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Temperament Traits Mark Liability for Coexisting Psychiatric Symptoms in Children with Elevated ADHD Symptoms

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Abstract

Objective: Among children with attention deficit hyperactivity disorder (ADHD), coexisting psychiatric disorders are common and associated with greater impairment and symptom persistence. Given that temperament traits are easily measured, developmentally stable, and variable among youth with ADHD, temperament profiles may be clinically useful for predicting liability for coexisting psychiatric symptoms in this population. **Methods:** Eighty-three children with ADHD symptoms participated. Caregivers rated their child's surgency, negative emotionality, and effortful control, as well as severity of internalizing and externalizing psychiatric symptoms. Hierarchical linear regressions were conducted to estimate associations between temperament traits and psychiatric symptoms, controlling for severity of ADHD. **Results:** Temperament ratings explained significant variance in psychiatric symptoms above and beyond ADHD symptoms alone. Symptoms of each coexisting psychiatric disorder was associated with a distinct temperament and ADHD symptom profile. **Conclusion:** Temperament ratings appear to have clinical utility for predicting coexisting psychiatric symptoms in children with elevated ADHD symptoms.

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Temperament Traits Mark Liability for Coexisting Psychiatric Symptoms in Children with Elevated ADHD Symptoms

Attention Deficit Hyperactivity Disorder (ADHD) is a common neurodevelopmental disorder characterized by persistent and impairing symptoms of inattention and/or hyperactivity-impulsivity across settings (American Psychiatric Association, 2013). Children with ADHD have high rates of coexisting disorders, including anxiety, depression, oppositional defiant disorder and conduct disorder (Costello et al., 2003; Spencer et al., 2007). Coexisting psychiatric conditions are associated with greater socioemotional, educational and occupational impairment and persistence of ADHD symptoms (Biederman et al., 2011; Larson et al., 2011). Adjunct behavioral and pharmacological interventions can be effective treatments for internalizing and externalizing symptoms in children with ADHD (Velő et al., 2019); however, currently clinicians are unable to predict risk for coexisting psychopathology in this population. Precision medicine care for children with ADHD will be advanced by proactive identification and treatment of individuals who are at increased risk of developing internalizing and externalizing disorders (Barbaresi et al., 2020). In the current study, we test whether temperament traits explain individual differences in coexisting psychiatric symptoms and may thereby serve as a useful clinical tool for predicting psychiatric outcomes among children with ADHD.

Coexisting Psychiatric Symptoms in ADHD

Externalizing symptoms, such as aggression, defiance, and oppositionality, are particularly common among children with ADHD, across population and clinical samples (Biederman et al., 1991; Jensen et al., 1997; Lahey et al., 1999). Children with coexisting ADHD have increased persistence of externalizing disorders relative to non-ADHD peers (August et al., 1999). Compared to individuals with ADHD alone, those with ADHD and oppositional defiant

disorder (ODD) or conduct disorder (CD) have poorer social functioning (Ostrander et al., 2006), higher rates of delinquency (Sibley et al., 2011), and poorer adult outcomes (Burke et al., 2014). Internalizing symptoms are also common, with approximately one-quarter of children with ADHD displaying coexisting anxiety and/or mood disorders, which are likewise associated with greater functional impairment compared to ADHD alone (Reid et al., 2015). Internalizing symptoms may exacerbate ADHD symptoms, leading to poorer quality of life and psychosocial difficulties (Bowen et al., 2008). Among individuals with depression, a comorbid ADHD diagnosis appears to prolong a depressive episode and increase the likelihood of recurrence, hospitalization and suicidality (Biederman et al., 2008; James et al., 2004; Rohde et al., 2001).

