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ARE EXECUTIVE FUNCTIONING DEFICITS CONCURRENTLY AND PREDICTIVELY ASSOCIATED WITH DEPRESSIVE AND ANXIETY SYMPTOMS IN ADOLESCENTS?

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Abstract

Background—The central objective of the current study was to evaluate how executive functions (EF), and specifically cognitive flexibility, were concurrently and predictively associated with anxiety and depressive symptoms in adolescence.

Method—Adolescents ($N = 220$) and their parents participated in this longitudinal investigation. Adolescents' EF was assessed by the Wisconsin Card Sorting Test (WCST) during the initial assessment, and symptoms of depressive and anxiety disorders were reported by mothers and youths concurrently and two years later.

Results—Correlational analyses suggested that youths who made more total errors (TE), including both perseverative errors (PE) and non-perseverative errors (NPE), concurrently exhibited significantly more depressive symptoms. Adolescents who made more TE and those who made more NPE tended to have more anxiety symptoms two years later. SEM analyses accounting for key explanatory variables (e.g., IQ, disruptive behavior disorders, and attention deficit hyperactive disorder) showed that TE was concurrently associated with parent reports of adolescent depressive symptoms.

Discussion—The results suggest internalizing psychopathology is associated with global (TE) and nonspecific (NPE) EF difficulties, but not robustly associated with cognitive inflexibility (PE). Future research with the WCST should consider different sources of errors which are posited to reflect divergent underlying neural mechanisms, conferring differential vulnerability for emerging mental health problems.

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Adolescents with elevated internalizing problems are at risk for myriad personal, academic, and social problems, including the development of major depressive disorder (MDD) and anxiety disorders (Weissman et al., 1999). Prospective longitudinal research on cognitive control mechanisms potentiating risk for internalizing disorders holds promise for improving our understanding of etiological pathways, and thereby refining treatment. Executive functioning (EF) has been conceptualized as higher order control mechanisms which orchestrate iterative processes engaged during conscious problem solving, encompassing sub-functions supporting problem representation, planning, execution, and evaluation (Zelazo, Carlson, & Kesek, 2008). While a broad array of EF components are likely to be implicated in internalizing problems, cognitive flexibility, the ability to contingently represent and apply different sets of rules to achieve changing goals, is a specific aspect of EF that has been posited as a neurocognitive endophenotype relevant for the understanding of both internalizing and externalizing psychopathology (Brent & Maalouf, 2009; Britton et al., 2010; Sjöwall, Roth, Lindqvist, & Thorell, 2013).

Adults with major depression have been found to show a global, diffuse pattern of EF deficits, including compromised functioning in attention (Purcell, Maruff, Kyrios, & Pantelis, 1997), behavioral inhibition (Murphy et al., 1999), memory (Ilsley, Moffoot, & O'Carroll, 1995), planning (Beats, Sahakian, & Levy, 1996), and flexible decision making (Channon, 1996; Murphy et al., 2001). Similarly, anxiety disorders in adults have been associated with cognitive inflexibility, as evidenced by less category achievement and more perseverative errors on the Wisconsin Card Sorting Test (WCST), a frequently used measure of EF (Bradbury et al., 2011; Fujii et al., 2013). Although important, it remains uncertain how applicable this research with adults is to adolescents.

The adolescent brain undergoes continuing development in regulatory brain regions associated with EF (Geidd, 2004). Some have speculated that those youths with effective EF are better able to process emotional information leading to both more flexible adaptive control over their environment and lower risk for psychopathology (Martel et al., 2007; Micco et al., 2009). Better EF may support youths' resilience against internalizing difficulties by enhancing their utilization of effective coping skills (Martel, Nigg, Wong, et al., 2007). Conversely, challenges with disengaging from a previously established conceptual frame can be viewed as a form of rigid rule reiteration (Zelazo, Carlson, & Kesek, 2008), possibly leading to cognitive styles that are characteristic of individuals with depressive and anxiety problems (Britton et al., 2010; Davis & Nolen-Hoeksema, 2000).

To date, there are few studies which have assessed EF with internalizing symptoms or problems in adolescents. Whereas EF impairments have been consistently implicated in adult anxiety and depression (Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari, & Lönnqvist, 2008), there has been inconsistent evidence in studies of adolescent psychopathology. Several studies of EF in samples that include a broad age range of

children and adolescents with internalizing disorders have documented impaired performance on several aspects of the WCST across both anxiety and depression (Beers & De Bellis, 2002; Shin et al., 2008). Considering a more impaired sample, Han and colleagues (2012) revealed that adolescents with MDD experienced more difficulties with sustained attention, compared with healthy youths without significant mental health concerns. Assessing another aspect of EF, Wilkinson and Goodyer (2006) found that adolescents with MDD were slower at acquiring new rules for set switching, compared with healthy controls. Similarly, Toren and colleagues (2000) found that, compared to healthy controls, youths with anxiety disorders showed more errors over all, more perseverative errors, and repetition of mistakes after receiving negative feedback on the WCST. In contrast, Kyte and colleagues (2005) failed to find EF differences between youths with and without depression. They studied adolescents with first episode MDD and found that depressed adolescents performed just as well as healthy controls on the Intra-Dimensional/Extra-Dimensional Set-Shifting task, which measures the ability to flexibly shift attention from one stimuli category to another. Similarly, Favre et al. (2009) assessed children and adolescents diagnosed with MDD on the WCST and the Trail Making Test, but failed to discern differences from healthy youths. Evaluating sustained attention in children and adolescents with MDD or anxiety disorders, Gunther and colleagues (2004) found that youths with internalizing psychopathology and their healthy counterparts exhibited comparable attentional performance. Therefore, the central objective of the current study was to clarify some of these discrepancies by considering EF and associated links with depressive and anxiety symptoms while accounting for relevant confounds not systematically assessed in previous research.

It is also critically important to examine how the links between EF and internalizing psychopathology change across adolescent development. Notably absent from the literature is longitudinal research on EF and the development of internalizing symptoms in adolescence. The few existing longitudinal studies have primarily focused on young children. For example, Riggs and colleagues (2003) conducted a prospective study on 60 1st and 2nd grade students in regular classrooms, over a period of two years. They found that more proficient inhibitory control and sequencing ability during the initial assessment were predictive of reductions in parent reported internalizing problems, as well as teacher and parent reported externalizing problems, by the follow up assessment. These findings suggested a developmental lag between the acquisition of EF skills and the manifestation of behavioral and emotional adaptations; it may take time for young children to incorporate newly acquired EF skills and translate them into regulators of undesirable behaviors. One could expect a similar pattern in adolescence, as youths increasingly face competing demands, such as academic expectations versus complex peer networks and romantic relationships, which would challenge their EF and could manifest as symptoms of distress. In the current study, we addressed the paucity of longitudinal investigations of EF and internalizing psychopathology during adolescence.

Study Objectives

In the current investigation, we recruited a community sample of adolescents who exhibited a range of internalizing problems, from normative to clinical levels. One aim of this study

was to clarify some of these discrepancies of past research by evaluating the concurrent associations of EF and internalizing psychopathology. The second aim of this study was to extend previous research and evaluate whether earlier EF predicted the development of anxiety and/or depressive symptoms. Initial results from correlational analyses were considered in addition to more comprehensive models which tested hypothesized links while accounting for comorbidity and other potentially important contributing factors (e.g., disruptive behavior disorders; Angold, Costello, & Erkanli, 1999; Dolan & Lennox, 2013; Kim, Kim, & Kwon, 2001; Sjöwall, Roth, Lindqvist, & Thorell, 2013). We hypothesized that more global EF deficits, cognitive inflexibility as noted in set shifting difficulties (perseverative errors on the WCST), and non-specific EF deficits (non-perseverative errors on the WCST) would be concurrently and predictively associated with higher depressive and anxiety symptoms in a large community sample of adolescents who were over-represented for internalizing psychopathology.

Method

Participants

Our sample consisted of 220 adolescents, aged 11-16 years at recruitment ($M = 13.67$, $SD = 1.52$), and their parents who were recruited to participate in a two wave longitudinal investigation of affective mechanisms in the development of psychopathology conducted at the National Institute of Mental Health (Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001). Recruitment strategies (see below) ensured that internalizing symptomatology was overrepresented in this community sample. Youths included in this study were 49.5% female, 70.0% White, and lived in predominantly two-parent families (76.9%) of middle to upper-middle socioeconomic status ($M = 52.9$, $SD = 10.8$ on the Hollingshead Index, 1975). The second wave of data collection occurred two years later ($M = 27.41$ mo, $SD = 6.10$), and included 177 youths (49.2% female) aged 13 to 19 years ($M = 15.6$, $SD = 1.6$) and their parents. Attrition analyses indicated that the Time 1 psychopathology profile, as assessed by the DISC-IV, did not differ between the youths who participated in the study at Time 2 and those who did not.

