and depressive symptoms (although conclusions did not differ when age and gender were excluded from analyses).

As hypothesized, the indirect effect of total ADHD symptoms on depressive symptoms via hedonic responsivity was statistically significant (see the a × b column in Table 2). Similarly, the indirect effect of inattentive ADHD symptoms via hedonic responsivity was statistically significant. In contrast, the indirect effect of hyperactive/impulsive ADHD symptoms via hedonic responsivity was not statistically significant).

Discussion

The aim of the current study was to examine hedonic responsivity as a potential endophenotype that may account for the covariation of depressive symptoms and ADHD symptoms among college students. Consistent with prior research (Norwalk, Norvilitis, & MacLean, 2009; Rucklidge, Brown, Crawford, & Kaplan, 2007), we found the following: (a) significant positive correlations were found between ADHD symptoms and depressive symptoms; (b) significant

Table 2
Summary of Point Estimates for Mediation of ADHD Symptoms Predicting Depression Symptoms

(IV)	Effect of IV on M (a)	Effect of M on DV (b)	Direct Effect (c′)	Total effect of IV on DV (c)	Indirect Effects (a × b)	95% BCA CI LL	95% BCA CI UL
1. ASRS	−0.021*	−4.83**	0.75**	0.85**	0.10[a]	0.02	0.27
2. ASRS-HI	−0.020	−5.43**	1.00**	1.11**	0.11	−0.08	0.45
3. ASRS-IA	−0.037*	−4.75**	1.14**	1.32**	0.18[a]	0.036	0.406

Note. IV = independent variable (ASRS = total score on Adult ADHD Self-Report Scale; ASRS-HI = Hyperactive/Impulsive Subgroup Score of ASRS; ASRS-IA = Inattention Subgroup Score of ASRS); DV = dependent variable (CESD = total score on Center for Epidemiological Studies Depression Scale); M = mediating variable (TPI-R = Responsivity Subscale of Tripartite Pleasure Inventory); BCA CI = Bias Corrected and Accelerated Confidence Interval; LL = Lower Limit; UL = Upper Limit.
For the indirect effects, confidence intervals that include zero are interpreted as nonsignificant.
[a]Signifies significant indirect effects; 5000 bootstrap samples.
**$p < .01$. *$p < .05$.

negative correlations of hedonic responsiveness to total ADHD symptoms, inattentive ADHD symptoms, and depressive symptoms; and (c) no association between hedonic responsiveness and hyperactive/impulsive ADHD symptoms. As hypothesized, a significant indirect effect of total ADHD symptoms on depressive symptoms via hedonic responsiveness was found. When ADHD symptoms were divided into an inattentive subgroup and a hyperactive/impulsive subgroup, a significant indirect effect on depressive symptoms via hedonic responsiveness was found for inattentive symptoms, but not for hyperactive/impulsive symptoms. This provides preliminary evidence that hedonic responsiveness may account specifically for the covariation of depressive symptoms and inattentive symptoms of ADHD.

To our knowledge, this is the first study that has examined the role of hedonic responsivity in the relationship between ADHD and depressive symptoms. Research using behavioral and neurological paradigms has linked dysfunctional reward processing with both depressive symptoms and ADHD symptoms (Forbes & Dahl, 2005; Bogdan & Pizzagalli, 2006; Pizzagalli et al., 2005; Pizzagalli et al., 2009; Luman et al., 2005). The present study replicated that pattern and extended past work by demonstrating that the relationship between ADHD and depressive symptoms was accounted for by impaired hedonic responsivity. Additionally, in line with previous work (Derefinko et al., 2008) and our hypothesis, the indirect effect of ADHD symptoms on depressive symptoms via hedonic responsivity was significant for the inattentive subtype but not the hyperactive/impulsive subtype. Perhaps, if it is difficult to remain attentive, individuals may have a hard time processing salient rewarding cues in their environment. This could give rise to anhedonia, resulting in sustained low levels of positive affect, which could ultimately give rise to depressive symptoms.

Alternatively, depression is also marked by attentional difficulties, as evidence by the inclusion of diminished ability to think or concentrate, or indecisiveness as a symptom for depression (American Psychiatric Association, 1994). One might expect that anhedonic forms of depression in particular might be more tightly linked with cognitive and motivational symptoms of depression, which could be manifested as ADHD-inattentive symptoms. By contrast, nonanhedonic and more distress-related forms of depression would be less likely to relate to amotivational characteristics.

