Dependent Stress Mediates the Relation between ADHD Symptoms and Depression

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Abstract

Background: Previous literature has established that ADHD symptoms are associated with increased dependent stress, or stress that an individual has a part in causing for themselves. Additionally, there is a strong link between dependent stress and depression. However, no previous studies have tested a longitudinal model examining dependent stress as a mediator in the relation between ADHD and subsequent depression symptoms. Furthermore, no studies have examined whether inattentive or hyperactive/impulsive ADHD symptoms are more predictive of dependent stress and depression. **Methods**: We examined this model in a semester-long longitudinal study of 153 emerging adult college students. Demographic and clinical (ADHD symptoms, dependent stress frequency, depression symptoms) information were collected via self-reported rating scales. **Results**: As hypothesized, the association between total ADHD symptoms at baseline and later depression symptoms was accounted for by dependent stress. There was no significant difference between the effects of inattentive versus hyperactive/impulsive ADHD symptoms on subsequent dependent stress and depression. **Conclusions**: These findings suggest that increased generation of stressors is one mechanism that explains increases in depression for individuals with ADHD symptoms.

Keywords: ADHD, dependent stress, depression

Dependent Stress Mediates the Relation between ADHD Symptoms and Depression

Depression is a debilitating mood disorder with onset typically during adolescence and young adulthood, and is prevalent at alarming rates during this developmental period (Mojtabai, Olson, & Han, 2016). Data from nationally representative samples indicate that approximately 11% of adolescents and young adults have experienced a major depressive episode in the last 12 months (Mojtabai et al., 2016), and approximately 30% will do so in their lifetime (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Many more people experience depressive symptoms at some point in their lives and these elevated depression symptoms in adolescence predict many negative outcomes in adulthood, including decreased health status (Ayuso-Mateos, Nuevo, Verdes, Naidoo, & Chatterji, 2010; Kessler et al., 2012). Because depression is so prevalent and debilitating, there is a pressing need to identify risk factors during this developmental period (Meinzer et al., 2016; Mojtabai et al., 2016).

One such risk factor may be Attention Deficit Hyperactivity Disorder (ADHD), which is highly comorbid with depression (Garnier-Dykstra, Pinchevsky, Caldeira, Vincent, & Arria, 2011). ADHD is a prevalent disorder with symptoms emerging before the age of 12, characterized by symptoms of inattention (ADHD-I), hyperactivity/impulsivity (ADHD-H), or both (ADHD-C). Among North American college students, the prevalence of clinically significant ADHD symptoms ranges from 2% to 12% (Nugent 2014; Garnier-Dykstra et al., 2011). Importantly, the rate of major depressive disorder (MDD) in youth with ADHD is 5.5 times higher than in youth without ADHD, with rates ranging from 12% to 50% (Daviss, 2008).

Co-morbid depression and ADHD pose special challenges as individuals with both disorders display higher levels of psychosocial impairment than youth with either ADHD or depression occurring alone (Daviss, 2008). Childhood ADHD significantly predicts higher levels

of depressive symptoms throughout emerging adulthood (Meinzer et al., 2016; Rabiner, Anastopoulos, Costello, Hoyle, & Swartzwelder, 2008), and individuals with co-morbid ADHD and depression have higher rates of depressive recurrence (Daviss, 2008). Chronic treatment of persistent ADHD symptoms can have a preventative effect on the development of depressive symptoms (Meinzer & Chronis-Tuscano, 2017), suggesting a potential causal relation between ADHD and depression. Yet, the mechanisms linking ADHD and depression are not yet fully understood.

Several theories about the relation between ADHD and depression have previously been explored. Given that ADHD typically precedes the onset of depression, studies of the association between ADHD and depression have focused on ADHD-related impairment or failure-related mediators, such that depression may follow from ADHD-related demoralization (Humphreys et al., 2013). The dual-failure model proposes that both academic and peer problems, resulting from disruptive behavior associated with ADHD, contribute to the development of depression (Humphreys et al., 2013; Patterson & Stoolmiller, 1991). In fact, parent-child, peer, and academic problems have been shown to mediate the longitudinal relation between ADHD and depression (Humphreys et al., 2013). However, these impairment and failure-related difficulties may actually be encompassed by a larger risk factor: stress.

Many studies have investigated stress-like constructs as potential mediators between ADHD and depression (e.g., Herman, Lambert, Ialongo, & Ostrander, 2007; Humphreys et al., 2013; Ostrander & Herman, 2006). However, no study has directly tested whether stress generation mediates the link between ADHD and depression. The present study proposes a new model for the association between ADHD symptoms and depression: individuals with ADHD

may experience higher levels of depression because their symptoms predispose them to the generation of stressors that subsequently increase their risk for depression.

Stress and Depression

Stress plays a critical role in the onset of depression (Colodro-Conde et al., 2017). In fact, the majority of clinically significant depressive episodes are triggered by stressful life events (Hammen, 2018). In community samples, more than 80% of individuals who meet criteria for clinically significant depression have experienced a recent major life event or ongoing stressor (Brown & Harris, 1989); these rates are high for clinical samples as well (Mazure, 1989). The diathesis-stress model explains this relation between stress and depression, stating that a disorder is a result of the interaction between vulnerability and stress (Colodro-Conde et al., 2017).

