An Evaluation of the Specificity of Executive Function Impairment in Developmental Psychopathology

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Objective: Deficits in executive function (EF) are common in neuropsychiatric disorders, but the specificity of these deficits remains unclear. The aim of the present study was to elucidate the pattern of EF impairment across psychopathologies in children and adolescents. Associations among components of EF with dimensions of psychopathology, including an overall psychopathology factor, were assessed.

Method: Participants (8–21 years) were from the Philadelphia Neurodevelopmental Cohort (N = 9,498). Data from a structured clinical screening interview were reduced to 5 dimensional domains using factor analyses: overall psychopathology, anxious-misery, fear, externalizing, and psychosis. EF components of attentional vigilance, response inhibition, conceptual flexibility, and working memory were assessed. Associations of clinical dimensions with general EF ability and with specific EF components were examined.

Results: EF ability showed common and domain-specific associations with clinical symptoms. General EF was directly associated with the general psychopathology, anxious-misery, and psychosis domains but not with the fear or externalizing domains. For the EF subcomponents, differences emerged in the magnitude and direction of the association between components and clinical domains. Poorer EF was typically associated with increased symptoms across clinical domains; however, in some instances, better EF ability was associated with greater symptom burden, particularly in the fear domain.

Conclusion: EF has widespread associations with psychopathology in youth. Findings showed some overlap in the type of EF impairment across clinical phenotypes, as indicated by similar patterns of associations between some clinical symptoms and EF. However, findings also showed domain-specific associations with EF that differed across EF components and clinical domains.

Key words: developmental psychopathology, executive function, adolescence, child development

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A strong link between executive function (EF) impairment and psychopathology appears across the lifespan and is documented in multiple neuropsychiatric disorders[1] at clinical and subclinical manifestations of the disorders. However, questions remain concerning the specificity of these EF deficits.[2] More research is needed to fully understand whether EF impairment is similar or varies across different dimensions of psychopathology and whether the different components of EF have comparable contributions within and across clinical domains. To address these questions, performance on multiple EF components must be directly compared across multiple clinical domains.[3] Thus, using the Philadelphia Neurodevelopmental Cohort (PNC), a large community-based youth sample, the present study aimed to elucidate the type and degree of EF dysfunction associated with multiple dimensions of psychiatric symptoms. To do this, the present study used bi-factor models to examine EF associations with a general psychology factor[4] and domain-specific dimensions of psychopathology.

Although there are alternative theoretical and empirical approaches to the study of EF,[5,6] it is widely accepted that EF is a hierarchical construct that encompasses component processes. These processes include the ability to orient and sustain attention (i.e., attention vigilance), shift attention and think flexibly (i.e., attentional/conceptual flexibility), inhibit responses, and maintain and update goal-related information in working memory. In children and adults, these cognitive processes are distinct but interrelated components of EF.[7,8] Therefore, understanding the role of separate EF components and the common EF ability that spans across components (i.e., general EF) is a critical step in better understanding EF deficits in psychopathology.

To date, few studies have investigated the specificity of EF deficits across multiple clinical domains.[9-13] Findings from meta-analyses suggest that the type and level of EF deficits are comparable for many psychiatric disorders.[2,14,15] Specifically, the effect sizes for EF impairment relative to healthy comparisons range from moderate to large for attention-deficit/hyperactivity disorder (ADHD),

Supplemental material cited in this article is available online.

schizophrenia, and bipolar disorder and from small to large for major depression. However, the extant work directly comparing EF performance across neuropsychiatric disorders yields mixed findings.[16-22] For example, some studies document worse EF performance in youth with ADHD compared with youth with anxiety disorders[19]; however, other work finds no such EF differences.[20]

Some recent work used structural equation modeling to form dimensions of psychopathology. This work suggests that general EF dysfunction is a nonspecific correlate of developmental psychopathology.[11-13] For example, Martel *et al.*[12] found that general EF was negatively associated with a general psychopathology factor but not with domain-specific externalizing, fear, or distress factors. Shanmugan *et al.*,[13] using a subset of the PNC cohort, reported robust negative associations between a general psychopathology factor and behavioral and neuroimaging indices of working memory; however, disorder-specific EF perturbations also emerged for psychosis spectrum, externalizing, and anxious-misery factors. Thus, further work is needed to better understand the common and unique associations between EF and specific domains of psychopathology.