Procedures

Youths and their parents were recruited through announcements (e.g., newspapers, flyers) from the Washington, D. C. metropolitan area. We aimed to establish an over-representation of youths with elevated problem scores in the study. Study selection primarily depended on youths' psychological profile and took place via two phases. Phone screening interviews using abbreviated versions of the Youth Self-Report (YSR; Achenbach, 1991) and Child Behavior Checklist (CBCL; Achenbach, 1991) were conducted with families to assess youth psychopathology with elevated problems (*t* score 63 or higher, primarily on internalizing or comorbid internalizing and externalizing symptoms). Youths with elevated internalizing and internalizing/externalizing pathology were over-represented in this community sample with about a third of the sample showing clinical levels of problems (*t* score 70 or higher), a third of the sample showing subclinical levels of problems (*t* score of 63 or higher) and a third of the sample showing normative levels of problems (*t* score less than 60 for internalizing, externalizing and attention problems; see Klimes-Dougan, Hastings, Granger, Usher, &

Zahn-Waxler, 2001 for further details). At both Time 1 and Time 2, mothers and youths visited a comfortable, apartment-like laboratory suite where they independently reported on adolescents' symptoms on the NIMH Diagnostic Schedule for Children, Version IV (DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). At Time 1, 180 adolescents completed the Wisconsin Card Sorting Test (WCST), a measure of executive functioning. During a home visit that preceded the Time 1 laboratory visit by about two weeks, all adolescents also completed the KBIT, a measure of global intellectual functioning.

Assessment of Psychopathology

Symptoms of depressive, anxiety, disruptive behavior, and attention deficit hyperactive disorders were assessed with the DISC-IV. At both Time 1 and Time 2 lab visits, clinical psychologists and trainees supervised by a clinical senior team member administered the DISC IV in separate interviews to both mothers and adolescents. Past year symptom counts yielded dimensional scores of adolescents' mood disorders (including major depressive disorder and dysthymia), anxiety disorders (including specific phobia, social phobia, separation anxiety disorder, generalized anxiety disorder, obsessive-compulsive disorder, panic disorder, and agoraphobia), attention-deficit hyperactivity disorder (ADHD), and disruptive behavior disorders (including oppositional-defiant disorder, and conduct disorder). To prevent artificial influences on the range of reported symptoms, skip outs were not used in our administration of the DISC-IV. This index of psychopathology has been commonly used (e.g., Hope, Adams, Reynolds, Powers, Perez, & Kelley, 1999) to represent dimensions of psychopathology.

Assessment of Executive Functioning

Wisconsin Card Sorting Test—We assessed EF with the WCST (Heaton, 1981) at Time 1, a task that draws on multiple components of EF including sustained attention, working memory, cognitive flexibility, and response inhibition (Godinez, Friedman, Rhee, Miyake, & Hewitt, 2012). The WCST was designed to evaluate abstract reasoning, concept formation, and cognitive flexibility in response to contingent changes in sorting principle (Grant & Berg, 1948; Nyhus & Barceló, 2009). This computerized version of the WCST included four reference cards, consisting of one red triangle, two green stars, three yellow crosses, and four blue circles. On each trial, participants received a test card and were faced with the task of learning the stimuli dimension (i.e., shape, color, or number) set as the current 'latent' sorting principle through trial and error. After indicating each of their target-reference sorts, participants received feedback (i.e., correct vs. incorrect). Unannounced changes in the operational sorting principle occurred after participants achieved 10 successive correct trials, demanding cognitive flexibility to ensure accurate set switching. Youths were given as much time as they needed in order to sort 128 target cards or reaching the maximum possible, six correct sorting criteria. The WCST indices of interest for the present investigation included total errors (TE) as a measure of global EF deficit, perseverative errors (PE) as an index of cognitive inflexibility, and non-perseverative errors (NPE) as a measure of non-specific EF deficits, as well as total number of trials youths needed to complete the task as an indicator of overall efficiency. The total number of trials was included as a covariate in all models.

Assessment of Intellectual Functioning

Kaufman Brief Intelligence Test (K-BIT; Kaufman & Kaufman, 1990)—The K-BIT is a screening tool that is designed to measure crystalized and fluid intelligence as represented respectively by performance on vocabulary and matrices sections. Developmental norms for individuals 5 to 90 years have been established based on a nationally representative standardization sample. For this study, we used the composite measure of the K-BIT as an estimate of intelligence quotient (IQ).

Results

Descriptive Trends of Cognitive and Clinical Data

Adolescents in this study were generally above the average range of IQ functioning ($M = 113.2$; $SD = 11.5$). As shown in Table 1, performance on the WCST was generally comparable with age normative data (Chelune & Baer, 1986). Table 1 also presents mean number of symptoms of depression, anxiety, ADHD, and disruptive behavior disorders, as reported by youths and their mothers at each of the two assessments. There was no significant change over time for depressive symptoms, however, consistent with some prior research (e.g, Costello, Copeland, & Angold, 2011), both mothers and youths reported that the adolescent participants experienced significantly fewer anxiety symptoms at Time 2 compared to Time 1. By contrast, youths endorsed a significant increase in disruptive behaviors from Time 1 to Time 2. Mothers reported fewer symptoms of anxiety and depression at both Time 1 and Time 2 than adolescents (Anxiety: $t_{Time\ 1}(210) = 7.21$, $p_{Time\ 1} < 0.01$; $t_{Time\ 2}(173) = 5.35$, $p_{Time\ 2} < 0.01$. Depression: $t_{Time\ 1}(209) = 4.09$, $p_{Time\ 1} < 0.01$; $t_{Time\ 2}(174) = 3.94$, $p_{Time\ 2} < 0.01$). Additionally, at Time 1, mothers reported more disruptive behavior symptoms than did adolescents ($t(208) = 4.80$, $p < 0.01$). Males and females did not significantly differ on the number of total errors ($t(209) = 0.67$, $p = 0.50$), number of perseverative errors ($t(209) = 0.63$, $p = 0.53$), or number of nonperseverative errors ($t(209) = 1.14$, $p = 0.26$).

Associations between WCST and Psychopathology: Correlational Analyses

Table 2 summarizes the zero-order correlations between youth WCST errors, youth KBIT IQ scores at Time 1, and mother- and youth-reported DISC-IV symptoms at Times 1 and 2. As predicted, total (TE), perseverative (PE), and nonperseverative errors (NPE) on the WCST were positively correlated with mother-reported symptoms of depression at Time 1. Moreover, TE and NPE were positively associated with mother-reported anxiety symptoms at Time 2. Other relationships were also noted on Table 2. PE, NE and total trials to complete the WCST were positively correlated. Total number of trials to complete the WCST was positively associated with various indices of psychopathology (e.g., ADHD symptoms at Times 1 and 2). IQ and psychopathology were generally negatively correlated.

Associations between WCST and Psychopathology: Structural Equation Modeling

Structural equation modeling (SEM) was used for analyses because it allowed for simultaneous predictions to multiple outcome measures, accounted for potentially intervening variables, and accommodated missing data (Muthén, Kaplan, & Hollis, 1987;

Allison, 2003). All SEM models were specified using Amos version 22 statistical software (Arbuckle, 2006). In addition to disruptive behavior disorder and ADHD symptoms, given that both IQ and number of trials shared associations with WCST errors and DISC-IV scores, it was important to account for these covariates in modeling structural associations at Times 1 and 2. For these models, psychopathology symptoms of the four clinical outcomes were allowed to fully covary to permit the assessment of patterns of comorbidity. Likewise, number of trials, age, and IQ were allowed to covary. Results for both concurrent and predictive SEM analyses are only presented for mother-reports of youth problems given that none of the models testing associations between EF and youth self-reports of symptoms were significant.

For both concurrent and prospective analyses, we evaluated two models. The first focused on TE on the WCST as an index of global EF difficulties, the second evaluated error specificity by simultaneously estimating PE as a specific measure of cognitive inflexibility and NPE as a measure of non-specific errors. Estimates of direct effects are summarized in Tables 3 through 6 and standardized regression coefficients (denoted by β) are provided as measures of effect size. The models are presented in Figures 1 through 4 where significant effects ($p < .05$) have black lines, marginal effects ($.05 < p < .10$) have grey lines, and absent lines were non-significant ($p > .10$). Full information maximum likelihood (FIML) estimation was used to estimate model parameters given that there were missing data for neuropsychological assessment during Time 1 and symptom scores at Time 2 (n = 40 for WCST and n = 43 for Time 2 DISC-IV). FIML uses all of the available information to estimate model parameters and does not impute values for missing data.