In addition to interacting with stress to cause depression (diathesis-stress), the stress generation hypothesis states that vulnerabilities can also lead to the generation of stressors, which may then lead to depression (for a review see Liu & Alloy, 2010). The stress generation hypothesis draws attention to the role of the individual as an active contributor rather than a passive player in his or her environment and indicates that people who are predisposed to depression symptoms exhibit behaviors that cause stressful life events (Hammen, 2006). Importantly, such dependent stressors, stressful events due at least in part to an individual's characteristics or behaviors (e.g., relationship problems or failing a test), are more predictive of depressive symptoms and major depressive episodes than independent stressors, or fateful events to which an individual does not contribute (e.g., a hurricane or an illness in the family) in longitudinal studies of adolescents and young adults (Kendler, Karkowski, & Prescott, 1999; Technow, Hazel, Abela, & Hankin, 2018). The probability of depression onset is estimated to be 80% greater for dependent stressors than for independent stressors (Kendler et al., 1999;

Technow et al., 2018) and those who have previously experienced an episode of depression report more dependent stressors compared to those who have not (Hammen, 1991). This highlights the importance of gaining a better understanding of stress generation and the vulnerabilities that may promote it. Several vulnerabilities including negative cognitive styles, rumination, and certain personality traits such as neuroticism have been shown to predict depression via stress generation, supporting this hypothesis (Liu & Alloy, 2010). Another factor that can lead to generation of dependent stress, and thus depression, may be ADHD, but this has not previously been directly tested.

ADHD and Stress

ADHD traits are robustly and positively associated with stress (Combs, Canu, Broman-Fulks, Rocheleau, & Nieman, 2015). The core symptoms of ADHD create and intensify stress for individuals by promoting inattentive, impulsive, and stimulus seeking behaviors as well as difficulty blocking out distractions (Jackson & Farrugia, 1997). Cross-sectional studies of adolescents (Overbey, Snell, & Callis, 2009) and adults (Combs et al., 2015; Hirvikoski, Lindholm, Nordenström, Nordström, & Lajic, 2009) with ADHD demonstrate these behaviors make navigating the day-to-day world more difficult, leading to stressful situations such as academic and job struggles and interpersonal rejection (Young, 2000). These may then cause significant impairment in many life activities and add to chronic heightened stress.

Because these stressful situations instigated by ADHD are the result of behaviors stemming from the individual, they are by nature dependent stressors. Indeed, one study found that dependent stressors including academic performance, relationship with friends, and relationship with family were among the top sources of concern for college students with ADHD (Beiter et al., 2015). Thus, ADHD may be characterized by stress generative behaviors that

increase levels of dependent stressors. Although there is evidence that ADHD symptoms increase specific types of stressors such as the ones listed above (e.g., interpersonal stress) (Herman et al., 2007; Humphreys et al., 2013; Ostrander & Herman, 2006), the relation between ADHD and overall dependent stressors has not yet been tested.

Furthermore, the relation between ADHD and dependent stress may differ between inattentive and hyperactive-impulsive symptoms. College students are more commonly diagnosed with symptoms of ADHD-I, at a rate of approximately 4.4%, than with ADHD-H, at a rate of 1.3% (Nugent & Smart, 2014; Combs et al., 2015). In a study of adults with ADHD, ADHD-H was less related to stressful life events than ADHD-I was, and those with ADHD-H had a significantly lower risk for major depression (Friedrichs, Igl, Larsson, & Larsson, 2012). Moreover, one study demonstrated that inattention, but not hyperactivity, was associated with certain dependent stressors, including increased peer and parent-child-problems, and heightened depression symptoms (Humphreys et al., 2013). This suggests that inattentive symptoms of ADHD may be more predictive of dependent stress and depression symptoms compared with hyperactive/impulsive symptoms.

Current study

In summary, ADHD is associated with dependent stressors and dependent stress predicts depressive symptoms, but no studies have tested whether overall dependent stress mediates the association between ADHD and depression. Additionally, despite evidence that inattentive symptoms are more related to stress and depression risk, no studies have tested this relation examining ADHD-I and ADHD-H separately. Therefore, the current study tests the hypothesis that there will be a significant longitudinal association between ADHD symptoms and depression that is mediated by dependent stress (Figure 1), such that over time, ADHD symptoms would

increase dependent stress frequency, and dependent stress will increase depression symptoms. We tested this longitudinally with three time points across a college semester to determine how baseline ADHD predicts stress in the weeks leading up to final exams, and how this predicts depression symptoms immediately following final exams, as this is a high-risk period (See Figure 1). We also tested this model cross-sectionally with a longer and more detailed measure of dependent stressful life events that was not collected at time 2 of the longitudinal design (See Figure 2). Additionally, we tested separate models with inattentive symptoms and hyperactive/impulsive symptoms each predicting dependent stress and depression to compare the effects. We hypothesized that ADHD-I would more strongly predict dependent stress and depression compared with ADHD-H symptoms (See Figures 3 4).

Methods

Participants

Participants were 153 undergraduate students at Brandeis University in Waltham, Massachusetts, recruited via flyers posted around campus and postings in Brandeis class Facebook groups. All participants were 18-23 years old, in order to capture the experience of adolescence and early adulthood, and were fluent in English, as the questionnaires were in English. There were no other exclusion criteria. This sample had a mean age of 19 and 69.28% identified as female while 25.49% identified as male, and 5.23% as other. All participants gave written informed consent. Retention in the study was high: 142 participants completed all three time-points of the study while one participant missed time-point 2, five participants missed time-point 3, and four participants missed both time-points 2 and 3. The Brandeis University IRB approved the study procedures.

Procedure

Data were collected as part of a larger longitudinal study investigating executive functioning, coping, and psychopathology. Each participant was followed over the course of a semester in order to capture ADHD symptoms at baseline (time-point 1), stress in the four weeks leading up to final exams (time-point 2), and depression during and immediately following finals (time-point 3) as this is a high-risk period. Stress was averaged across the four weeks leading up to finals to capture total stress generation during this high-stress period. All measures were also collected at time-point 1 for cross-sectional analysis.

Measures

Questionnaires were administered online through the secure Qualtrics system, using a unique access code as the only participant identifier.