Growing evidence suggests that within a given clinical phenotype different components of EF have unique patterns of associations with the disorder.[23-26] For ADHD, deficits in certain EF components are larger (i.e., response inhibition) than others (i.e., attention shifting).[27] This suggests that specific EF components might be better markers of certain psychopathologies. Although less work has examined EF deficits in anxiety, prior studies suggest that different patterns of EF impairment occur in subtypes of anxiety (e.g., generalized anxiety disorders, social phobia, posttraumatic stress disorders).[18,20,28,29] Notably, some work found links between higher EF ability and anxiety symptomatology in children and adults.[13,23,30,31] For example, White *et al.*[23] found that enhanced inhibition predicted higher levels of anxiety in children at temperamental risk for anxiety.

In summary, it is important to understand the type and degree of EF dysfunction that is common and unique across domains of psychopathology. To address these issues, the present study directly assessed associations among multiple components of EF and multiple domains of psychopathology using a bi-factor model with a general psychopathology factor and domain-specific factors (i.e., anxious-misery, fear, externalizing, and psychosis spectrum). We examined whether impairment in a general EF factor was a disorder-specific or trans-diagnostic factor associated with general psychopathology. Next, we examined whether different EF components differentially related to the different clinical domains.

METHOD

Participants

The PNC is a community-based sample of youth. Enrollment was based at the Center of Applied Genomics at the Children's Hospital of Philadelphia (CHOP) and the Brain Behavior Laboratory at the University of Pennsylvania (detailed in Calkins *et al.*[32,33] and Satterthwaite *et al.*[34]). All study participants previously consented for genomic studies while seeking pediatric services at the CHOP. From this pool of approximately 50,000, 18,344 individuals who had consented for recontact and met the inclusion criteria were randomly selected (with stratification for sex, age, and ethnicity) for participation in the PNC study. Exclusionary criteria were lack of English proficiency and medical conditions that would significantly affect brain function or interfere with the ability to complete study procedures. Of note, participants were not recruited from psychiatric services; thus, the sample is not enriched with psychiatric treatment-seeking individuals.

A total of 9,498 youths enrolled in the study; they were 8 to 21 years old at the time of enrollment. Complete and valid assessment across the entire EF battery was obtained for 8,856 participants. For the analyses that relied only on data from one of the EF tasks, more participants were included in the given analyses: Penn Continuous Performance Task (PCPT; n = 9,145), Penn Conditional Exclusion Test (PCET; n = 9,188), and Penn Letter N-Back Test (N-back; n = 9,004). In the sample with valid data from at least 1 EF task, the mean age of participants was 14.23 years (standard deviation [SD] 3.64). More information on the sample's age distribution is provided in Supplement 1 (available online). In the sample, 52% were girls; 57% were Caucasian; 32% were African-American; and 11% were of mixed or other race. Years of maternal and paternal education were 14.5 (SD 2.4) and 14.3 (SD 2.7), respectively.

After the study procedures were explained, consent was obtained from participants at least 18 years old; assent and parent/legal guardian permission were obtained for participants younger than 18 years of age. All study procedures were approved by the CHOP and University of Pennsylvania institutional review boards.