Concurrent Associations

The first set of SEM analyses evaluated the concurrent links between EF and mother-reported symptoms of psychopathology, accounting for gender, age, IQ, and number of trials. Fit indices for the global model indicated acceptable fit of the model to data (model containing TE: CFI = 1.00, TLI = 1.07, RMSEA = .00, 90% CI for RMSEA = [.00, .04]). Consistent with predictions, TE was significantly associated with depressive symptoms ($p < .05$; see Table 3 and Figure 1). Thus, the significant correlation between TE and mother-reported depression was minimally affected by accounting for comorbid symptoms of disruptive behavior and ADHD, IQ, number of trials, gender and age. In addition, youths with higher IQ made significantly fewer TE (*Estimate* = −86.41, $p < .000$), and youths who took more trials to complete the task made significantly more TE (*Estimate* = 298.64, $p < .000$). The associations unrelated to TE included girls having fewer disruptive behavior ($\beta = -.24$, $p < .000$) and ADHD symptoms ($\beta = -.25$, $p < .000$) than boys, and youths with higher IQ having fewer disruptive ($\beta = -.21$, $p < .01$) and ADHD symptoms ($\beta = -.31$, $p < .001$). There was significant co-variation among the four psychopathology symptom scores, confirming the importance of evaluating comorbidity.

Fit indices for the model evaluating error specificity indicated acceptable fit of the model to data (model containing PE and NPE: CFI = 1.00, TLI = 1.07, RMSEA = .00, 90% CI for RMSEA = [.00, .03]). Since PE and NPE were highly correlated (Table 2), we modeled error specificity by evaluating both types of errors as predictors of contemporaneous

psychopathology. For this error specificity model, neither type of errors was concurrently associated with clinical symptoms (see Table 4 and Figure 2). Contrary to predictions, PE and NPE were not significantly associated with concurrent depressive or anxiety symptoms. The only associations with errors were that youths with higher IQ made fewer NPE (*Estimate* = −42.02, *p* < .000), and youths who took more trials to complete the task made more NPE (*Estimate* = 156.73, *p* < .000). Across the two contemporaneous models then, the reported association between mother-reported concurrent depressive symptoms and EF deficits on the WCST appeared to reflect global as opposed to specific types of errors.

Prospective Associations

The second set of SEM analyses examined predictive associations between indices of EF at Time 1 and mother-reported psychopathology symptoms assessed two years later, at Time 2. In addition to the covariances that were estimated in the contemporaneous models, the four forms of psychopathology at the second time were allowed to covary, and EF was allowed to covary with the other measures at Time 1. In an effort to diminish the complexity of the models and to address the corresponding limitations of power, direct paths from age and IQ to Time 2 psychopathologies were not specified in these models because exploratory analyses failed to show associations between these indexes and internalizing pathology at Time 2. Covariances between age and EF indices and between IQ and EF indices were estimated.

Fit indices for the global model indicated acceptable fit of the model to data (model containing TE: CFI = .95, TLI = .85, RMSEA = .09, 90% CI for RMSEA = [.07, .11]). Contrary to predictions, there were no significant associations between Time 1 TE and Time 2 depressive or anxiety symptoms. However, youths who made more TE at Time 1 tended to have more anxiety symptoms at Time 2 (see Table 5 and Figure 3). Thus, controlling for all covariates as well as the stability of DISC-IV symptom scores did not fully account for the significant correlation between Time 1 TE and Time 2 anxiety symptoms. Also, Time 1 TE was not related to any other Time 2 symptom scores. All symptom scores were significantly stable over time, and there were numerous significant predictive links across symptom scores (i.e., Time 1 anxiety predicted Time 2 depression and ADHD; Time 1 disruptive behavior predicted Time 2 depression, anxiety and ADHD). These results indicated that earlier TE marginally predicted future symptoms of anxiety only.

Fit indices for the error specificity model indicated acceptable fit of the model to data (model containing PE and NPE: CFI = .96, TLI = .84, RMSEA = .09, 90% CI for RMSEA = [.07, .11]). Contrary to predictions, there were no significant associations between Time 1 PE and Time 2 depressive or anxiety symptoms. However, youths who made more NPE at Time 1 tended to have more anxiety problems at Time 2 (see Table 6 and Figure 4). Thus, again, the correlation between Time 1 NPE and Time 2 anxiety symptoms was not fully attributable to other measured variables. In addition, all of the associations between predictors and psychopathology identified in the prior model were maintained. Together, these analyses extended the previous model on global EF deficits, as measured by TE, and implied that it was specifically the number of NPE that accounted for the marginal association between earlier EF deficits and prospective anxiety symptoms.

Discussion

This study considered distributed attention and cognitive flexibility functions implicated in executive functioning (EF) and their concurrent and predictive associations with internalizing symptomatology. Our results provided evidence that youths who exhibited higher levels of global EF deficits (i.e., TE) showed significantly more concurrent depressive symptoms. There was also preliminary evidence that prospectively, having more global EF deficits and making more non-perseverative errors tended to predict youths having more anxiety symptoms two years later. Contrary to predictions, the results of this study failed to provide convincing evidence that perseverative errors, an indicator of cognitive inflexibility, were associated with internalizing psychopathology. An important advance of this work is that, given the controls specified in SEM, the findings linking EF and internalizing psychopathology could not be solely attributed to such contributing factors as intellectual functioning, the number of trials it took for participants to complete the WCST, or symptoms of comorbid externalizing psychopathology (i.e., ADHD and disruptive behavior). These findings advance our understanding of EF in adolescents exhibiting internalizing symptoms by suggesting that EF deficits may characterize youths with concurrent depressive symptoms, but contribute to the emergence of anxiety symptoms over development. These longitudinal inferences are speculative given the trend effects and correlational design of the study, but warrant further investigation.

Error Specificity

EF has many interrelated and non-independent components; on the WCST, youths who made more perseverative errors also made more non-perseverative errors. Although we hypothesized that cognitive inflexibility as measured by perseverative errors on the WCST would be specifically linked with internalizing psychopathology, our findings suggest that global EF errors are implicated in both depression and anxiety, while non-perseverative errors tended to be associated with prospective anxiety symptoms during adolescence. Degl'Innocenti and colleagues (1998) similarly found that depressed adults made more non-perseverative errors, but not more perseverative errors on the WCST compared with healthy controls. They suggested that individuals with MDD were sufficiently flexible, as they chose an incorrect sorting rule that was not previously reinforced; however, they did not alter their sorting adaptively after receiving negative feedback. Non-perseverative errors on the WCST reflect difficulties with sustained attention, working memory, and response inhibition, depending on more precise reasons for the incorrect sorts made (Barceló, 1999; Barceló & Knight, 2002; Godinez et al., 2012). For example, sustained attention deficits coded as non-perseverative errors are generated by youths who break set after finding the correct current sorting principle and subsequently choosing another category that was not previously reinforced. This could be seen as consistent with our prior work showing difficulties with sustained attention (as measured by the Continuous Performance Task) in adolescents diagnosed with MDD (Han et al., 2012). Future research examining associations among psychopathology and WCST error indices will be enhanced by more precisely defining types of errors (e.g., those made prior to or after learning the current latent sorting principle; Godinez, Friedman, Rhee, Miyake, & Hewitt, 2012) as non-perseverative errors are posited

to reflect several different underlying neural mechanisms (Nyhus & Barceló, 2009) which confer differential vulnerability for psychopathology presentation.

Intact Cognitive Flexibility in at Risk Youths

Conversely, we did not find evidence linking cognitive inflexibility, as measured by perseverative errors on the WCST, with depressive symptoms concurrently or prospectively. Previous studies have shown equivocal findings with respect to the presence of cognitive inflexibility in adolescents with MDD (Kyte, Goodyer, & Sahakian, 2005; Wilkinson & Goodyer, 2006). Although we assessed depressive symptoms with a clinical interview, few adolescents in our sample met criteria for a clinical diagnosis of MDD. Cognitive inflexibility may reflect a feature of EF deficit more characteristic of severe, recurrent depression while subclinical depressive symptoms may be linked with an array of non-specific EF deficits. It is important to recognize that across the two assessments, depressive symptoms did not increase and hence, it is possible that we did not study a long enough window of development in order to identify how initial EF deficits confer risk for the exacerbation of existing depressive symptoms. Alternatively, cognitive flexibility as measured in perseverative errors might not be a precursor, or early-emerging correlate, of depression. The reasonably consistent evidence for concurrent links between depression in adults and cognitive inflexibility (Castaneda et al., 2008) could emerge from the accumulated experiences of living with MDD, leading to patterns of rigid and maladaptive cognitive control. In other words, cognitive inflexibility may be a consequence of depression, rather than a cause. This possibility could be examined in longitudinal repeated-measures investigations of individuals with depression developing from late adolescence into adulthood.