Genomic studies indicate at least partially overlapping genetic etiology for ADHD and coexisting psychopathology, particularly externalizing (Brikell et al., 2018; Hamshere et al., 2013) and depressive symptoms (Brikell et al., 2018). Nigg and colleagues (2019) reported that ADHD polygenic risk scores were higher among children with both ADHD and emotion dysregulation relative to those with ADHD alone, suggesting a continuum wherein ADHD with coexisting psychopathology represents the extreme tail of genetic liability for ADHD. However, in that study, polygenic risk scores did not differentiate among subdomains of emotion dysregulation. Thus, the degree to which the etiology of ADHD and coexisting psychopathology is shared remains unknown.

Temperament and Psychopathology

Temperament is commonly defined as constitutionally based, individual differences in the domains of surgency, negative emotionality, and effortful control (Rothbart, Ahadi, et al., 2000; Rothbart & Derryberry, 1981) that are at least moderately stable across childhood and adolescence (Kopala-Sibley et al., 2018; Rothbart, Derryberry, et al., 2000). Temperament can

be easily measured via parent-report as early as infancy (Gartstein & Rothbart, 2003). Twin and molecular genomic studies indicate a genetic etiology for temperament traits (Schmitz et al., 1996; Zwir et al., 2018), further supporting their biological basis and potential to be evaluated early in life. Altogether, temperament is a promising measure of risk and resilience among clinical populations.

ADHD, which begins in childhood, has been conceptualized as a combination of extreme temperament traits or a particularly maladaptive temperament (Martel et al., 2009; Sonuga-Barke, 2005). However, children with ADHD show a range of temperament profiles, including those characterized by high surgency (i.e. positive affect and approach) and high negative emotionality (i.e., irritability and sadness) (Karalunas et al., 2014; Karalunas et al., 2019; Nigg et al., 2004). Temperament profiles among youth with ADHD are associated with individual differences in ADHD symptom severity, maladaptive behaviors, and personality traits (De Pauw & Mervielde, 2011; Salgado et al., 2009; Yoo et al., 2006), and may map onto individual differences in neurobiology (Karalunas et al., 2014). Moreover, preliminary longitudinal research suggests children with ADHD and an "irritable" subtype (characterized by high negative affect and low effortful control) are at increased risk for coexisting psychiatric disorders at one-year (Karalunas et al., 2014) and two-year (Karalunas et al., 2019) follow ups.

Studies of longitudinal associations between temperament traits and internalizing and externalizing psychopathology, independent of ADHD, suggest moderate specificity. High surgency and activity level in preschoolers is predictive of ODD and CD in later childhood and adolescence (Forbes et al., 2017; Stringaris et al., 2010). In contrast, extreme low surgency in young children predicts high internalizing symptoms in adolescence (Forbes et al., 2017; Leve et al., 2005).

Negative emotionality in early development appears to be a risk factor for later psychiatric dysregulation, broadly. Some specificity has been reported between childhood negative emotionality and later internalizing (Bould et al., 2014; Gilliom et al., 2002) or externalizing (Eisenberg et al., 2009) symptoms. However, the extant literature generally indicates both internalizing and externalizing symptoms are predicted by high levels of negative emotions in childhood (Eisenberg et al., 2005; Forbes et al., 2017; Lemery et al., 2002).

In contrast, high effortful control, which is defined by self-regulation across behaviors and emotions (Rothbart & Bates, 2007), may be a protective factor against psychopathology. Children without clinical psychiatric symptoms have high levels of effortful control (Eisenberg et al., 2009), while low effortful control in childhood is associated with greater externalizing symptoms and ADHD severity both concurrently and prospectively (Eisenberg et al., 2009; Forbes et al., 2017; Kochanska & Knaack, 2003; Olson et al., 2005).

Current Study

The current study tests the hypothesis that parent-reported temperament traits, specifically surgency, negative emotionality (NE), and effortful control (EC), explain variance in coexisting psychiatric symptoms among school-aged children with elevated ADHD symptoms. Specifically, based on our review of the extant literature, we hypothesize that variance in both internalizing and externalizing symptom domains (anxiety, depression, ODD, CD) will be explained by high NE, while variance in externalizing symptoms (ODD, CD) will additionally be explained by high surgency and low EC. Given that ADHD is itself a risk factor for adverse developmental trajectories and adult psychopathology, we propose that measurement of temperament in this population will be clinically useful if it explains variance in psychiatric symptoms above and beyond what is accounted for by severity of ADHD symptom domains.