Clinical Assessment

Psychopathology was assessed through a computerized structured clinical screening interview, which was administered by trained clinical coordinators as previously detailed.[32] Interviews were conducted in the laboratory or at the participant's home, depending on participant preference. The clinical assessment was adapted from the National Institute of Mental Health Genetic Epidemiology Research Branch Schedule for Affective Disorders and Schizophrenia[35] and included assessment for mood (major depressive episode, manic episode), anxiety (generalized anxiety disorder, social anxiety disorder, specific phobia, separation anxiety disorder, panic disorder, posttraumatic stress disorder, obsessive-compulsive disorder, agoraphobia), behavior (oppositional defiant disorder, conduct disorder), ADHD, eating (anorexia, bulimia), psychosis spectrum, and suicidal thoughts and behavior. Each section included a screen of several questions assessing disorder-related symptoms. Additional psychosis-related items (n = 12) from the Prevention through Risk Identification, Management, and Education (PRIME) Screen–Revised,[36] to assess subthreshold positive symptoms, and the Scale of Prodromal Symptoms (SOPS; n = 6) from the Structured Interview for Prodromal Syndromes (SIPS),[37] to assess negative/disorganized symptoms, were included in the interview.[33] The disorder-related screen items, additional psychosis items, and 4 general mental health treatment questions were used in the bi-factor model of psychopathology (112 items). Information related to frequency, duration, level of impairment, and distress associated with each disorder also was collected during the interview; however, this information was not included in the bi-factor model. The interviews were administered to probands (participants 11–21 years old) and collaterals (parent or legal guardian of participants 8–17 years old).

Executive Function Assessment

The EF tasks were administered as part of a larger cognitive assessment battery, the Penn Computerized Neurocognitive Battery.[38] Three EF tasks were used to assess the following EF components: response inhibition, attentional vigilance, conceptual flexibility, and working memory performance. A general EF score also was created by averaging the *z*-scored EF components (attentional vigilance, response inhibition, conceptual flexibility, and working memory). Additional variables were generated using a signal detection approach. More information about EF tasks and scoring is available in Supplement 1 (available online).

Penn Continuous Performance Task. The PCPT measured response inhibition and attentional vigilance (aka sustained attention). Stimuli were numbers or letters (target) or non-letters or non-numbers (foil). Participants were told to respond to targets by button press (go trials) and refrain from responding to foil presentations (no-go trials). A response inhibition score was created based on the number of true negatives (i.e., accuracy on no-go/foil trials). An attentional vigilance measure was calculated based on the number of true positives (i.e., accuracy on go/target trials).

Penn Conditional Exclusion Test. The PCET was used as a measure of abstraction and conceptual flexibility (often referred to as cognitive or attentional flexibility). Four stimuli were presented simultaneously, and participants needed to determine which stimulus was the "odd man out" based on various visual features of the stimuli (e.g., shape, size). Feedback ("correct" or "incorrect") was provided after each trial, from which participants needed to deduce the stimulus exclusion rule. The rule switched throughout the task, requiring abstraction abilities to discover the rule and conceptual flexibility for shifts between rule sets. A performance score was created to reflect overall correct responses and total learning.

Penn Letter N-Back Test. For this working memory task, a series of letters was presented on the screen. Participants were required to respond to a given letter across 3 conditions (i.e., working memory loads): 0-back (respond when letter X appears), 1-back (respond when the current letter is the same as the previous letter), and 2-back (respond when the current letter is the same as the letter before the previous letter). A working memory score was created based on overall accuracy.

Signal Detection-Based Variables. Additional variables were generated using a signal detection approach. Prior work suggests that response bias (i.e., tendency to be cautious or impulsive) can influence standard accuracy-based calculations[39] and vary across psychopathologies.[40] Thus, indices of discrimination accuracy (P_r) and response bias (B_r) were calculated for the PCPT and N-back.[39,41,42] Additional information on these variables is provided in Supplement 1 (available online).

Data Analysis

Structural equation modeling assessed the relations between EF and psychopathology. There were 5 main models (i.e., general EF, vigilance, response inhibition, conceptual flexibility, and working memory). Clinical items from the proband report (or collateral report for children <11 years old, for whom proband data were not available) were modeled as a confirmatory bi-factor model,[43] which allows each item to load not only on a specific clinical factor (e.g., psychosis spectrum), but also on an overall ("general") psychopathology factor. The existence of the general factor allows the specific factors to be modeled as orthogonal to each other and to the general factor. We modeled 4 specific factors based on previous work with these data[13] and on theory. Krueger[44] demonstrated that mental disorders (not including psychosis) can be grouped into 3 categories—externalizing, anxious-misery, and fear—and we had additional items relating to psychosis, for a total of 4 specific factors. Preliminary exploratory item-factor analysis of these data strongly supported this decision, with 4 obliquely rotated factors clearly representing the 4 specific factors listed earlier.