Developmental and Measurement Considerations

To place our main finding of the significant association between global EF deficits and adolescents' concurrent depressive symptoms in a developmental framework, it is important to ponder potential sources of variability which may contribute to differences in patterns of EF-psychopathology associations reported in studies of adults versus those of adolescents (Bradbury, Cassin, & Rector, 2011; Kyte, Goodyer, & Sahakian, 2005). Two important sources of clinical variability are illness duration and contextual amplification. Major depression is often a recurring illness (Weissman et al., 1999); as such, an adult with depression is more likely than an adolescent with depression to have had a longer course of the illness. Cognitive flexibility may have been preserved in our sample due to the shorter duration of illness, compared with adult clinical samples. Adults also have more social and occupational responsibilities than adolescents on average, and thus, EF deficits associated with depression may not be discernable until contextual demands tax the individual's neurocognitive system sufficiently. Inconsistencies within the adolescent literature may stem from factors such as differences between community versus clinical samples, duration and intensity of symptoms, patterns of comorbidity, age range studied and accompanying level of neurological maturation, potential neurocognitive changes incurred by medication use, and the facets of EF assessed by different tasks. Regarding the component functions tapped by the WCST (i.e., cognitive flexibility, sustained attention, working memory), the

functional maturity of brain regions supporting these aspects of EF show different developmental courses (Crone, Zanolie, van Leijenhorst, Westenberg, & Rombouts, 2008). We may not have found significant associations between cognitive flexibility difficulties and internalizing psychopathology due to the more protracted development of brain regions involved, which extends well into emerging adulthood (Casey, Galvan, & Hare, 2005; Kelly et al., 2009). Moreover, these methodological concerns generally impact the interpretation of adult studies as well. Therefore, we recommend future studies of EF in internalizing psychopathology maximize their assessment of clinical specificity and use diverse, developmentally appropriate tasks to probe patterns of EF deficits and preserved skills.

Types of Executive Functions and Longitudinal Implications

Given the purely cognitive features engaged by the WCST, it is plausible that youths with more depressive symptoms may have performed more poorly overall due to difficulties with motivation. Some of our prior research has found that when assessed on affective decision making, a ventromedial prefrontal cortex mediated form of this type of EF (Hongwanishkul, Happaney, Lee, & Zelazo, 2005), depressed adolescent girls and boys showed distinct profiles of performance (Han et al., 2012). On the Iowa Gambling Task, depressed girls were harm avoidant, selecting more frequently from advantageous decks, whereas depressed boys were more impulsive, picking more from disadvantageous decks. Thus, while the current study did not show evidence of cognitive flexibility deficits being linked with depressive symptoms in a community sample of adolescents, our prior work showed impulsive affective decision making in a clinical sample of depressed adolescent boys. Future efforts will be needed to discern whether task differences or sample differences contributed to these discrepancies.

This is the first longitudinal study that has documented different patterns of EF deficits for adolescents with elevated depressive symptoms as compared to adolescents with elevated anxiety symptoms. In contrast to the pattern by which global EF deficits were significantly associated with depressive symptoms concurrently, the prospective analyses showed that youths who exhibited more global EF deficits and those who made more non-perseverative errors during the initial assessment tended to have more anxiety symptoms two years later. Some previous studies of youths with clinical diagnoses of anxiety disorders found concurrent EF deficits (Shin et al., 2008; Toren et al., 2000), but other work has failed to show a concurrent link between internalizing problems and EF deficits (Romer et al., 2009). The adolescents in our study showed a decline in anxiety symptoms over the two year period. It is possible that the subtle yet multi-faceted EF deficits captured by non-perseverative errors on the WCST were not associated with early-emerging anxiety symptoms due to other strengths or protective factors, such as the fact that these youths generally had strong intellectual capacities. However, as they entered later adolescence and faced increasingly competing demands for balancing academic performance, complex peer networks, and forays into romantic relationships, youths with broad EF deficits may have experienced more stress and difficulties with effective coping.

The results of this study assessed symptomatology using multiple informants. However, it was somewhat surprising that links between internalizing symptoms and EF were only

supported in the models when mothers were informants on adolescents' psychopathology and not when youths were informants on their own problems. This pattern of results was noted for both concurrent and predictive links with EF, despite the slightly more restricted range of problems identified by parents (lower means and standard deviations). Developmental research on informant discrepancies for internalizing symptoms has shown that as people age, reporting discrepancies increase (van der Ende, Verhulst, & Tiemeier, 2012). Parental reports on internalizing symptoms might capture symptoms that are more associated with the directly observable cognitive and behavioral features of internalizing psychopathology, rather than affective and subjective features like dysphoria (Hastings, Klimes-Dougan, Brand, Kendziora, & Zahn-Waxler, 2014), and the former could be more related to EF than the latter. Thus, future research examining links between EF and psychopathology as rated by multiple informants could approach data analysis using signed difference scores, comparing informant pairs.

Limitations and Future Directions

The findings of this study should be interpreted in the context of several important limitations.

Using SEM allowed us to account for a number of important intervening variables. While this sample is reasonably large, the array of factors accounted for in the models were accompanied by some power constraints. Given that many of the hypothesized links were marginally significant, it is likely that larger samples will be essential for determining with confidence these links between EF and internalizing psychopathology. In addition, the relatively high SES and IQ in the sample of youths we studied might have limited the ability to detect associations between EF and mental health symptoms by limiting the range on both constructs. As such, the extent to which our findings could be generalized to socioeconomically and intellectually diverse samples is not estimable as both environmental and biological factors contribute to regulatory dynamics guiding development (Lickliter & Honeycutt, 2003).

We only assessed EF during the initial time point and were therefore not able to evaluate developmental continuity versus change in EF and the impact of EF development on youths' emerging internalizing symptoms. Additionally, employing diverse assessments of EF would facilitate the delineation of more precise links between clinical symptoms and underlying neurocognitive difficulties. We only assessed "cool," or more cognitive, forms of EF. Future research evaluating cognitive flexibility would benefit by including emotionally salient paradigms such as the Affective Go/No-go, as previous studies using emotionally neutral attentional switching tasks also have not consistently revealed deficits associated with internalizing psychopathology (Kyte, Goodyer, & Sahakian, 2005; Wilkinson & Goodyer, 2006). Given that emotion dysregulation is characteristic of internalizing psychopathology, it would be informative for future studies to include assessments of both "cool" and "hot" EF, as the latter are more affectively and motivationally salient (Prencipe, Kesek, Cohen, Lamm, Lewis, & Zelazo, 2011) such as affective attentional bias (Epp, Dobson, Dozois, & Frewen, 2012) and reward decision making (Paulus & Yu, 2012).

Future cross disciplinary collaborations that advance multilevel research on mood and anxiety disorders will increase our understanding of the etiology and developmental change of internalizing psychopathology, particularly by incorporating neurobiological methods (Han, Miller, Cole, Zahn-Waxler, & Hastings, in press; Hastings, Kahle, & Han, 2014; Klimes-Dougan, et al., in press). Though yet to be used extensively to identify adolescents at risk for developing internalizing disorders, combining neuropsychological testing while undergoing neuroimaging provides a design for determining how key prefrontal regions (e. g., dorsolateral prefrontal cortex and anterior cingulate cortex) respond to specific cognitive challenges. In addition, incorporating neurophysiological methods could increase the specificity of our understanding of multi-level markers of internalizing psychopathology. For example, behavioral performance on established EF tasks can be comparable between clinical and non-clinical groups, despite neurophysiological differences suggesting that anxious individuals recruit increased activity of key frontal regions (e.g., dorsolateral prefrontal cortex) in order to compensate for decreased efficiency, thereby achieving adequate attentional control (Basten et al., 2011; Berggren & Derakshan, 2013).

Conclusions

In a community sample of adolescents at risk for internalizing psychopathology followed across two years, we found that global EF deficits were significantly associated with concurrent depressive symptoms, whereas both global EF deficits and non-perseverative errors on the WCST tended to predict future anxiety symptoms. These findings failed to provide evidence that cognitive flexibility was impaired in youths with internalizing psychopathology. This work highlights the importance for future research to consider EF deficits more precisely as perseverative and non-perseverative errors are posited to reflect different underlying neurophysiology and confer differential vulnerability for emerging mental health problems. Additionally, this work may suggest a direction for future prevention and intervention initiatives. It is possible that many of the newly emerging interventions focused on enhancing cognitive flexibility (Ives-Deliperi, Howells, Stein, Meintjes, & Horn, 2013) are not ideally suited to this population; conversely, attention training (e.g., Bar-Haim, 2010; Calkins, McMorran, Siegle & Otto, 2014) may be more strongly indicated for those showing early signs of depressive and anxiety disorders.

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REFERENCES

Achenbach, TM. Manual for the Child Behavior Checklist/4-18 and 1991 Profile. Author, Burlington; VT: 1991.