Method

Procedures

One-hundred-four school-aged children with a historical diagnosis of ADHD were recruited via flyers, clinic referrals, and community outreach in Seattle and the greater Puget Sound area. Of these, 92 met inclusion criteria for a research diagnosis of ADHD. Recruitment exclusion criteria were a diagnosis of autism spectrum disorder, known genetic syndrome, intellectual disability, perinatal trauma (e.g., < 32 weeks gestation, intracranial hemorrhage, need for resuscitation), prenatal exposure to substances, history of seizures, or colorblindness. Study procedures involved a single, three-hour visit to a university laboratory during which participants completed cognitive and neurophysiological testing and a caregiver completed questionnaires about their child's temperament, medical history, ADHD symptoms, and psychiatric symptoms. Caregivers of children currently taking psychotropic medications for ADHD were instructed to rate the child's temperament and behavior when off medications. Primary caregivers consented their child to participate; children provided verbal and written assent. All procedures were approved by the University of Washington Institutional Review Board.

Participant Characteristics

The final sample of participants included 83 (32.5% female) children, ages 7 to 11 years old (M = 9.22, SD = 1.38), 60% of whom were prescribed stimulant and/or other medications. Participants recruited to the larger study were excluded from the current analyses due to suspicion of autism (n = 3); full scale IQ < 80 (n = 2); nonindependence of data due to sibling enrollment (n = 2); or more than 15% of items were skipped by the caregiver on the temperament questionnaire (n = 2). Participants were predominately Caucasian (62.7%). Primary caregiver education levels included high school or equivalent (7.2%), some college (10.8%), 2-year college

(1.2%), trade school (1.2%), 4-year college (33.7%), master's degree (34.9%), or doctorate degree (10.8 %).

Measures

ADHD Symptoms

Current severity of ADHD symptoms was measured using caregiver report on the DSM-5 ADHD Scale on the Child Behavior Checklist 6-18 (CBCL; Achenbach, 2014) and the Strengths and Weakness of ADHD and Normal Behavior (SWAN) rating scale (Lakes et al., 2011; Swanson et al., 2012). The CBCL is a widely used measure that produces an age- and sex-normed DSM-5 ADHD T-score, which has mean = 50, standard deviation = 10. The SWAN measures adaptive and maladaptive behaviors associated with the 18 DSM-5 ADHD symptoms using a balanced, 7-point Likert scale, with anchors ranging from 7=*far below,* to 1=*far above* relative to same-aged peers. The SWAN has demonstrated strong internal and external validity (Arnett et al., 2013; Lakes et al., 2011). Symptoms marked "*below*" and "*far below*" were considered to be in the clinical range. Mean inattention (IA) severity was calculated as the average rating on items 1-9, and mean hyperactivity/impulsivity (H/I) was calculated as the average rating on items 10-18.

Participants were characterized as meeting criteria for currently elevated ADHD symptoms and thus included in the current analyses if their caregiver endorsed at least six clinical symptoms in either IA or H/I domains of the SWAN (4.8%), caregiver ratings on the CBCL DSM-5 ADHD scale indicated symptom severity at least 1.5 standard deviations above the mean (i.e., T ≥ 65; 33.7%), or both (61.4 %). On average, participants had moderately high SWAN ADHD symptom counts (IA $M = 6.00$, $SD = 2.44$; H/I $M = 4.01$, $SD = 2.78$) and CBCL ADHD T-scores ($M = 69.66$, $SD = 6.84$).