This measurement model was estimated simultaneously with the effects of interest. That is, the observed independent variables of interest (i.e., general EF or one of the subcomponents) was allowed to relate to the latent variables in the measurement model, and these effects were estimated simultaneously with the measurement model itself. The EF variable (general EF or subcomponent) was modeled separately, as well as with the following covariates: sex, age, and race. Interactions among EF, sex, and age also were included. In each model, observed variables (including interactions) were allowed to correlate with each other and relate to all latent variables in the measurement model. All models were estimated in Mplus (http://www.statmodel.com/company.shtml) using the mean- and variance-adjusted weighted least squares (wlsmv) estimator.[45] Model fit was assessed using cutoff criteria suggested in prior work.[46,47] Factor loadings from the general EF model appear in the supplemental material (Table S1, available online). Model fits and results for the 4 models using signal detection variables are presented in the supplemental material (Table S2, available online). To correct for type 1 error inflation, the p value was set to less than or equal to .001.

RESULTS

Model Fit

Figure 1 shows the structural equation model with associations between general EF and the bi-factor model of psychopathology. Fit of all models was acceptable (often excellent). Specifically, for the model including overall EF, the comparative fit index was equal to 0.96, the Tucker-Lewis index was equal to 0.96, and the root mean-square error of approximation was equal to 0.027 ± 0.001. The models containing the EF subcomponents of attentional vigilance, response inhibition, and working memory had the following fit indices: comparative fit index 0.95, 0.96, and 0.94, respectively; Tucker-Lewis index 0.95, 0.95, and 0.94, respectively; and root mean-square error of approximation 0.027 ± 0.001, 0.027 ± 0.001, and 0.027 ± 0.001, respectively. Owing to convergence problems with the mean- and variance-adjusted weighted least squared estimator in the conceptual flexibility model, this model was estimated with the Bayes estimator, which does not provide conventional fit indices.

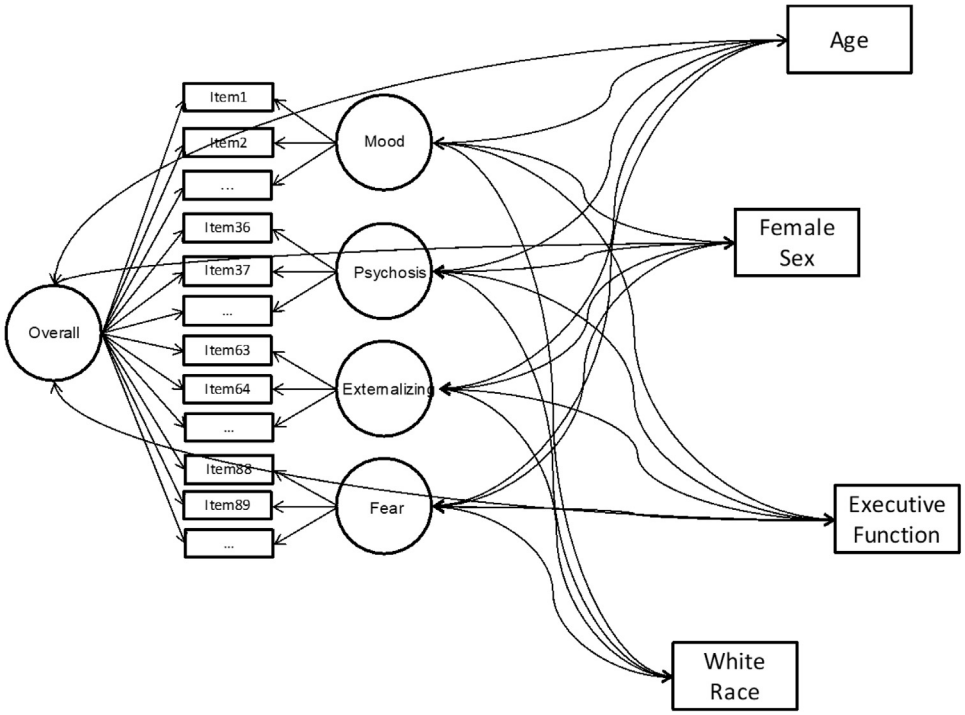
Associations Between General EF and Clinical Domains