Adult ADHD Self-Report Scale (ASRS) (Kessler et al., 2005). The ASRS is a continuous measure of ADHD symptoms. The questionnaire contains 18 items that assess how frequently the participant experienced symptoms over the last 6 months. Participants respond on a 5-point Likert scale ranging from (0) never to (4) very often. Two subscales of the ASRS indicate symptoms associated with ADHD-I (e.g. "How often do you have trouble remembering appointments or obligations?") or ADHD-H (e.g. "How often do you feel restless or fidgety?"). Each subscale contains nine items reflecting the DSM-IV-TR criteria for each subtype. Higher scores indicate higher ADHD symptoms as well as overall ADHD severity. The ASRS is a reliable and valid scale for evaluating ADHD in adults and shows a high internal consistency (Cronbach's alpha 0.88) and high concurrent validity (ICC = 0.84) (Adler et al., 2006). Internal consistency in the current study was good ($\alpha = .823$).

The Adolescent/Adult Life Events Questionnaire Revised (ALEQ-R). This study uses a revised version of the ALEQ (Abramson & Hankin, 2002), the ALEQ-R (Fassett-Carman,

Hankin, & Snyder, 2018). The ALEQ-R assesses the frequency of independent and dependent stressful life events typical of adolescents and emerging adults on a 5-point Likert scale (0=never; 1=rarely; 2=sometimes, 3=frequently; 4=always). The full ALEQ-R used at time-point 1 asks participants to reflect on the past six months and contains 63 negative life events. The questionnaire was modified to assess 11 negative life events occurring over the past week for use in the four weekly questionnaires to decrease participant burden. Two researchers (BLH and HRS) independently coded each item in the original ALEQ-R as independent, dependent, or neither (ambiguous or mixed). Inter-rater agreement was high (kappa = 0.83); for each of the seven items where the two raters disagreed, one researcher labelled it as neither while the other classified it as dependent or independent. In these cases, the more conservative label of neither was used, resulting in a total of 26 independent (e.g., "You were sick or had a medical procedure and it interfered with work/college, or your activities"), 23 dependent (e.g., "Problems or arguments with teachers/professors or a boss/supervisor"), and 16 neither items (Fassett-Carman et al., 2018). Of the 11 items in the short-form ALEQ-R, nine items were classified as dependent stressors and two items were classified as independent stressors. For the purpose of the current study, only dependent stressor events were assessed. The cross-sectional time-point 1 analysis used the average of dependent stress frequency over the past six months in the 63-item questionnaire collected at baseline. For the longitudinal analysis, dependent stress frequencies during each of the four weeks leading up to finals, measured using the short-form questionnaire, were averaged to obtain a composite score for time-point 2. Dependent stress frequency, as measured by the ALEQ-R has been related to depression in prior research (Fassett-Carman et al., 2018).

Beck Depression Inventory (BDI-II) (Beck, Steer, & Brown, 1996). The BDI-II is a self-report measure of depression that assesses 21 items within the past week. Each question is answered on a 4-point Likert scale ranging from 0-3, with higher scores indicating more severe symptoms (e.g. (0) I do not feel sad to (3) I am so sad and unhappy that I cannot stand it). The BDI-II is a reliable measure of depression symptoms with Cronbach's α ranging from .92 to .93 and test-retest reliability at .93 (Beck, Steer, & Brown, 1996). This is also a valid measure of depression symptoms with good convergent validity with other measures of depression (r = .71-.93). Internal consistency in the current study was good (α = .898).

Data Analysis

Mediation analyses were run in Mplus with estimator FIML to handle missing data and 10,000 bootstrap iterations for robust estimation. Monte Carlo power analyses with 10,000 repetitions demonstrated that we have adequate power above 0.8 to detect medium effect sizes of β = 0.3 for each path. Scores that were three standard deviations away from the mean were excluded from analysis as outliers; three scores were excluded on the ALEQ and two on the BDI.

We tested the effect of ADHD symptoms at time-point 1 on dependent stress at time-point 2 and the effect of dependent stress at time-point 2 on depression at time-point 3. We also tested the effect of ADHD symptoms at time-point 1 on depression at time-point 3 with dependent stress at time-point 2 as a mediator. Questionnaires completed at time-point 1 were used for the cross-sectional analysis. We tested the effect of ADHD symptoms on dependent stress as well as dependent stress on depression. We also investigated dependent stress as a mediator in the relation between ADHD symptoms and depression.

Results

Longitudinal Model

Descriptive Statistics and Bivariate Correlations. Descriptive statistics are reported in Table 1 and bivariate correlations in Table 2. At time-point 1, ADHD and dependent stress (r = .343, $p < .01$) and ADHD and depression (r = .368, $p < .01$) were significantly correlated. Additionally, dependent stress significantly correlated with depression at time-point 1 (r = .527, $p < .01$).

Longitudinally, depression at time-point 1 significantly correlated with depression at time-point 3 (r = .397, $p < .01$) and dependent stress at time-point 1 significantly correlated with dependent stress at time-point 2 (r = .515, $p < .01$). ADHD at time-point 1 significantly correlated with dependent stress at time-point 2 (r = .333, $p < .01$) and dependent stress at time-point 2 significantly correlated with depression at time-point 3 (r =.392, $p < .01$). Independent samples t-tests indicate that women scored significantly higher on depression symptoms at time-point 1 than men, $t(142) = -2.30$, $p = 0.23$. No other measures had significant gender differences (see Table S2).

Longitudinal total effect model (Figure 1 and Table 3). This model tested the effects of ADHD at time-point 1 on depression at time-point 3, controlling for depression at time-point 1, age, and gender. Increased ADHD scores at time-point 1 significantly predicted increased depression scores at time-point 3 (β = .245, p = .004).

Longitudinal mediation path model (Figure 1 and Table 3). This model tested the effects of ADHD symptoms at time-point 1 on depression at time-point 3, mediated by dependent stress at time-point 2, controlling for depression and dependent stress at time-point 1 as well as age and gender. Higher ADHD scores at time-point 1 significantly predicted higher dependent stress at time-point 2, controlling for age, gender, and dependent stress at baseline (β = .183, p = .006). Increased dependent stress at time-point 2 significantly predicted increased

depression symptoms at time-point 3, (β = .262, p = .004). There was a significant indirect path from ADHD at time-point 1 to depression at time-point 3, mediated by dependent stress at time-point 2, (β = .048, p = .048). There was also a significant direct path from time-point 1 ADHD to time-point 3 depression (β = .178, p = .037). The results of the regression indicated that the variables in this model explained 27.5% of the variance in depression symptoms at time-point 3 (R^2 = .275). Similar patterns were found in the cross-sectional analysis at time-point 1 (see Table S1).