Child Temperament

The Temperament in Middle Childhood Questionnaire -Third Edition (TMCQ; Simonds et al., 2007; Simonds & Rothbart, 2004) was used to measure dimensions of temperament and higher-order traits. The measure includes 157 items that derive 17 subscale dimensions of temperament, which in turn load onto three higher-order factors of Negative Emotionality (NE), Surgency, and effortful control (EC) (Simonds & Rothbart, 2009). Internal consistency of the higher order factors was good (EC $\alpha = .80$, Surgency $\alpha = .88$, NE $\alpha = .87$), consistent with previous research (Nystrom & Bengtsson, 2017; Simonds, 2006).

Emotional and Behavioral Concerns

CBCL DSM-5 oriented scales of anxiety, depression, ODD, and CD were used as dependent internalizing and externalizing variables in the regression analyses. In the current sample, age- and sex-standardized CBCL diagnostic scale scores ranged from average to clinically elevated for Anxiety ($M = 57.51$, $SD = 7.97$), Depression ($M = 62.73$, $SD = 7.81$), ODD ($M = 63.34$, $SD = 9.49$), and CD ($M = 60.99$, $SD = 9.74$). Because normative scores on the CBCL DSM-5 oriented scales correct for both age and sex differences and prior literature suggests inherent sex differences in temperament traits (Else-Quest et al., 2006), we calculated age-standardized (but not sex-corrected) CBCL scale values by regressing the raw scale scores on age and saving the standardized residuals for the regression analyses.

Statistical Analyses

All analyses were conducted in SPSS 26.0. In preliminary analyses, we tested for sex and age differences associated with temperament and psychiatric ratings, with a plan to control for any significant demographic effects in the regression models. Additionally, we examined bivariate Pearson correlations among independent and dependent variables. Next, a series of

hierarchical linear regressions were conducted with depression, anxiety, ODD, and CD as dependent variables and ADHD and temperament traits as independent variables. SWAN IA and H/I severities were entered as independent variables at step one, followed by temperament traits (Surgency, NE, EC) at stage two. Unstandardized beta coefficients are reported in the text. Standardized beta coefficients are reported in Tables 2 and 3.

Results

Preliminary analyses

T-tests revealed no sex differences in IA or H/I symptom severity (p's > .500), temperament traits (p's ≥ .057), or internalizing and externalizing symptoms (p's > .249). Likewise, bivariate Pearson correlations indicated no associations between age and IA or H/I symptom severity (p's > .560), temperament traits (p's > .088), or internalizing and externalizing symptoms (p's = 1.00).

Table 1 shows the results of bivariate correlations among temperament, psychiatric and ADHD symptom ratings. Notably, temperament domains were uncorrelated with one another, supporting independence of these higher-order factors. NE was positively correlated with all internalizing and externalizing psychiatric symptoms, but not ADHD symptom severity. Higher surgency was associated with reduced anxiety, but increased externalizing and H/I symptoms. Lower EC was correlated with increased externalizing and ADHD symptom severity.

INSERT TABLE 1 ABOUT HERE

Depression

Model 1, with depression as the dependent variable and IA and H/I as independent variables, was not statistically significant: $F(2,80) = 0.03$, $p = .967$, $R^2 = .00$. The addition of temperament traits as predictive variables in model 2 revealed a statistically significant model

with a moderate amount of variance explained: $F(5, 77) = 6.05$, $p < .001$, $\Delta R^2 = .28$. Depression was linearly associated with NE (B = 1.07, *SE*= .21, $p < .001$) and negatively associated with EC (B = -0.66, *SE* = .30, $p = .030$) but not related to surgency (B = -0.06, *SE* = .21, $p = .793$), over and above IA (B = -0.11, *SE* = .18, $p = .538$), and H/I (B = -0.12, *SE* = .15, $p = .447$).