Table 1 presents specific associations between general EF and the clinical factors from the bi-factor model. Total EF was significantly related to symptom levels for the general psychopathology, anxious-misery, and psychosis domains. For these domains, lower general EF scores were associated with increased symptom levels. This pattern of association was strongest for the psychosis domain. General EF was not directly related to fear or externalizing symptoms.

Associations Between EF Components and Clinical Domains

The magnitude of relations among the EF components and symptom levels differed across the 5 clinical domains

FIGURE 1 Example of the structure equation model with general executive function used to assess associations between executive function and the bi-factor model of psychopathology. *Note:* Interactions among executive function, age, and sex are not shown.



(Table 1). With only a few exceptions, each of the separate EF components significantly predicted symptom levels across the 5 domains; however, the strength and direction of these associations differed across domains. None of the 4 EF components had significant associations with all 5 clinical domains. Anxious-misery and psychosis were the only domains that had significant associations with all EF components. The externalizing and fear domains had the fewest associations with EF components. Although most EF-symptom associations were negative (i.e., poorer EF was related to higher symptom levels), some positive associations emerged. For example, higher attentional vigilance and working memory abilities were related to higher symptom burden in the fear domain. Surprisingly, conceptual flexibility also showed positive associations with symptoms in the psychosis and general psychopathology domains.

Interactions Between EF and Age and Sex

As presented in Table 1, many significant sex-by-EF and age-by-EF interactions emerged across the 5 clinical domains. As such, only the largest interactions and those within the general psychopathology model are highlighted in the text. For the significant EF-by-sex interactions for general psychopathology, high EF was associated with fewer general psychopathology symptoms for girls compared with boys. Large interactions emerged for the anxious-misery domain. Across the EF factors, better EF was associated with fewer anxious-misery symptoms in boys than in girls, and in some cases (i.e., general EF and response inhibition), better EF in girls was associated with more anxious-misery symptoms. For the age interactions, the relations of poor general EF, response inhibition, and working memory with higher general psychopathology symptoms tended to be stronger for younger children. Also, the positive association that emerged between conceptual flexibility and general psychopathology was present only in younger children. Large age-by-EF interactions emerged for the fear domain, showing that higher EF, across the subcomponents, tended to be related to higher fear symptoms in younger children, but in older youth, higher EF was related to lower fear symptoms.

DISCUSSION

Most neuropsychiatric disorders, and even subclinical manifestations of disorders, are associated with EF impairments; however, the specificity of these associations is unclear. To help delineate the role of EF impairment across clinical phenotypes, the present study used a large community sample to assess associations between multiple EF components and different domains of psychopathology, including an overall psychopathology factor. We found overlap in the type of EF impairment across clinical symptoms, as indicated by the significant associations between EF and overall psychopathology and similarities in some of the patterns of associations between EF and symptom levels across domain-specific clinical factors. However, EF also showed significant domain-specific associations that differed across EF components and clinical domains.