Allison PD. Missing data techniques for structural equation modeling. Journal of Abnormal Psychology. 2003; 112:545–557. [PubMed: 14674868]

Angold A, Costello EJ, Erkanli A. Comorbidity. Journal of Child Psychology and Psychiatry. 1999; 40(1):57–87. [PubMed: 10102726]

Arbuckle, JL. Amos 7.0 user's guide. SPSS; Chicago: 2006.

Barceló F. Electrophysiological evidence of two different types of error in the Wisconsin Card Sorting Test. Neuroreport. 1999; 10:1299–1303. [PubMed: 10363943]

Barceló F, Knight RT. Both random and perseverative errors underline WCST deficits in prefrontal patients. Neuropsychologia. 2002; 40:349–356. [PubMed: 11684168]

Bar-Haim Y. Research review: Attention bias modification (ABM): a novel treatment for anxiety disorders. Journal of Child Psychology and Psychiatry. 2010; 51(8):859–870. [PubMed: 20456540]

Basten U, Stelzel C, Fiebach CJ. Trait anxiety modulates the neural efficiency of inhibitory control. Journal of Cognitive Neuroscience. 2011; 23(10):3132–3145. [PubMed: 21391763]

Beats BC, Sahakian BJ, Levy R. Cognitive performance in tests sensitive to frontal lobe dysfunction in the elderly depressed. Psychological Medicine. 1996; 26:591–603. [PubMed: 8733217]

Beers SR, De Bellis MD. Neuropsychological function in children with maltreatment-related posttraumatic stress disorder. American Journal of Psychiatry. 2002; 159(3):483–486. [PubMed: 11870018]

Berggren N, Derakshan N. Attentional control deficits in trait anxiety: why you see them and why you don't. Biological Psychology. 2013; 92(3):440–446. [PubMed: 22465045]

Bradbury C, Cassin SE, Rector NA. Obsessive beliefs and neurocognitive flexibility in obsessive-compulsive disorder. Psychiatry Research. 2011; 187:160–165. [PubMed: 21112643]

Brent DA, Maalouf FT. Pediatric depression: is there evidence to improve evidence-based treatments? Journal of Child Psychology and Psychiatry. 2009; 50(1-2):143–152. [PubMed: 19220597]

Britton JC, Rauch SL, Rosso IM, Killgore WDS, Price LM, Ragan J, Chosak A, Hezel DM, Pine DS, Leibenluft E, Pauls DL, Jenike MA, Stewart SE. Cognitive inflexibility and frontal-cortical activation in pediatric Obsessive Compulsive Disorder. Journal of the American Academy of Child and Adolescent Psychiatry. 2010; 49(9):944–953. [PubMed: 20732630]

Calkins AW, McMorran KE, Siegle GJ, Otto MW. The effects of computerized cognitive control training on community adults with depressed mood. Behavioural and Cognitive Psychotherapy. 2014; 3:1–12.

Cantwell DP, Lewinsohn PM, Rohde P, Seeley JR. Correspondence between adolescent report and parent report of psychiatric diagnostic data. Journal of the American Academy of Child & Adolescent Psychiatry. 1997; 36(5):610–619. [PubMed: 9136495]

Casey BJ, Galvan A, Hare TA. Changes in cerebral functional organization during cognitive development. Current Opinion in Neurobiology. 2005; 15(2):239–244. [PubMed: 15831409]

Castaneda AE, Tuulio-Henriksson A, Marttunen M, Suvisaari J, Lönnqvist J. A review on cognitive impairments in depressive and anxiety disorders with a focus on young adults. Journal of Affective Disorders. 2008; 106(1-2):1–27. [PubMed: 17707915]

Channon S. Executive dysfunction in depression: The Wisconsin Card Sorting Test. Journal of Affective Disorders. 1996; 39:107–114. [PubMed: 8827419]

Chelune GJ, Baer RA. Developmental norms for the Wisconsin Card Sorting Test. Journal of Clinical and Experimental Neuropsychology. 1986; 8:219–228. [PubMed: 3722348]

Costello EJ, Copeland W, Angold A. Trends in psychopathology across the adolescent years: What changes when children become adolescents, and when adolescents become adults?: Trends in psychopathology across the adolescent years. Journal of Child Psychology and Psychiatry. 2011; 52(10):1015–1025. [PubMed: 21815892]

Crone EA, Zanolie K, van Leijenhorst L, Westenberg PM, Rombouts SARB. Neural mechanisms supporting flexible performance adjustment during development. Cognitive, Affective, & Behavioral Neuroscience. 2008; 8(2):165–177.

Davis RN, Nolen-Hoeksema S. Cognitive inflexibility among ruminators and nonruminators. Cognitive Therapy and Research. 2000; 24(6):699–711.

Degl'Innocenti A, Agren H, Backman L. Executive deficits in major depression. Acta Psychiatrica Scandinavica. 1998; 97(3):182–188. [PubMed: 9543305]

Dolan M, Lennox C. Cool and hot executive function in conduct-disordered adolescents with and without co-morbid attention deficit hyperactivity disorder: relationships with externalizing behaviors. Psychological Medicine. 2013; 43(11):2427–2436. [PubMed: 23363609]

Epp AM, Dobson KS, Dozois DJA, Frewen PA. A systematic meta-analysis of the Stroop task in depression. Clinical Psychology Review. 2012; 32(4):316–328. [PubMed: 22459792]

Favre T, Hughes C, Emslie G, Stavinoha P, Kennard B, Carmody T. Executive functioning in children and adolescents with Major Depressive Disorder. Child Neuropsychology. 2009; 15(1):85–98. [PubMed: 19089695]

Fujii Y, Kitagawa N, Shimizu Y, Mitsui N, Toyomaki A, Hashimoto N, Kako Y, Tanaka T, Asakura S, Koyama T, Kusumi I. Severity of generalized social anxiety disorder correlates with low executive functioning. Neuroscience Letters. 2013; 543:42–46. [PubMed: 23562516]

Giedd JN. Structural magnetic resonance imaging of the adolescent brain. Annals of the New York Academy of Science. 2004; 1021:77–85.

Godinez DA, Friedman NP, Rhee SH, Miyake A, Hewitt JK. Phenotypic and genetic analyses of the Wisconsin Card Sort. Behavioral Genetics. 2012; 42(2):209–220.

Grant DA, Berg E. A behavioral analysis of degree of reinforcement and ease of shifting to new responses in Weigl-type card-sorting problem. Journal of Experimental Psychology. 1948; 38:404–411. [PubMed: 18874598]

Günther T, Holtkamp K, Jolles J, Herpertz-Dahlmann B, Konrad K. Verbal memory and aspects of attentional control in children and adolescents with anxiety disorders or depressive disorders. Journal of Affective Disorders. 2004; 82:265–269. [PubMed: 15488256]

Han G, Klimes-Dougan B, Jepsen S, Ballard K, Nelson M, Houri A, Kumra S, Cullen K. Selective neurocognitive impairments in adolescents with major depressive disorder. Journal of Adolescence. 2012; 35:11–20. [PubMed: 21782233]

Han, G.; Miller, JG.; Cole, PM.; Zahn-Waxler, C.; Hastings, PD. Adolescents' internalizing and externalizing problems predict their affect-specific HPA and HPG axes reactivity. Developmental Psychobiology. 2015. DOI 10.1002/dev.21268. [E-Pub ahead of print]

Hastings, PD.; Kahle, SS.; Han, G. Developmental affective psychophysiology: Using physiology to inform our understanding of emotional development. In: Lagattuta, KH., editor. Children and emotion: New insights into developmental affective sciences. Vol. 26. Karger; Basel: 2014. p. 13-28.Contributions to human development

Hastings PD, Klimes-Dougan B, Brand A, Kendziora KT, Zahn-Waxler C. Regulating sadness and fear from outside and within: Mothers' emotion socialization and adolescents' parasympathetic regulation predict the development of internalizing difficulties. Development and Psychopathology. 2014; 26:1369–1384. [PubMed: 25422967]

Heaton, RK. The Wisconsin Card Sorting Test manual. Psychological Assessment Resources Inc; Odessa: 1981.

Hollingshead, AB. Four factor index of social status. 1975. Unpublished paper

Hongwanishkul D, Happaney KR, Lee WS, Zelazo PD. Assessment of hot and cool executive function in young children: age-related changes and individual differences. Developmental Neuropsychology. 2005; 28(2):617–644. [PubMed: 16144430]

Hope TL, Adams C, Reynolds L, Powers D, Perez RA, Kelley ML. Parent vs. self-report: Contributions toward diagnosis of adolescent psychopathology. Journal of Psychopathology and Behavioral Assessment. 1999; 21(4):349–363.