Anxiety

In Model 1, ADHD symptoms accounted for a minority of variance in anxiety (F [2,80] = 6.32, $p = .003$, $R^2 = .14$), with significant linear associations between anxiety and both IA (B = 0.38, *SE* = .18, $p = .040$) and H/I (B = 0.30, *SE* = .14, $p = .039$). Model 2, with temperament variables added, accounted for additional variance: $F(5, 77) = 13.01$, $p < .001$, $\Delta R^2 = .32$. In the final model, anxiety was positively associated with NE (B = 1.06, *SE*= .19, $p < .001$), and negatively associated with surgency (B = -0.42, *SE*= .19, $p = .028$), but not related to EC (B = -0.18, *SE*= .26, $p = .483$). Associations with ADHD symptoms remained statistically significant for H/I (B = 0.30, *SE* = -.13, $p = .030$), but not IA (B = 0.24, *SE* = -.16, $p = .131$).

INSERT TABLE 2 ABOUT HERE

Oppositional Defiant Disorder

Model 1, with ADHD symptoms as independent variables, was statistically significant, (F[2,80] = 10.87, $p < .001$, $R^2 = .21$), driven primarily by a linear association between H/I and ODD: B = 0.64, *SE*= -.14, $p < .001$. Temperament predictors explained additional variance in ODD: $F(5, 77) = 12.67$, $p < .001$, $\Delta R^2 = .24$. ODD was positively associated with NE (B = 0.70, *SE*= .19, $p < .001$) and surgency (B = 0.44, *SE*= .19, $p = .020$) and negatively associated with EC (B = -.1.04, *SE* = .26, $p < .001$), over and above the association with H/I (B = 0.34, *SE* = .13, $p = .013$).

Conduct Disorder

The omnibus model with ADHD symptoms as independent variables and CD as the dependent variable approached statistical significance (F [2,80] = 3.09, p = .051, R^2 = .072), driven by a linear association with H/I (B = 0.37, *SE* = .15, p = .015). Model 2, with temperament traits added, was statistically significant: F (5,77) = 5.35, p = .001, ΔR^2 = .186. CD was positively associated with NE (B = 0.53, *SE* = .22, p = .017) and negatively associated with EC (B = -1.08, *SE* = .304, p = .001), but not related to surgency (B = 0.27, *SE* = .22, p = .215). H/I was no longer significantly associated with CD in Model 2 (B = 0.14, SE = .16, p = .357).

INSERT TABLE 3 ABOUT HERE

Discussion

Children with elevated ADHD symptoms are at high risk for developing coexisting psychiatric disorders. Critically, the extant literature indicates that coexisting psychopathology in this population is associated with increased psychosocial impairment that may exceed functional impacts of core ADHD symptoms. This challenges clinicians to proactively screen, identify and treat youth at risk for developing complex ADHD, preferably in early childhood (Barbaresi et al., 2020). In the current study, we report distinct temperament profiles associated with increased risk for symptoms of internalizing and externalizing symptoms among 83 school age children with high levels of ADHD symptoms. The combination of ADHD and temperament traits explained a substantial portion of the variance (25 – 46%) of coexisting psychopathology. Specifically, a profile of high NE and low EC was associated with depressive symptoms and CD; high NE and low surgency were associated with anxiety; and high NE, high surgency, and low EC explained variance in ODD. We propose that given the likely biological etiology of temperament, evidence for stability of temperament traits from infancy through adolescence, and

ease of measurement, parent-rated temperament traits may be useful clinical tools for early identification and prediction of psychiatric liability in this highly vulnerable population.

Heightened NE has repeatedly been cited as a risk factor for general psychopathology (Tackett et al., 2013). Consistent with this research, NE explained independent variance in all parent-reported internalizing and externalizing symptom domains in the current study, with moderate to large effect sizes. NE was not correlated with either IA or HI, and the associations between NE and coexisting psychiatric symptoms were statistically significant even when severity of ADHD symptoms was controlled. Thus, our findings support high NE as an independent marker of risk for psychopathology among youth with ADHD.