Exploratory Longitudinal Model by ADHD Subtype

ADHD subtype total effect models (Figure 2, Figure 3, and Table 4). These models tested the effects of ADHD-I and ADHD-H respectively at time-point 1 on depression at time-point 3, controlling for depression at time-point 1, age, and gender. Increased ADHD-I symptoms at time-point 1 significantly predicted increased depression scores at time-point 3, (β = .180, p = .037). Increased ADHD-H symptoms at time-point 1 also significantly predicted increased depression scores at time-point 3 (β = .210, p = .012).

ADHD subtype mediation path models (Figure 2, Figure 3, and Table 4). These models tested the effects of ADHD-I and ADHD-H respectively at time-point 1 on depression at time-point 3, mediated by dependent stress at time-point 2, controlling for depression, dependent stress, age, and gender at baseline. Higher ADHD-I symptoms at time-point 1 significantly predicted higher dependent stress at time-point 2 (β = .153, p = .038). Increased dependent stress at time-point 2 significantly predicted increased depression symptoms at time-point 3, (β = .284, p = .001). However, the indirect path from ADHD-I at time-point 1 to depression at time-point 3 mediated by dependent stress at time-point 2 did not reach significance (p = .078). The direct path from time-point 1 ADHD-I to time-point 3 depression was also not significant (p = .128).

Higher ADHD-H symptoms at time-point 1 significantly predicted higher dependent stress at time-point 2 (β = .161, p = .013). Increased dependent stress at time-point 2 significantly predicted increased depression symptoms at time-point 3, (β = .266, p = .003). However, the indirect path from ADHD-H at time-point 1 to depression at time-point 3 mediated by dependent stress at time-point 2 did not reach significance (β = .043, p = .062). There was a significant direct path from time-point 1 ADHD-H to time-point 3 depression (β = .167, p = .047). Wald tests indicated that there were no significant differences between the effects of ADHD-I and ADHD-H on dependent stress ($\chi 2$ (1) = 1.709, p =.191) or depression ($\chi 2$ (1) = .039, p = .844).

Discussion

Although previous research has identified that individuals with ADHD are at a higher risk for depression (Garnier-Dykstra, et al., 2011), and that stress generation may be a mediating mechanism, this has not been tested previously. Thus, the current study contributed a new, longitudinal, stress generation model. We hypothesized that higher levels of ADHD symptoms at time-point 1 would increase depression symptoms at time-point 3, mediated by increased dependent stress at time-point 2. Additionally, we predicted that ADHD-I symptoms would more strongly predict dependent stress and depression versus ADHD-H symptoms. As hypothesized, ADHD symptoms at time-point 1 significantly predicted increased depression symptoms at time-point 3, mediated by increased dependent stress at time-point 2. Contrary to our hypothesis, there was no significant difference between the effects of ADHD-I and ADHD-H on dependent stress and depression.

Longitudinal Model

These findings contribute to the growing body of knowledge on the relations between ADHD symptoms and dependent stress, as well as dependent stress and depression. ADHD

symptoms leading to stress generation over time is consistent with previous research (Beiter et al., 2015; Jackson & Farrugia, 1997). The primary characteristics of ADHD may bring on a cascade of difficult life events, ranging from trouble creating strong social relationships to retaining employment. Specifically, impaired attention, interpersonal skills, initiative, and task persistence may hinder academic as well as occupational functioning (Combs et al., 2015). Additionally, college-age adults with ADHD demonstrate significantly lower task performance in the areas of concentration, motivation, and information processing (Combs et al., 2015), which may lead to further generation of stressors (e.g., doing poorly on an exam).

Among individuals with ADHD, dependent stress generation may be especially troublesome. There are inherent stress response differences between those with and without ADHD (Combs et al., 2015; Lackschewitz, Hüther, & Kröner-Herwig, 2008). Adults with ADHD have elevated physiological stress responses and have a greater difficulty in recovering from elevated stress levels as measured by salivary cortisol (Hirvikoski et al., 2009). Coupled with the knowledge that ADHD increases dependent stressors for an individual, these stress response differences demonstrate that dependent stress generation among those with ADHD symptoms could be particularly problematic.

The significant relation between dependent stress and subsequent depression in this study is also well verified by previous research (Hammen, 2018; Kendler et al., 1999; Technow et al., 2018). This may be because perceiving that one played a role in causing a stressor for oneself could lead to depression symptoms such as feelings of low self-esteem, worthlessness, and hopelessness. This is consistent with research on cognitive styles which has identified that generally attributing stressful events as stemming at least in part from the self is associated with depression symptoms (Huang, 2015; Scallion & Cummings, 2018).

Longitudinal Model by ADHD Subtype

Contrary to our hypothesis, there were no significant differences between the effects of inattentive and hyperactive/impulsive symptoms on dependent stress and depression. Although this was not consistent with the majority of previous literature (Combs et al., 2012; Friedrichs et al., 2012; Humphreys et al., 2013, Willcutt et al., 2012), our findings are consistent with a smaller subset of literature which found no significant difference between ADHD subtype symptoms (Eiraldi, Power, & Nezu, 1997; Nelson & Gregg, 2012; Power, Costigan, Eiraldi, & Leff, 2004). It may be that different ADHD symptoms are associated with different types of dependent stressors. A specific study found that inattention, but not hyperactivity/impulsivity, was related to specific dependent stressors such as peer and parent-child problems (Humphreys et al., 2013). While inattention may be related to these specific stressors, overall dependent stress may not be reliant on a specific ADHD symptom type. Additionally, a review article identified that hyperactivity is strongly associated with negative peer regard while inattention is strongly associated with social isolation (Willcutt et al., 2012). Thus, the different ADHD symptom types may lead equally to stress generation, however, via different mechanisms

Limitations

This study has several limitations that are important to note. First, this study used all self-report questionnaires. This may have led to recall or reporting bias and participants misreporting symptoms or the frequency of their symptoms. However, self-report measures have frequently been used to confirm ADHD symptomatology in college-aged students and previous research has shown that college-age participants are reliable reporters of ADHD symptoms (Gray, Woltering, Mawjee, & Tannock, 2014; Salla, Galéra, Guichard, Tzourio, & Michel, 2017; Silverstein et al.,

2014). Additionally, the BDI has been well validated across studies and has good agreement with clinician-rated symptoms (Beck, et al., 1996).