TABLE 1 Executive Function (EF) Components (Vigilance, Response Inhibition, Conceptual Flexibility, Working Memory) Predicting Clinical Domains

	Overall Psychopathology		Anxious Misery		Fear		Externalizing		Psychosis	
	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*
General EF										
EF	**−0.172**	.000	**−0.292**	.000	−0.075	.032	−0.050	.134	**−0.349**	.000
Age	**0.255**	.000	**−0.138**	.000	**−0.114**	.000	**−0.111**	.000	**−0.198**	.000
Sex	**−0.094**	.000	**0.326**	.000	**0.315**	.000	**−0.076**	.000	**0.067**	.000
Race	**−0.234**	.000	**0.222**	.000	0.025	.074	0.015	.284	0.000	.989
EF × age	**0.569**	.000	**−0.418**	.000	**−0.586**	.000	**−0.444**	.000	**−0.219**	.000
EF × sex	**−0.425**	.000	**0.913**	.000	**0.683**	.000	**0.391**	.000	**0.582**	.000
ATT										
ATT	−0.080	.085	**−1.007**[a]	.000	**0.605**	.000	−0.086	.152	**−0.394**	.000
Age	**0.294**	.000	**−0.244**	.000	0.033	.146	**−0.171**	.000	**−0.184**	.000
Sex	**−0.096**	.000	**0.345**	.000	**0.279**	.000	**−0.07**	.000	**0.084**	.000
Race	**−0.212**	.000	**0.17**	.000	0.011	.481	−0.02	.143	−0.012	.416
ATT × age	**0.409**	.000	**0.45**	.000	**−1.122**[a]	.000	**−0.325**	.000	−0.094	.153
ATT × sex	**−0.343**	.000	**0.789**	.000	**0.427**	.000	**0.401**	.000	**0.478**	.000
RI										
RI	**−0.128**	.000	**−0.472**	.000	0.093	.019	−0.086	.052	**−0.401**	.000
Age	**0.291**	.000	**−0.203**	.000	**−0.118**	.000	**−0.143**	.000	**−0.236**	.000
Sex	**−0.103**	.000	**0.319**	.000	**0.311**	.000	**−0.060**	.000	**0.062**	.000
Race	**−0.206**	.000	**0.190**	.000	−0.010	.475	−0.017	.194	−0.024	.106
RI × age	**0.466**	.000	−0.121	.008	**−0.602**	.000	**−0.366**	.000	−0.080	.097
RI × sex	**−0.407**	.000	**0.835**	.000	**0.598**	.000	**0.375**	.000	**0.544**	.000
CF										
CF	**0.128**	.000	**−0.228**	.000	−0.056	.061	**−0.292**	.000	**0.166**	.000
Age	**0.223**	.000	**0.147**	.000	**−0.019**	.000	**−0.092**	.000	**−0.045**	.000
Sex	**−0.067**	.000	**0.238**	.000	**0.220**	.000	**−0.145**	.000	−0.014	.167
Race	**−0.185**	.000	**0.176**	.000	**−0.058**	.000	**−0.026**	.000	**−0.107**	.000
CF × age	**−0.146**	.000	**0.170**	.000	−0.006	.435	**0.202**	.000	**−0.194**	.000
CF × sex	−0.007	.305	**0.067**	.000	0.038	.007	0.017	.124	0.014	.229
WM										
WM	**−0.282**	.000	**−1.294**[a]	.000	**1.167**[a]	.000	−0.196	.006	**−0.547**	.000
Age	**0.279**	.000	**−0.251**	.000	0.083	.003	**−0.184**	.000	**−0.205**	.000
Sex	**−0.073**	.000	**0.340**	.000	**0.222**	.000	**−0.072**	.000	**0.070**	.000
Race	**−0.228**	.000	**0.123**	.000	0.052	.005	−0.018	.203	−0.035	.032
WM × age	**0.583**	.000	**0.680**	.000	**−1.567**[a]	.000	**−0.228**	.000	0.065	.389
WM × sex	**−0.331**	.000	**0.873**	.000	**0.321**	.000	**0.394**	.000	**0.531**	.000

Note: Boldface indicates significant effects (p ≤ .001). ATT = attentional vigilance; CF = conceptual flexibility; RI = response inhibition; WM = working memory.
[a]*Although model estimation terminated normally, coefficients with an absolute value larger than 1.0 were possible because of non-positive–definite residual variance and covariance matrices.*