Given the overlap between behaviors associated with poor EC and core symptoms of ADHD (Martel et al., 2009; Nigg, 2006), one could argue that these constructs are not easily dissociable. In the current study, low EC was indeed correlated with IA and HI severities, but the associations were modest (Pearson correlation coefficients = .22 and .23, respectively). Moreover, low EC explained variance in depression, ODD and CD severities over and above that which was accounted for by ADHD symptoms. These results are consistent with mostly independent effects of EC and ADHD symptoms on coexisting psychopathology. Altogether, our findings suggest EC is not simply an analog of ADHD symptoms but instead has potential to contribute significantly to clinical assessment of youth with ADHD.

Low levels of EC have previously been associated with neurocognitive correlates of ADHD, including deficits in inhibition, working memory and processing speed (Wiersema & Roeyers, 2009). Though common, cognitive deficits in ADHD are not universal, and neuropsychological profiles vary considerably across individuals (Fair et al., 2012; Nigg, 2010). Moreover, neurocognitive profiles do not appear to distinguish ADHD from typically developing

youth (Fair et al., 2012; Loo et al., 2018), and cognitive deficits are likely correlates, rather than endophenotypes, of ADHD symptoms (Arnett et al., 2017). This literature parallels the research by Karalunas and colleagues (Karalunas et al., 2014) that reports considerable heterogeneity in temperament profiles among youth with ADHD, with or without coexisting psychopathology. The results of the current study support a growing body of research suggesting temperament traits may be candidate endophenotypes for ADHD (Nyman et al., 2012). Likewise, the current results underscore the importance of a dimensional approach to investigation of individual differences in behavioral and cognitive symptoms, consistent with the Research Domain Criteria (RDoC; Insel, 2014) initiative by the National Institute of Mental Health. In future work, we plan to examine whether temperament profiles, and particularly low EC, likewise explain individual differences in covariation across ADHD, psychiatric and neurocognitive symptoms among children.

Our results diverge somewhat from previous literature that has found associations between low EC and externalizing, but not internalizing symptoms. In contrast, we found that high NE/low EC was predictive of depression (although the effect of EC was smaller than the effect of NE in association with depression) as well as ODD and CD, but not anxiety. Relatedly, bivariate correlations indicated ODD and CD were both associated with depression. Thus, among children with elevated ADHD symptoms, parent-rated depression and externalizing behaviors appear to covary. This could be due in part to the fact that irritability is a common feature of depression, externalizing disorders, and ADHD (Mick et al., 2005). Moreover, polygenic risk analysis indicates depressive symptoms share common genetic variance with ADHD (Du Rietz et al., 2018). Importantly, coexisting mood disorders among children with ADHD are likely under-detected within pediatric outpatient settings (Rhodes et al., 2012). These findings raise the

question of whether traditional depression rating scales and clinical interviews show measurement invariance across children with and without ADHD.

Likewise, to our knowledge, the degree to which ADHD treatments improve depressive symptoms has not been systematically evaluated. Evidence for secondary effects of stimulant medications on depression in youth with ADHD varies across studies (Daviss, 2008). The seminal Multimodal Treatment of ADHD (MTA) study indicated that the sharpest rate of ADHD symptom reduction was among those with either medication management or combined behavioral and pharmacological treatment (Jensen, 1999). However, secondary analysis indicated that among those with coexisting internalizing disorders, the steepest rate of improvement was among those receiving combined treatment (Conners et al., 2001). Another secondary analysis found that a behavioral intervention designed to treat externalizing symptoms in children also reduced depressive symptoms (Webster-Stratton & Herman, 2008). Given youth with ADHD and depression are more likely to develop self-harm symptoms (Hurtig et al., 2012; Patros et al., 2013), accurate screening and development of evidence-based interventions for this high-risk group deserves attention in future research.