The use of self-report measures also limits the findings to symptomatology rather than clinical diagnoses. Future research could verify self-report questionnaires with clinical interviews in order to get better symptom-level information and data on the diagnostic composition of the sample. Moreover, future research may recruit participants from a referred sample rather than a community sample to test these relations at more severe levels of ADHD and depression.

Additionally, the gender distribution of the sample in this study was heavily weighted towards female participants. Although symptoms of ADHD are not gender specific, women are more likely to report both dependent stress and depression symptoms than men are (Hankin, Mermelstein, & Roesch, 2007; Rucklidge, 2008). Additionally, the probability of a child with ADHD developing major depressive disorder is higher for women (Jerrell, McIntyre, & Park, 2015). Consequently, the gender distribution may have caused the data to be more indicative of mechanisms in females rather than the general population. Future research should assess gender as a potential moderator to determine whether this mediation path differs for males and females. Additional studies may also aim to recruit a more diverse sample of participants so they may more accurately describe the population at large.

Finally, the results of the study may not be generalizable to other samples, as it was comprised solely of Brandeis University undergraduate students aged 18-23. This study was specifically focused on college-aged students because this is a high-risk period for the onset and recurrence of depression. However, it is uncertain whether the results would generalize to other age groups such as children, older adults, or even non-student populations. Brandeis University

is also a small, liberal arts school that attracts a certain type of student. These students are extremely academically focused but also very aware of mental health on campus. This may lead to more stress induced around academic issues but also higher reporting of psychopathological symptoms. Despite these limitations, the current study still makes an important contribution to scientific literature due to the limited prior research linking ADHD, dependent stress, and depression in a longitudinal model.

Future Directions and Implications

The results of this study suggest a number of future directions aimed at continuing to understand the relations between ADHD symptoms, dependent stress, and depression. One such future direction may be to examine dependent stress severity as a potential mediator in the relation between ADHD symptoms and subsequent depression. The current model assessed dependent stress frequency; however, previous literature has demonstrated that ADHD symptoms are positively associated with perceived stress as well (Combs et al., 2015; Hirvikoski et al., 2009; Salla et al., 2017). Further, subjective perceptions of stress can trigger depression following even a minor or imagined event (Hammen, 2018). This indicates that perceptions of stress such as the severity of a given stressor may be an important mediating factor in the relation between ADHD and depression in addition to stress frequency.

Additionally, the current research has implications for interventions aimed at treating or preventing depression in those with ADHD symptoms. The results indicate that disrupting the link between ADHD symptoms and dependent stress could prevent subsequent depression symptoms. Therefore, interventions that combat dependent stress-generative tendencies in those with ADHD should be explored. One such treatment for adults with ADHD is structured skills training (Hirvikoski et al., 2011). This group behavioral intervention targets themes such as

"impulse control" and "chaos and control" and trains participants to use mindfulness and behavioral analysis as strategies for improved impulse control as well as organizational skills, among others. Thus, targeting deficits associated with ADHD through skill building exercises may reduce the incidence of dependent stressors and decrease the risk for developing subsequent depression. Disrupting the link between dependent stressors and depression may also be an important point of intervention for individuals with ADHD. The Behaviorally Enhancing Adolescents' Mood (BEAM) depression prevention program for adolescents with ADHD targets emotion regulation deficits (Meinzer, Hartley, Hoogesteyn, & Pettit, 2018). By providing training on how to regulate responses to stressors, this intervention decreases depression symptoms. Future interventions should aim to combine such therapies in order to teach skills that will prevent dependent stressors from emerging but in the case that they do, will provide training on how to regulate responses to these stressors to prevent future depression.

Conclusions

In sum, the current study demonstrates that dependent stress is an important mediating factor in the longitudinal relation between ADHD symptoms and depression. This study created a cohesive model of ADHD, dependent stress, and depression, as previous research had not examined all factors in a singular model. In the future, it will be important to study how other aspects of dependent stress such as stress severity function in the mediation model and how knowledge from this study can be incorporated into clinical interventions.

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Table 1. Descriptive Statistics

Measure	n	Mean	SD	Skew	Kurtosis
T1 ADHD-I	153	2.52	1.55	0.12	-0.80
T1 ADHD-H	153	3.66	2.61	0.71	-0.26
T1 ADHD Overall	153	6.18	3.68	0.41	-0.48
T1 Dependent Stress	151	13.53	8.38	0.73	-0.02
T1 Depression	151	8.48	6.89	1.00	0.52
T2 Dependent Stress	148	11.33	8.86	1.11	0.85
T3 Depression	143	8.93	8.75	1.81	5.13
Age	153	19.22	1.20	0.78	-0.27

Note. T1, time-point 1; T2, time-point 2; T3, time-point 3.