The extant work linking EF and latent dimensions of psychopathology finds strong associations with general psychopathology rather than domain-specific factors.[11,12] In the present study, all EF components were significantly associated with overall psychopathology, except for attentional vigilance. This finding supports prior work suggesting that the type of EF impairment linked to overall psychopathology is widespread and involves multiple components of EF. However, in the present study, EF also was associated with several domain-specific symptoms. Indeed, direct effects between EF factors and symptoms were detected for all domain-specific factors. Moreover, the magnitude of these domain-specific associations was often larger than associations between EF and the general psychopathology factor. For example, poor general EF was more strongly related to anxious-misery and psychosis-spectrum symptoms than to general psychopathology symptoms.

The present study also found differences in the strength of associations between the specific EF components and symptom levels within and across clinical domains. For example, for anxious-misery and fear symptoms, attentional vigilance and working memory had more robust relations to symptoms than response inhibition or conceptual flexibility. Conversely, for the general psychopathology and psychosis

domains, the magnitude of effects across EF subcomponents was fairly similar. Taken together, these findings support previous work[23-25,27] that suggests EF components differentially relate to neuropsychiatric symptom clusters. Moreover, these findings suggest that it is important for future work to assess different EF components, because the strength of association between clinical symptoms and EF components varied. Had only response inhibition been used as a proxy for EF in the present study, EF impairment would have appeared relatively similar across clinical domains. However, had working memory been used as a proxy for EF, more pronounced differences between the domains would have emerged.

Although most EF components had negative associations between EF ability and symptom levels, some positive associations emerged. For example, in the fear domain, better ability in attentional vigilance and working memory was linked to higher fear symptoms. Fear symptoms also were related to a cautious response style on the continuous performance task. Positive, albeit weak, associations also emerged between conceptual flexibility and symptoms in the general psychopathology and psychosis domains. Prior work has linked anxiety-related symptoms to enhanced EF abilities, including better performance monitoring, working memory, and response inhibition.[13,23,30,48-50] Thus, increased ability of some EF components might serve to cause or maintain certain clinical symptoms related to fear. Alternatively, at least in a community sample, anxiety or other subthreshold symptoms might improve certain EF functions[51] or task motivation. It is worth noting that the positive associations found within the fear domain did not emerge for the anxious-misery domain. Thus, although the anxious-misery domain (reflecting negative mood and intrusive negative thoughts) and the fear domain (reflecting nervousness and fear across multiple contexts) are often considered highly related constructs, notable EF differences were detected in the present study. These differences add to a growing literature showing that distress-related and fear-related disorders differ on multiple cognitive and affective processes.[52-54]

Understanding how the relations between EF and psychopathology can differ by sex and age is important, as these factors can significantly affect processes underlying psychopathology and EF.[55,56] We found that the EF-symptom relation differed as a function of age and sex; however, the direction of these interactions differed across clinical domains and EF components. Of note, when positive associations did emerge between EF and symptoms, interaction effects suggested that the pattern was present only for younger children; in older youth, EF had little effect on symptom levels or higher EF was related to lower fear symptoms. For sex differences, within the general psychopathology factor, a pattern emerged in which higher EF tended to be more protective (related to lower symptoms) in girls than in boys. However, for the domain-specific factors, higher EF tended to be more protective in boys. For the age and sex interactions, the causal nature of these interactions is unclear. It will be important for future longitudinal work to tease apart the directionality of these associations.

There are several limitations to the present study. The study used uncorrelated dimensional measures to isolate distinct clinical phenotypes and decrease issues of comorbidity. Although this approach enabled better examination of specificity of EF deficits across clinical domains, the results might not hold when comparing clinical groups according to their diagnostic categories or in instances of high comorbidity across clinical domains. Another limitation concerns the cross-sectional nature of the study; we cannot ascertain whether EF dysfunction is a consequence of psychopathology or a developmental risk factor or both. Prior work suggests that the directionality of such associations might differ among EF components.[57,58] In addition, the level of variance explained by the domain-specific factors is minimal, which could influence the present pattern of results. Moreover, we assessed EF in neutral contexts; associations between EF and psychopathology might differ when EF is assessed during emotional contexts.[59]