An unanticipated result of our study was that ADHD symptom severity only explained a minority of independent variance in coexisting symptom profiles (range: 0 – 21%). Severity of IA and HI were associated with anxiety in the first step of the regression model, but the association with IA was no longer significant once the temperament traits were added. The association between ADHD and anxiety therefore appears to be at least partially explained by temperament, although a mediation model was not explicitly tested. On the other hand, H/I remained a significant predictor of anxiety even with temperament traits in the model. This suggests that elevated H/I symptoms confer unique risk for anxiety in this population, and the

etiology of coexisting anxiety in the context of ADHD may partially overlap with the etiology of the H/I symptom domain.

Unlike anxiety, variance in depression was not explained by ADHD symptom severity. Anxiety and depression are distinct psychiatric constructs, despite often presenting together in clinical patients (Melton et al., 2016). Our results support distinct neurobiological pathways for coexisting anxiety versus depression in the context of ADHD (Caron & Rutter, 1991; Jarrett & Ollendick, 2008). We propose that temperament should be included in future models of multifinality and equifinality in psychiatric outcomes (Cicchetti & Rogosch, 1996).

Consistent with prior research, ODD was associated with maladaptive temperament across all three domains (Zastrow et al., 2018). Among clinical samples, upwards of 60% of youth with ADHD meet criteria for coexisting ODD (Connor et al., 2010). In community based samples, over 40% of children with ADHD meet criteria for ODD (Jensen, 1999) and the hazard ratio for ODD or CD has been reported at 9.54 (Yoshimasu et al., 2012). Examination of continuous ADHD and ODD traits among a population cohort indicates ODD symptoms rarely occur without symptoms of ADHD (Arnett et al., 2016). ODD is marked by emotion dysregulation, including aggressive outbursts, tantrums and refusal. The association between H/I symptoms and ODD was partially mediated by temperament traits. Thus our findings are consistent with the conceptualization of coexisting ADHD and emotion dysregulation as a manifestation of extremely maladaptive temperament, possibly driven by high genetic risk (Nigg et al., 2019). Interestingly, coexisting ODD in youth with ADHD may be moderated by environmental risk and parenting style (August et al., 1999; Noordermeer et al., 2017), which is contrary to the hypothesis of a strictly biological basis for temperament. Future intervention research should examine whether changes in temperament explain reduced ODD symptoms

among youth with ADHD following parent behavior management training or other behavioral treatments.

The temperament profiles associated with ODD and CD differed in that CD was not associated with elevated surgency or H/I. Thus, children with elevated ADHD who are at risk for CD symptoms may be expected to be emotionally reactive and have poor self-control, but do not necessarily have corresponding levels of social approach and impulsivity. Diagnoses of CD are less common among children with ADHD (27%; Larson et al., 2011), due to the severity of the clinical criteria and older age of onset. The current study likely under-recruited youth with CD due to our younger age range and study referral sources.

Studies of typically developing youth have reported sex differences in temperament traits, wherein females have higher rates of effortful control and males higher rates of surgency (Else-Quest et al., 2006). In contrast, the current sample did not show significant sex differences in temperament traits or coexisting psychopathology. Given the young age of our sample, this finding could be explained by recruitment bias, as females with high rates of externalizing symptoms may be more likely to be diagnosed with ADHD earlier. However, prior research indicates there are no sex differences in rates of comorbid disorders among youth with ADHD in population samples (Yoshimasu et al., 2012), and research on sex differences in temperament traits within ADHD samples is limited. Thus, our results may reflect an interesting phenomenon of reduced sex differences in temperament among children with ADHD. Lack of associations between demographic characteristics and ADHD treatment response has so far challenged the community of clinicians seeking to provide precision medicine care to this population (Rucklidge, 2010). The current study presents evidence that temperament traits are dissociable

from sex in this group, underscoring the potential for temperament to inform clinical decision making.