Ilsley JE, Moffoot APR, O'Carroll RE. An analysis of memory dysfunction in major depression. Journal of Affective Disorders. 1995; 35:1–9. [PubMed: 8557882]

Ives-Deliperi VL, Howells F, Stein DJ, Meintjes EM, Horn N. The effects of mindfulness-based cognitive therapy in patients with bipolar disorder: a controlled functional MRI investigation. Journal of Affective Disorders. 2013; 150(3):1152–1157. [PubMed: 23790741]

Kaufman, AS.; Kaufman, NL. Manual for the Kaufman Brief Intelligence Test. American Guidance Service; Circle Pines, MN: 1990.

Kelly AM, Di Martino A, Uddin LQ, Shehzad Z, Gee DG, Reiss PT, Margulies DS, Castellanos FX, Milham MP. Development of anterior cingulate functional connectivity from late childhood to early adulthood. Cerebral Cortex. 2009; 19(3):640–657. [PubMed: 18653667]

Kim MS, Kim JJ, Kwon JS. Frontal P300 decrement and executive dysfunction in adolescents with conduct problems. Child Psychiatry and Human Development. 2001; 32(2):93–106. [PubMed: 11758881]

Klimes-Dougan B, Hastings PD, Granger DA, Usher BA, Zahn-Waxler C. Adrenocortical activity in at-risk and normally developing adolescents: Individual differences in salivary cortisol basal levels, diurnal variation, and responses to social challenges. Development and Psychopathology. 2001; 13(3):695–719. [PubMed: 11523855]

Klimes-Dougan, B.; Eberly, LE.; Schreiner, MW.; Kurkiewicz, P.; Houri, A.; Schlesinger, A.; Thomas, KM.; Mueller, BA.; Lim, KO.; Cullen, KR. Multilevel assessment of the neurobiological threat system in depressed adolescents: Interplay between the limbic system and HPA axis. Development and Psychopathology. in press

Kyte ZA, Goodyer IM, Sahakian BJ. Selected executive skills in adolescents with recent first episode major depression. Journal of Child Psychology and Psychiatry. 2005; 46(9):995–1005. [PubMed: 16109002]

Lickliter R, Honeycutt H. Developmental dynamics: toward a biologically plausible evolutionary psychology. Psychological Bulletin. 2003; 129(6):819–835. [PubMed: 14599279]

Loeber R, Green SM, Lahey BB, Stouthamer-Loeber M. Differences and similarities between children, mothers, and teachers as informants on disruptive child behavior. Journal of Abnormal Child Psychology. 1991; 19(1):75–95. [PubMed: 2030249]

Martel MM, Nigg JT, Wong MM, Fitzgerald HE, Jester JM, Puttler LI, Glass JM, Adams KM, Zucker RA. Childhood and adolescent resiliency, regulation, and executive functioning in relation to adolescent problems and competence in a high-risk sample. Development and Psychopathology. 2007; 19(2):541–563. [PubMed: 17459183]

Martel M, Nikolas M, Nigg JT. Executive function in adolescents with ADHD. Journal of the American Academy of Child and Adolescent Psychiatry. 2007; 46(11):1437–1444. [PubMed: 18049293]

Micco JA, Henin A, Biederman J, Rosenbaum JF, Petty C, Rindlaub LA, Murphy M, Hirshfeld-Becker DR. Executive functioning in offspring at risk for depression and anxiety. Depression and Anxiety. 2009; 26(9):780–790. [PubMed: 19434624]

Murphy FC, Rubinsztein JS, Michael A, Rogers RD, Robbins TW, Paykel ES, Sahakian BJ. Decision-making cognition in mania and depression. Psychological Medicine. 2001; 31:679–693. [PubMed: 11352370]

Murphy FC, Sahakian BJ, Rubinsztein JS, Michael A, Rogers RD, Robbins TW, Paykel ES. Emotional bias and inhibitory control processes in mania and depression. Psychological Medicine. 1999; 29:1307–1321. [PubMed: 10616937]

Muthén B, Kaplan D, Hollis M. On structural equation modeling with data that are not missing completely at random. Psychometrika. 1987; 52:431–462.

Nyhus E, Barceló F. The Wisconsin Card Sorting Test and the cognitive assessment of prefrontal executive functions: A critical update. Brain and Cognition. 2009; 71:437–451. [PubMed: 19375839]

Paulus MP, Yu AJ. Emotion and decision-making: affect-driven belief systems in anxiety and depression. Trends in Cognitive Science. 2012; 16(9):476–483.

Prencipe A, Kesek A, Cohen J, Lamm C, Lewis MD, Zelazo PD. Development of hot and cool executive function during the transition to adolescence. Journal of Experimental Child Psychology. 2011; 108(3):621–637. [PubMed: 21044790]

Purcell R, Maruff P, Kyrios M, Pantelis C. Neuropsychological function in young patients with unipolar major depression. Psychological Medicine. 1997; 27:1277–1285. [PubMed: 9403899]

Riggs NR, Blair CB, Greenberg MT. Concurrent and 2-year longitudinal relations between executive function and the behavior of 1st and 2nd grade children. Child Neuropsychology. 2003; 9(4):267–276. [PubMed: 14972705]

Romer D, Betancourt L, Giannetta JM, Brodsky NL, Farah M, Hurt H. Executive cognitive functions and impulsivity as correlates of risk taking and problem behavior in preadolescents. Neuropsychologia. 2009; 47(13):2916–2926. [PubMed: 19560477]

Shaffer D, Fisher P, Lucas CP, Dulcan MK, Schwab-Stone ME. NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. Journal of the American Academy of Child & Adolescent Psychiatry. 2000; 39:28–38. [PubMed: 10638065]

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Shin MS, Choi H, Kim H, Hwang JW, Kim BN, Cho SC. A study of neuropsychological deficit in children with obsessive-compulsive disorder. European Psychiatry. 2008; 23:512–520. [PubMed: 18514491]

Sjöwall D, Roth L, Lindqvist S, Thorell LB. Multiple deficits in ADHD: executive dysfunction, delay aversion, reaction time variability, and emotional deficits. Journal of Child Psychology and Psychiatry. 2013; 54(6):619–627. [PubMed: 23061803]

Toren P, Sadeh M, Wolmer L, Eldar S, Koren S, Weizman R, Laor N. Neurocognitive correlates of anxiety disorders in children: A preliminary report. Journal of Anxiety Disorders. 2000; 14(3): 239–247. [PubMed: 10868982]

Van der Ende J, Verhulst FC, Tiemeier H. Agreement of informants on emotional and behavioral problems from childhood to adulthood. Psychological Assessment. 2012; 24(2):293–300. [PubMed: 21928910]

Weissman MM, Wolk S, Goldstein RB, Moreau D, Adams P, Greenwald S, Klier CM, Ryan ND, Dahl RE, Wickramaratne P. Depressed adolescents grown up. Journal of the American Medical Association. 1999; 281(18):1707–1713. [PubMed: 10328070]

Wilkinson PO, Goodyer IM. Attention difficulties and mood-related ruminative response style in adolescents with unipolar depression. Journal of Child Psychology and Psychiatry. 2006; 47(12): 1284–1291. [PubMed: 17176383]