Table 2. Bi-variate Correlations

Measure	1	2	3	4	5	6	7	8
1. ADHD-I	-							
2. ADHD-H	.539**	-						
3. T1 ADHD Overall	.802**	.935**	-					
4. T1 Dependent Stress	.208*	.358**	.343**	-				
5. T1 Depression	.313**	.332**	.368**	.527**	-			
6. T2 Dependent Stress	.245**	.324**	.333**	.515**	.311**	-		
7. T3 Depression	.248**	.337**	.341**	.296**	.397**	.392**	-	
8. Age	-.030	-.099	-.083	-.042	-.157	.025	.029	-

Note. T1, time-point 1; T2, time-point 2; T3, time-point 3. * $p < .05$. ** $p < .01$

Table 3. Regression Table

Outcome Variable	Predictor	*β*	SE	Est./SE	*p*	R^2
		Longitudinal Total Effect Model				
T3 Depression	T1 ADHD (c)	.245	.085	2.896	.004**	.220
	T1 Depression	.299	.090	3.321	.001**	
	Age	.099	.078	1.281	.200	
	Gender	.112	.074	1.513	.130	
		Longitudinal Mediation Path Model				
T2 Dependent Stress	T1 ADHD (a)	.183	.067	2.724	.006**	.297
	T1 Dependent Stress	.444	.077	5.789	<.001**	
	Age	.052	.078	.675	.500	
	Gender	.074	.061	1.198	.231	
T3 Depression	T1 ADHD (direct effect c')	.178	.085	2.085	.037*	.275
	T1 Depression	.240	.090	2.660	.008**	
	T2 Dependent Stress (b)	.262	.091	2.896	.004**	
	Age	.074	.073	1.019	.308	
	Gender	.088	.077	1.138	.255	
		Longitudinal Mediation Path Model Indirect Effect				
T1 ADHD → T2 Dependent Stress → T3 Depression		.048	.024	1.979	.048*	

Note. T1, time-point 1; T2, time-point 2; T3, time-point 3. * $p < .05$. ** $p < .01$

Table 4. Inattentive ADHD Symptom Regression Table

Outcome Variable	Predictor	β	SE	Est./SE	p	R^2
		Longitudinal Total Effect Model				
T3 Depression	T1 ADHD-I (c)	.180	.086	2.082	.037*	.197
	T1 Depression	.330	.096	3.447	.001**	
	Age	.091	.079	1.159	.246	
	Gender	.112	.072	1.553	.121	
		Longitudinal Mediation Path Model				
T2 Dependent Stress	T1 ADHD-I (a)	.153	.074	2.072	.038*	.290
	T1 Dependent Stress	.475	.074	6.411	<.001**	
	Age	.044	.077	.569	.569	
	Gender	.076	.061	1.246	.213	
T3 Depression	T1 ADHD- I (direct effect c')	.128	.084	1.524	.128	.263
	T2 Dependent Stress (b)	.284	.090	3.175	.001**	
	T1 Depression	.257	.094	2.717	.007**	
	Age	.067	.074	0.901	.367	
	Gender	.088	.075	1.171	.242	
		Longitudinal Mediation Path Model Indirect Effects				
T1 ADHD-I → T2 Dependent Stress → T3 Depression		.044	.025	1.763	.078	

Note. T1, time-point 1; T2, time-point 2; T3, time-point 3. * $p < .05$. ** $p < .01$

Table 5. Hyperactive/Impulsive ADHD Symptom Regression Table

Outcome Variable	Predictor	*β*	SE	Est./SE	*p*	R^2
Longitudinal Total Effect Model						
T3 Depression	T1 ADHD-H (c)	.210	.083	2.510	.012*	.187
	T1 Depression	.297	.091	3.244	.001**	
	Age	.104	.081	1.281	.200	
	Gender	.089	.076	1.177	.239	
Longitudinal Mediation Path Model						
T2 Dependent Stress	T1 ADHD-H (a)	.161	.065	2.475	.013*	.290
	T1 Dependent Stress	.451	.076	5.899	<.001**	
	Age	.054	.078	.683	.494	
	Gender	.062	.062	.995	.320	
T3 Depression	T1 ADHD-H (direct effect c')	.167	.084	1.982	.047*	.271
	T2 Dependent Stress (b)	.266	.089	2.978	.003**	
	T1 Depression	.251	.088	2.858	.004**	
	Age	.078	.072	1.081	.280	
	Gender	.077	.079	.971	.332	
Longitudinal Mediation Path Model Indirect Effects						
T1 ADHD-H → T2 Dependent Stress → T3 Depression		.043	.023	1.867	.062	

Note. T1, time-point 1; T2, time-point 2; T3, time-point 3. * $p < .05$. ** $p < .01$