It is important to acknowledge that the CBCL simply measures frequency of developmentally-inappropriate symptoms associated with CD, such as cruelty to others and destruction of property and is based on parent report. A limitation of the study is that standardized diagnostic evaluations of coexisting disorders were not conducted for the current study. Although dimensional measurement of coexisting psychiatric traits is well aligned with literature on temperament and the Research Domain Criteria initiative (Musser & Raiker, 2019), future research should evaluate the predictive validity of our temperament profiles for DSM-based diagnoses in a wider age range.

Conclusions

ADHD is associated with a highly heterogeneous clinical presentation and developmental trajectory, which presents significant challenges for provision of precision medicine care. To date, we are unaware of any clinical tools that predict liability for future coexisting psychopathology among young children with ADHD, beyond family history and presence of current symptoms. As neurobiological and genomic research methods are developed to identify at-risk children at younger ages, it will likewise be important to identify reliable and efficient markers of coexisting psychopathology in this population. The current study suggests parent-ratings of temperament may be one such tool.

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Table 1

Correlations Among Temperament and Psychiatric Symptom Severities

	DEP	ANX	ODD	CD	IA	HI	NE	Surgency	EC
DEP	1								
ANX	**.51*****	1.00							
ODD	**.34****	**.23***	1.00						
CD	**.42*****	.14	**.75*****	1.00					
IA	-.01	**.30****	-.05	.01	1.00				
HI	.02	**.30****	**.42*****	**.26***	**.306****	1.00			
NE	**.48*****	**.58*****	**.32****	**.23***	.06	0.18	1.00		
Surgency	-.08	**-.23***	**.36****	**.22***	-.19	**.34****	-0.14	1.00	
EC	-.16	-.09	**-.40*****	**-.39*****	**-.22***	**-.23***	.04	-0.16	1.00

Note. Bolded font indicates two-tailed significance values at *$p < .05$, **$p < .01$, and ***$p < .001$. DEP, ANX, ODD and CD = CBCL age-standardized DSM-5 Depression, Anxiety, Oppositional Defiant Disorder, Conduct Disorder scores, respectively. IA and HI = SWAN Inattention and Hyperactivity/Impulsivity mean scores, respectively. NE = TMCQ Negative Emotionality. EC = TMCQ Effortful Control.

Table 2

Hierarchical Models of Coexisting Internalizing Symptoms

		Depression		Anxiety	
Step	Independent Variable	R^2	β	R^2	β
Model 1		.001	—	.136**	—
	SWAN IA	—	-.018	—	.228*
	SWAN H/I	—	.029	—	.229*
Model 2		.282***	—	.458***	—
	SWAN IA	—	-.068	—	.145
	SWAN H/I	—	-.089	—	.224*
	NE	—	.507***	—	.503***
	Surgency	—	-.030	—	-.219*
	EC	—	-.225*	—	-.062

Note. *$p < .05$. **$p < .01$. *** $p < .001$. Beta coefficients are standardized. SWAN IA = SWAN Inattentive Symptom severity. SWAN H/I = SWAN Hyperactive-Impulsive Symptom severity. NE = TMCQ Negative Emotionality. EC = TMCQ Effortful Control.

Table 3

Hierarchical Models of Coexisting Externalizing Symptoms

		ODD		CD	
Step	Independent Variable	R^2	β	R^2	β
Model 1		.214***	—	.072	—
	SWAN IA	—	-.199	—	-.076
	SWAN H/I	—	.483***	—	.281*
Model 2		.451***	—	.258***	—
	SWAN IA	—	-.185	—	-.093
	SWAN H/I	—	.259*	—	.109
	NE	—	.333***	—	.250*
	Surgency	—	.233*	—	.143
	EC	—	-.355***	—	-.369**

Note. *$p < .05$. **$p < .01$. *** $p < .001$. Beta coefficients are standardized. SWAN IA = SWAN Inattentive Symptom severity. SWAN H/I = SWAN Hyperactive-Impulsive Symptom severity. NE = TMCQ Negative Emotionality. EC = TMCQ Effortful Control.