Another potential explanation of the association between ADHD symptoms and depressive symptoms may be the repeated experience of failures among individuals with ADHD. Adolescents and young adults with ADHD often exhibit considerable impairment in social skills (Shaw-Zirt et al., 2005) or romantic relationships (Canu & Carlson, 2007; Murphy & Barkley, 1996). Repeated failures in these domains may lead them to experience less pleasure or enjoyment from these experiences, which in turn could lead to depressive symptoms.

Because of the cross sectional design, this model could not be tested within the current study.

Regardless of the mechanism, results suggest that anhedonia may be a potential endophenotype that marks an underlying shared vulnerability that influences both ADHD and depression. Thus, clinicians may wish to be more vigilant about screening for depression, and specifically anhedonic symptoms, in individuals presenting the inattentive subtype of ADHD. Conversely, clinicians may also wish to screen for ADHD, specifically for the inattentive subtype, in individuals with depression with anhedonia, rather than in depressed patients without anhedonia.

The findings of this study should be taken in light of several limitations. The cross-sectional study design prevents conclusions regarding the direction of associations. Self-report rating scales were used as measures of depression and ADHD symptoms in a nonclinical sample. Past research has indicated that individuals with ADHD often underreport their symptoms (Sibley et al., 2010); therefore, it may be possible that the severity of ADHD symptomology was minimized by participants, which could have restricted the range of ADHD symptom scores and hence resulted in underestimates of the strength of the associations of ADHD to depressive symptoms and hedonic responsivity. Future studies are encouraged to replicate the present findings using multiple informants.

In addition, hedonic responsivity was measured through a self-report questionnaire given the preliminary nature of this research. Now that initial evidence has been obtained to support hedonic responsivity as a potential explanatory variable for the covariation between symptoms of ADHD and depression, neurological (e.g., functional brain imaging of neural responses to rewards) and objective laboratory (e.g., indicators of behavioral reactions to reward) measures of hedonic responsiveness (e.g., Dichter, Kozink, McClernon, & Smoski, 2011; Pizzagalli et al., 2005) should be incorporated in future studies to obtain multiple measures of the construct.

Additional limitations of the current study relate to the sample: college students, the majority of whom were of Hispanic/Latino ethnicity. Consequently, these results may not generalize to other samples, including samples with predominantly nonminority makeup, older adults, or adolescents who may display different presentations of ADHD symptoms and depressive symptoms. Although the sample consisted primarily of ethnic minority students, mean scores on the ASRS and CESD were comparable to those reported in other college student samples (Garnier-Dykstra, Pinchevsky, Caldeira, Vincent, & Arria, 2010; Wells, Klerman, & Deykin, 1987; Ridner, Staten, & Danner, 2005). This pattern is consistent with findings of cross-cultural equivalence in the expression of ADHD (Bauermeister, Canino, Polanczyk, & Rohde, 2010). Unfortunately, data were not available on participants' first language (i.e., English or another language), but all participants were enrolled in a university in the United States, the majority of the sample was born in the United States, and those who were not born in the United States typically immigrated during childhood or early adolescence. As such, English language skills likely had a minimal impact on the validity of responses to the self-report measures.

Finally, the use of a college student sample likely resulted in a somewhat restricted ranged of ADHD symptom scores because individuals with ADHD are less likely to attend a 4-year college than individuals without ADHD (Kuriyan et al., 2010). Future studies are encouraged to replicate the present findings in clinical samples and among participants from various racial and ethnic backgrounds.

In summary, preliminary evidence was found to support hedonic responsiveness as a potential explanatory variable for the covariation between ADHD symptoms and depressive symptoms in a college student sample. When examined for the two subtypes of ADHD symptoms, the relationship was only significant for the inattentive subtype of ADHD not for the hyperactive/impulsive subtype. These findings suggest that one potential endophenotype for ADHD and depression is impaired hedonic responsiveness. Future research is encouraged to examine this relationship within a clinical sample as well as to incorporate other measures of hedonic responsivity. If this finding is replicated using other measures of hedonic responsivity, longitudinal designs, and in clinical samples, then it would suggest hedonic responsivity as a target for preventing the development of depression among individuals with ADHD. Finally, given the weak to moderate strength of the correlations between hedonic responsivity and symptom

measures, future research is encouraged to consider additional constructs that may account for additional variance in the association between ADHD symptoms and depressive symptoms.

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