Zelazo, PD.; Carlson, SM.; Kesek, A. Development of executive function in childhood. In: Nelson, CA.; Luciana, M., editors. Handbook of developmental cognitive neuroscience. 2nd. MIT Press; Cambridge, MA: 2008. p. 553-574.
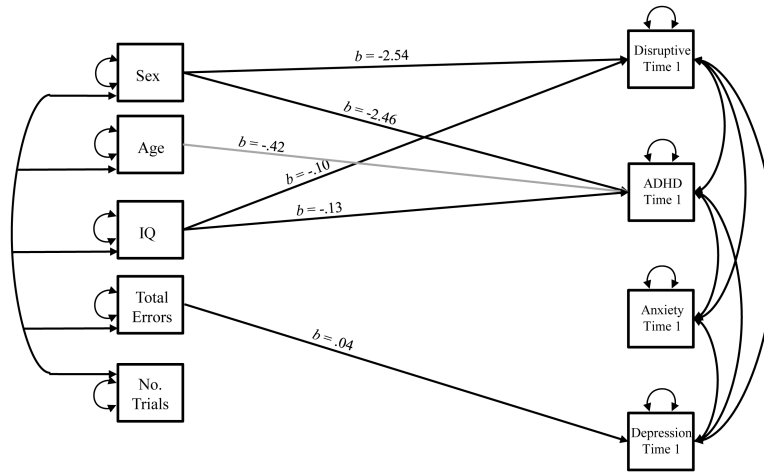
Figure 1.
Model of Concurrent Associations among Time One Psychopathology Symptoms and Total Errors.
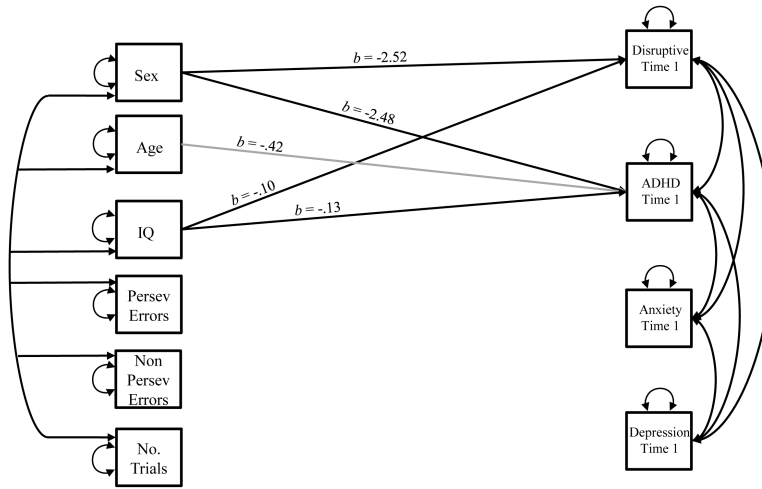
Figure 2.
Model of Concurrent Associations among Time One Psychopathology Symptoms and Error Specificity.
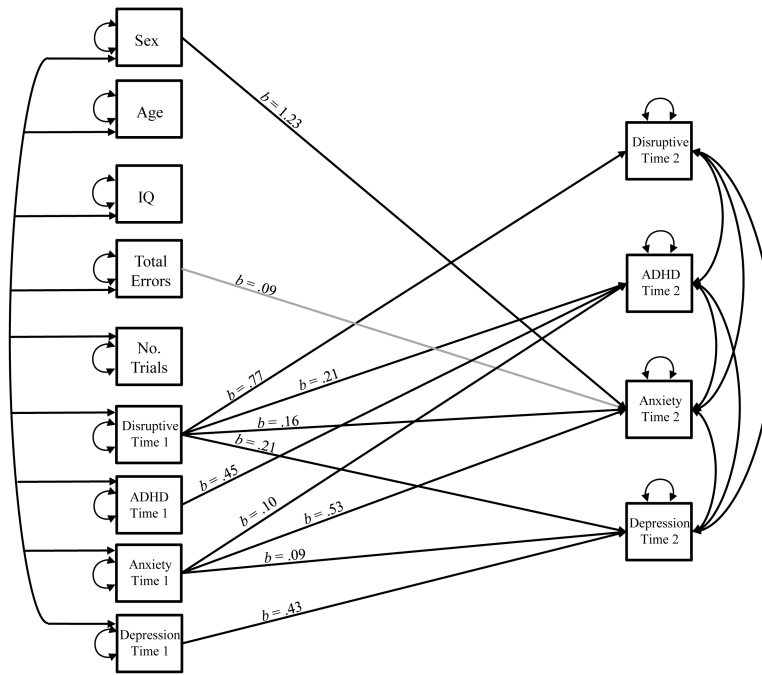
Figure 3.
Longitudinal Model for Predicting Time Two Psychopathology Symptoms from Total Errors.
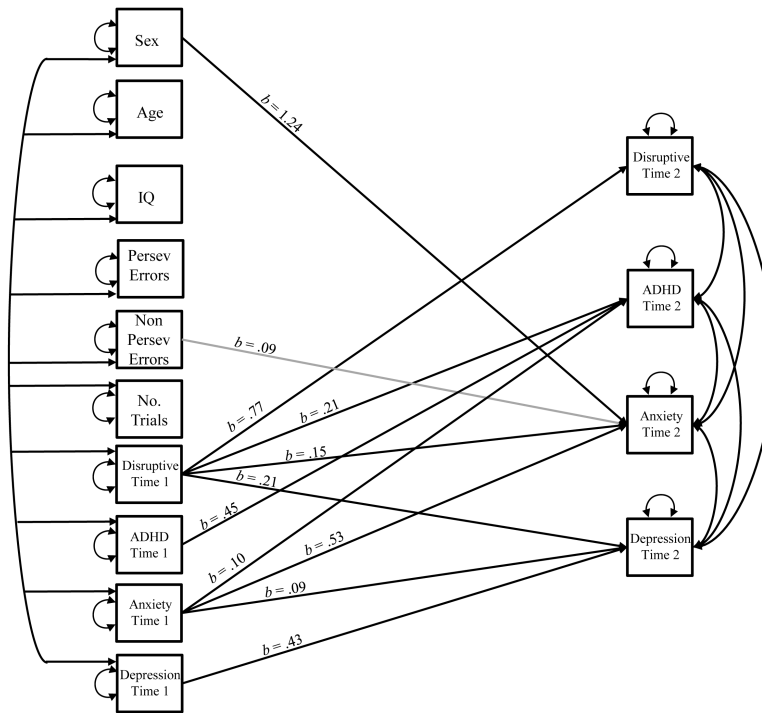
Figure 4.
Longitudinal Model for Predicting Time Two Psychopathology Symptoms from Error Specificity.

Table 1

Mean number (standard deviation) of WCST performance and anxiety, depressive, disruptive, and ADHD symptoms by visit

Measure (Reporter)	Time 1 N = 220	Time 2 N = 177	t	p
WCST				
Total Errors	32.8 (20.5)			
Perseverative Errors	16.0 (11.5)			
Nonperseverative Errors	16.8 (10.8)			
Total Number of Trials	109.6 (19.0)			
Symptom Counts				
DISC Anxiety (M)	6.6 (6.6)	4.8 (5.5)	4.10	<0.01
DISC Anxiety (Y)	10.8 (7.8)	8.0 (6.6)	5.49	<0.01
DISC Depression (M)	5.2 (3.8)	4.7 (4.1)	0.87	0.38
DISC Depression (Y)	6.7 (4.5)	6.4 (4.8)	1.40	0.16
DISC Disruptive (M)	9.9 (5.3)	9.4 (5.5)	1.60	0.11
DISC Disruptive (Y)	8.1 (5.6)	8.9 (6.2)	−2.29	<0.05
DISC ADHD (M)	5.4 (5.0)	4.2 (4.7)	3.42	<0.01
DISC ADHD (Y)	5.4 (4.2)	4.8 (4.2)	1.72	0.09

Note: WCST = Wisconsin Card Sort Task; DISC = Diagnostic Interview Schedule for Children IV. M = Mother; Y = Youth.

Table 2

Correlations among measures of intellectual ability, executive functions, and psychopathology symptoms

		1.	2.	3.	4.	5.
1	KBIT IQ	–				
2	WCST Total Errors	−0.36[*]	–			
3	WCST Perseverative Errors	−0.33[*]	0.92[*]	–		
4	WCST Nonperseverative Errors	−0.33[*]	0.91[*]	0.69[*]	–	
5	WCST Total Number of Trials	−0.32[*]	0.76[*]	0.64[*]	0.75[*]	–
6	DISC Anxiety (M) T1	−0.14[*]	0.10	0.09	0.08	0.04
7	DISC Anxiety (Y) T1	−0.02	0.09	0.10	0.06	0.15[*]
8	DISC Depression (M) T1	−0.15[*]	0.19[*]	0.16[*]	0.18[*]	0.10
9	DISC Depression (Y) T1	0.03	0.04	0.06	0.03	0.08
10	DISC Disruptive (M) T1	−0.22[**]	0.09	0.07	0.11	0.07
11	DISC Disruptive (Y) T1	−0.17[*]	−0.03	−0.02	−0.03	0.03
12	DISC ADHD (M) T1	−0.33[**]	0.20[**]	0.19[**]	0.17[*]	0.21[**]
13	DISC ADHD (Y) T1	−0.29[**]	0.10	0.09	0.09	0.16[*]
14	DISC Anxiety (M) T2	−0.11	0.16[*]	0.13	0.17[*]	0.04
15	DISC Anxiety (Y) T2	−0.11	0.06	0.08	0.03	0.07
16	DISC Depression (M) T2	−0.07	0.12	0.09	0.13	0.11
17	DISC Depression (Y) T2	0.09	−0.04	−0.05	−0.03	0.02
18	DISC Disruptive (M) T2	−0.25[**]	0.11	0.08	0.13	0.13
19	DISC Disruptive (Y) T2	−0.17[*]	−0.02	−0.04	0.01	0.09
20	DISC ADHD (M) T2	−0.28[**]	0.18[*]	0.17[*]	0.16[*]	0.16[*]
21	DISC ADHD (Y) T2	−0.21[**]	0.09	0.06	0.10	0.11

Notes: Correlations flagged

KBIT = Kaufman Brief Intelligence Test; WCST = Wisconsin Card Sorting Test; DISC = Diagnostic Interview Schedule for Children IV, assessed at Time 1 (T1) and at Time 2 (T2); M = mother report; Y = youth report; and ADHD = Attention Deficit Hyperactive Disorder.