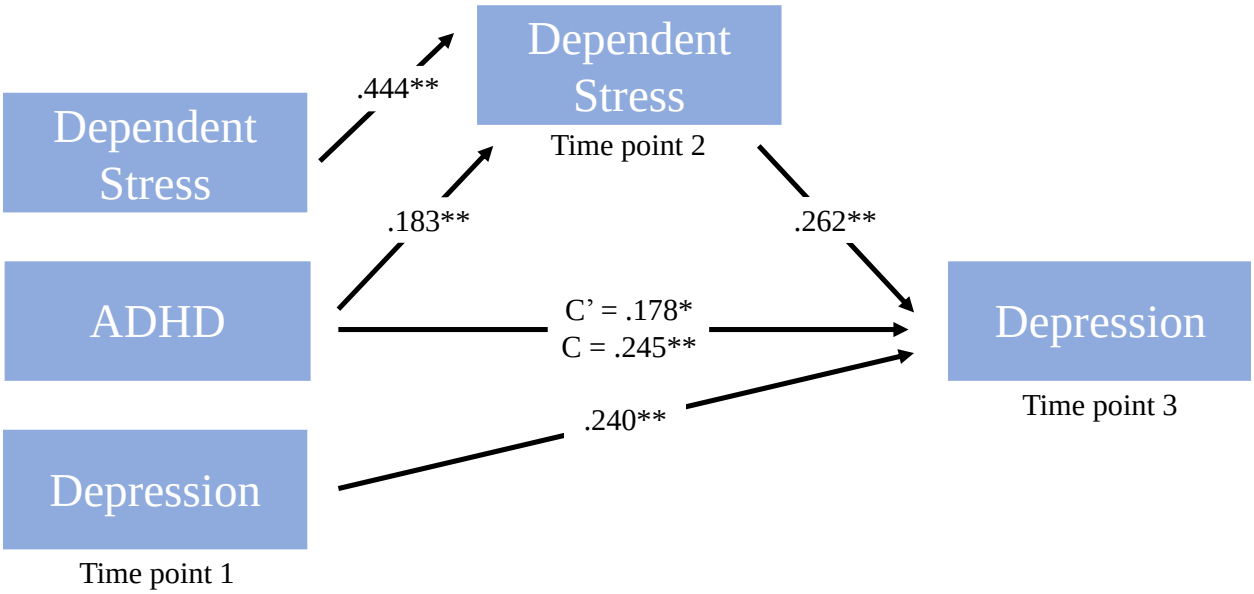

Figure 1. Path models with ADHD at time-point 1, dependent stress at time-point 2, and depression at time-point 3. ADHD symptoms significantly prospectively predicts depression via the dependent stress indirect pathway. * $p <$ .05. ** $p <$ .01.

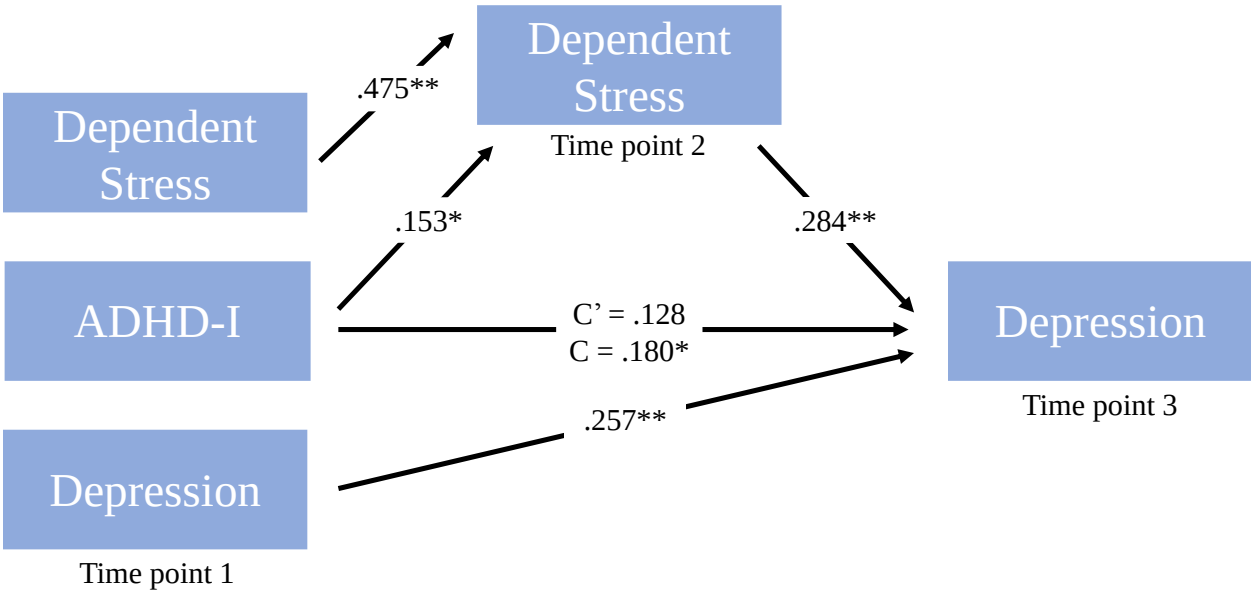

Figure 2. Path models with inattentive ADHD symptoms at time-point 1, dependent stress at time-point 2, and depression at time-point 3. Inattentive ADHD symptoms do not significantly predict depression via the dependent stress indirect pathway. * $p < .05$. ** $p < .01$.

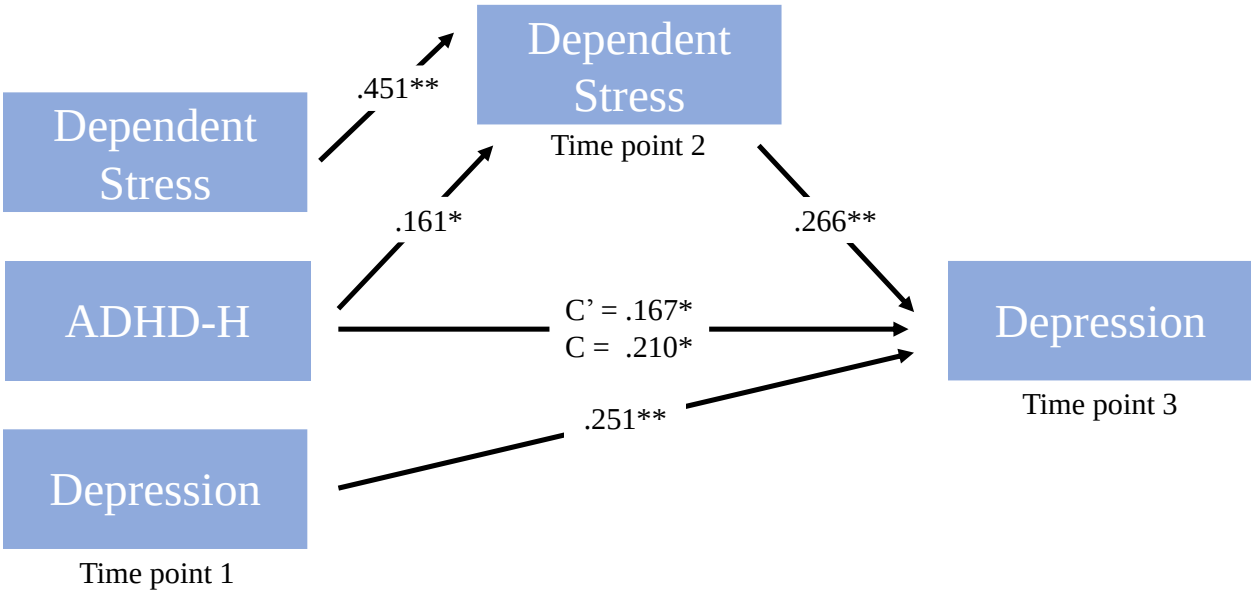

Figure 3. Path models with hyperactive/impulsive ADHD symptoms at time-point 1, dependent stress at time-point 2, and depression at time-point 3. Hyperactive/impulsive ADHD symptoms significantly prospectively predict depression via the dependent stress indirect pathway. * $p < .05$. ** $p < .01$.

Supplemental Materials

Cross Sectional Model

Cross sectional total effect model (Table S1 and Figure S1). This model tested the effects of ADHD on depression, controlling for age and gender. Higher ADHD symptom scores significantly predicted higher depression scores at time-point 1, (β = .361, p < .001).

Cross sectional mediation path model (Table S1 and Figure S1). This model tested the effects of ADHD symptoms on depression, mediated by dependent stress, while controlling for age and gender. Higher ADHD scores at baseline significantly predicted higher dependent stress scores (β = .378, p < .001) and higher dependent stress scores significantly predicted higher depression scores (β = .462, p < .001). There was a significant indirect path from ADHD to depression mediated by dependent stress, (β = .175, p < .001). There was also a significant direct path from ADHD to depression with dependent stress as a mediator, (β = .183, p = .039). The results of the regression indicated that the variables in this model explained 31% of the variance in depression symptoms (R^2 = .310).

Table S1. Regression Table

Outcome Variable	Predictor	β	SE	Est./SE	p	R^2
		Total Effect Model				
Depression	ADHD (c)	.378	.066	5.725	<.001**	.140
	Age	-.114	.076	-1.498	.134	
	Gender	.211	.068	3.078	.002**	
		Mediation Path Model				
Dependent Stress	ADHD (a)	.358	.074	4.829	<.001**	.199
	Age	-.006	.075	-.080	.936	
	Gender	.128	.082	1.559	.119	
Depression	ADHD (direct effect c')	.224	.070	3.191	.001**	.310
	Dependent Stress (b)	.430	.071	6.035	<.001**	
	Age	-.112	.069	-1.625	.104	
	Gender	.157	.059	2.661	.008**	
		Mediation Path Model Indirect Effect				
ADHD → Dependent Stress → Depression		.154	.040	3.833	<.001**	

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

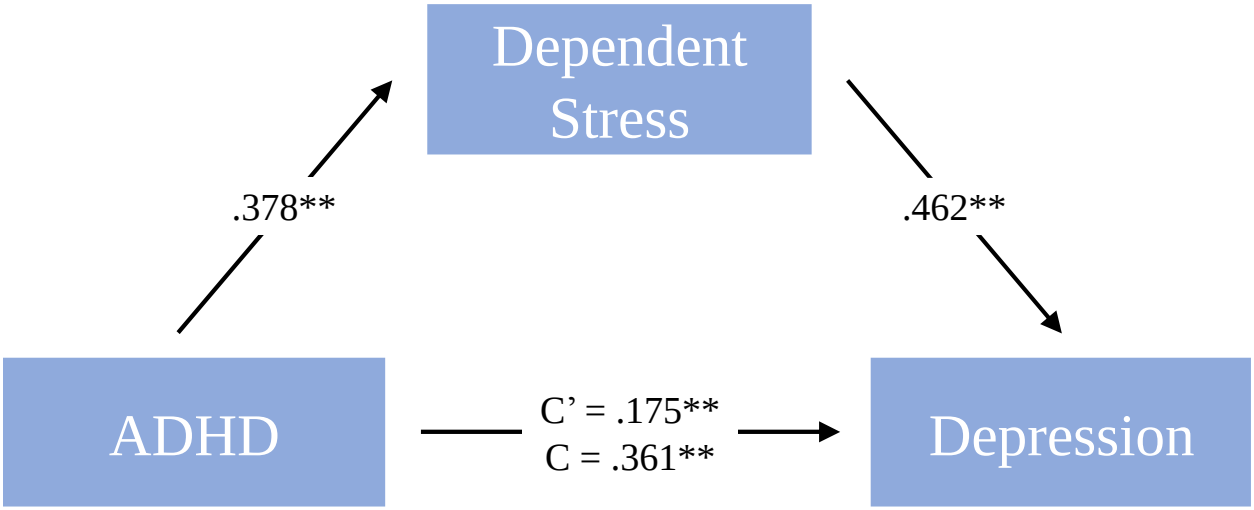

Figure S1. Path models with ADHD, dependent stress, and depression at baseline. ADHD symptoms predict depression via the dependent stress indirect pathway. ** $p < .01$.

Table S2. T-Test Gender Differences

		n	Mean	SD	t	df	p
T1 ADHD-I	Male	39	2.74	1.57	1.384	143	.169
	Female	106	2.35	1.51			
T1 ADHD-H	Male	39	3.59	2.64	0.333	143	.740
	Female	106	3.43	2.45			
T1 ADHD Overall	Male	39	6.33	3.76	0.832	143	.407
	Female	106	5.78	3.45			
T1 Dependent Stress	Male	39	12.38	7.89	-1.126	141	.262
	Female	104	14.16	8.60			
T1 Depression	Male	39	6.33	5.03	-2.299	142	.023*
	Female	105	9.22	7.21			
T2 Dependent Stress	Male	39	10.11	6.63	-1.086	138	.279
	Female	101	11.95	9.75			
T3 Depression	Male	38	6.50	6.10	-1.916	133	.057
	Female	97	9.22	7.85			

Note. T1, time-point 1; T2, time-point 2; T3, time-point 3. * $p < .05$.