[*]
$p < .05$

[**]
$p < .01$

Table 3

Total Errors Predicting Contemporaneous Psychopathology Symptoms

Dependent Variable	Predictor Variable	Est.	β	s.e.	z	p
Disruptive Time 1						
	Sex	−2.538	−0.241	0.688	−3.690	0.000
	Age	−0.112	−0.032	0.241	−0.465	0.642
	IQ	−0.096	−0.209	0.033	−2.879	0.004
	Total Errors	0.004	0.015	0.027	0.146	0.884
	No. of Trials	−0.005	−0.018	0.029	−0.167	0.867
ADHD Time 1						
	Sex	−2.463	−0.250	0.610	−4.040	0.000
	Age	−0.416	−0.128	0.214	−1.944	0.052
	IQ	−0.132	−0.308	0.030	−4.458	0.000
	Total Errors	0.003	0.013	0.024	0.136	0.892
	No. of Trials	0.016	0.061	0.026	0.604	0.546
Anxiety Time 1						
	Sex	0.990	0.075	0.887	1.116	0.264
	Age	−0.117	−0.027	0.311	−0.377	0.707
	IQ	−0.071	−0.123	0.043	−1.643	0.100
	Total Errors	0.047	0.146	0.034	1.360	0.174
	No. of Trials	−0.036	−0.104	0.038	−0.960	0.337
Depression Time 1						
	Sex	−0.450	−0.059	0.509	−0.884	0.377
	Age	0.180	0.071	0.178	1.006	0.314
	IQ	−0.024	−0.073	0.025	−0.989	0.323
	Total Errors	0.043	0.230	0.020	2.163	0.031
	No. of Trials	−0.014	−0.071	0.022	−0.659	0.510

Table 4

Error Specificity Predicting Contemporaneous Psychopathology Symptoms

Dependent Variable	Predictor Variable	Est.	β	s.e.	z	p
Disruptive Time 1						
	Sex	−2.523	−0.240	0.687	−3.671	0.000
	Age	−0.107	−0.031	0.241	−0.445	0.656
	IQ	−0.096	−0.210	0.033	−2.887	0.004
	Perseverative Errors	−0.017	−0.036	0.044	−0.375	0.708
	Nonperseverative Errors	0.031	0.065	0.054	0.576	0.565
	No. of Trials	−0.009	−0.031	0.030	−0.285	0.776
ADHD Time 1						
	Sex	−2.483	−0.252	0.609	−4.080	0.000
	Age	−0.421	−0.130	0.214	−1.969	0.049
	IQ	−0.131	−0.307	0.030	−4.453	0.000
	Perseverative Errors	0.030	0.069	0.039	0.755	0.450
	Nonperseverative Errors	−0.032	−0.071	0.048	−0.664	0.506
	No. of Trials	0.020	0.079	0.027	0.765	0.444
Anxiety Time 1						
	Sex	0.987	0.075	0.887	1.113	0.266
	Age	−0.118	−0.027	0.311	−0.380	0.704
	IQ	−0.071	−0.123	0.043	−1.643	0.100
	Perseverative Errors	0.051	0.088	0.057	0.889	0.374
	Nonperseverative Errors	0.042	0.068	0.070	0.594	0.552
	No. of Trials	−0.036	−0.102	0.039	−0.919	0.358
Depression Time 1						
	Sex	−0.441	−0.058	0.508	−0.867	0.386
	Age	0.183	0.073	0.179	1.026	0.305
	IQ	−0.024	−0.073	0.025	−0.992	0.321
	Perseverative Errors	0.030	0.089	0.033	0.909	0.363
	Nonperseverative Errors	0.060	0.171	0.040	1.499	0.134
	No. of Trials	−0.017	−0.083	0.022	−0.750	0.453

Table 5

Total Errors Predicting Prospective Psychopathology Symptoms

Dependent Variable	Predictor Variable	Est.	β	s.e.	z	p
Disruptive Time 2						
	Sex	0.305	0.028	0.512	0.595	0.552
	Total Errors	−0.011	−0.041	0.020	−0.547	0.585
	No. of Trials	0.029	0.102	0.021	1.379	0.168
	Disruptive Time 1	0.772	0.752	0.059	13.110	0.000
	ADHD Time 1	0.093	0.085	0.067	1.389	0.165
	Anxiety Time 1	0.011	0.013	0.046	0.242	0.809
	Depression Time 1	−0.058	−0.041	0.088	−0.658	0.510
ADHD Time 2						
	Sex	−0.580	−0.063	0.451	−1.288	0.198
	Total Errors	−0.009	−0.039	0.017	−0.502	0.616
	No. of Trials	0.012	0.049	0.019	0.637	0.524
	Disruptive Time 1	0.206	0.236	0.052	3.965	0.000
	ADHD Time 1	0.449	0.480	0.059	7.575	0.000
	Anxiety Time 1	0.102	0.147	0.040	2.552	0.011
	Depression Time 1	0.134	0.112	0.077	1.738	0.082
Anxiety Time 2						
	Sex	1.226	0.109	0.613	2.001	0.045
	Total Errors	0.040	0.147	0.024	1.703	0.089
	No. of Trials	−0.029	−0.098	0.025	−1.142	0.253
	Disruptive Time 1	0.157	0.148	0.071	2.208	0.027
	ADHD Time 1	−0.042	−0.037	0.081	−0.522	0.601
	Anxiety Time 1	0.532	0.627	0.054	9.787	0.000
	Depression Time 1	0.060	0.041	0.105	0.567	0.571
Depression Time 2						
	Sex	0.316	0.038	0.503	0.628	0.530
	Total Errors	−0.018	−0.090	0.019	−0.929	0.353
	No. of Trials	0.025	0.115	0.021	1.197	0.231
	Disruptive Time 1	0.207	0.266	0.058	3.540	0.000
	ADHD Time 1	−0.071	−0.085	0.067	−1.058	0.290
	Anxiety Time 1	0.092	0.148	0.045	2.057	0.040
	Depression Time 1	0.434	0.405	0.086	5.019	0.000

Table 6

Error Specificity Predicting Prospective Psychopathology Symptoms

Dependent Variable	Predictor Variable	Est.	β	s.e.	z	p
Disruptive Time 2						
	Sex	0.333	0.031	0.510	0.653	0.513
	Perseverative Errors	−0.046	−0.096	0.033	−1.403	0.160
	Nonpers. Errors	0.038	0.075	0.040	0.929	0.353
	No. of Trials	0.022	0.076	0.022	0.995	0.320
	Disruptive Time 1	0.769	0.747	0.059	13.081	0.000
	ADHD Time 1	0.108	0.098	0.067	1.600	0.110
	Anxiety Time 1	0.012	0.015	0.045	0.263	0.792
	Depression Time 1	−0.076	−0.054	0.088	−0.866	0.387
ADHD Time 2						
	Sex	−0.580	−0.063	0.451	−1.285	0.199
	Perseverative Errors	−0.010	−0.024	0.029	−0.341	0.733
	Nonpers. Errors	−0.007	−0.017	0.036	−0.202	0.840
	No. of Trials	0.012	0.048	0.019	0.606	0.544
	Disruptive Time 1	0.206	0.235	0.052	3.952	0.000
	ADHD Time 1	0.449	0.481	0.060	7.536	0.000
	Anxiety Time 1	0.102	0.147	0.040	2.552	0.011
	Depression Time 1	0.134	0.111	0.078	1.726	0.084
Anxiety Time 2						
	Sex	1.243	0.111	0.611	2.033	0.042
	Perseverative Errors	0.003	0.006	0.039	0.080	0.936
	Nonpers. Errors	0.092	0.179	0.048	1.899	0.058
	No. of Trials	−0.037	−0.126	0.026	−1.427	0.154
	Disruptive Time 1	0.154	0.145	0.071	2.164	0.030
	ADHD Time 1	−0.029	−0.026	0.081	−0.358	0.720
	Anxiety Time 1	0.534	0.629	0.054	9.850	0.000
	Depression Time 1	0.042	0.029	0.105	0.405	0.686
Depression Time 2						
	Sex	0.316	0.038	0.504	0.627	0.531
	Perseverative Errors	−0.017	−0.047	0.032	−0.524	0.600
	Nonpers. Errors	−0.020	−0.052	0.040	−0.492	0.623
	No. of Trials	0.025	0.117	0.021	1.173	0.241
	Disruptive Time 1	0.207	0.266	0.059	3.531	0.000
	ADHD Time 1	−0.071	−0.085	0.067	−1.050	0.293
	Anxiety Time 1	0.092	0.148	0.045	2.054	0.040
	Depression Time 1	0.434	0.405	0.087	5.014	0.000