These limitations notwithstanding, our findings highlight important differences in the strength and direction of associations between EF, general EF and specific components, and psychopathology across development. These findings suggest that future work should consider the differences across EF components, because the strength of association between clinical symptoms and EF components differed. Moreover, the present findings suggest that future treatment and prevention work might benefit by targeting deficits in specific EF components (e.g., working memory) for a given disorder (e.g., depression). &

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SUPPLEMENT 1

Participant Age

Participants were 8 to 21 years old at the time of recruitment (mean age 14.23 years, standard deviation [SD] 3.64; kurtosis and skewness were −1.14 and 0.016, respectively). There were 2,358 participants 8 to 10 years old (mean 9.44, SD 0.84), 2,135 participants 11 to 13 years old (mean 12.49, SD 0.85), 2,323 participants 14 to 16 years old (mean 15.48, SD 0.86), 2,162 participants 17 to 19 years old (mean 18.34, SD 0.8), and 434 participants at least 20 years old (mean 20.72, SD 0.5).

Executive Function Tasks From the Penn Computerized Neurocognitive Battery

Penn Continuous Performance Task. On this task, the stimuli consisted of 7-segment displays (e.g., similar to the display on a digital clock). Stimuli were presented on the screen for 300 ms followed by a blank screen for 700 ms. Total allowable response time per trial was 1,000 ms. The task was presented in 2 blocks with a total of 144 trials; targets were numbers during block 1 and letters for block 2. Several behavioral measures were generated from the Penn Continuous Performance Task (PCPT; response inhibition and attentional vigilance). Response inhibition represents the participant's overall ability to appropriately inhibit a motor response, and attentional vigilance represents the ability to focus attentional resources on specific stimuli. Of note, the PCPT attentional vigilance score loads highly on a general executive function factor.[1]

Penn Conditional Exclusion Test. On this task, the stimuli consisted of shapes that could vary across several dimensions. These dimensions were the shape (e.g., square, star), size (e.g., large, small), or the thickness of the lines that formed the shape (e.g., thick, thin). On each trial, the participant was presented with 4 shapes and needed to determine which shape was the "odd man out" (i.e., the dimension by which all but 1 of the stimuli matched). The rule (i.e., dimension by which the 1 stimulus "differed") changed after 10 consecutive correct trials. Participants were not informed of the rule switch and needed to learn the new rule through the feedback presented after every trial. The task consisted of a maximum of 144 trials, with a total of 48 possible trials for each new learning rule. A performance score was created to reflect overall correct responses and total rule learning by multiplying the overall correct responses by the number of rules learned (a value of 1 was added to the number of rules learned to avoid multiplication by 0 when no rules were learned).

Penn Letter N-Back Test. On this task, stimuli were presented for 500 ms, with an interstimulus interval of 2,500 ms. A total number of 90 trials were presented.

Signal Detection Measures. P_r reflects the ability to accurately detect whether the trial was a "go" or "no-go" and is conceptually similar to d' but does not require a correction in instances of small numbers (i.e., no errors of commission).[2] For B_r, negative values reflect a cautious response style (i.e., bias to withhold response), and positive values reflect an impulsive response style (i.e., bias to execute response). P_r was calculated as the hit rate ([0.5 + correct targets trials]/[1 + total target trials]) minus the false alarm rate ([.05 + incorrect foil trials]/[1 + total foil trials]). B_r was calculated with the formula (false alarm rate/[1 − P_r]) − 0.05. Measures of P_r and B_r are mathematically independent.

Bi-Factor Model

Model Loadings and Variance Explained. The values for Ω_H, $\Omega_{anxious\text{-}misery}$, Ω_{fear}, $\Omega_{externalizing}$, and $\Omega_{psychosis}$ are 0.86, 0.04, 0.03, 0.03, and 0.02, respectively. The sum of squared loadings for general psychopathology, anxious-misery, fear, externalizing, and psychosis are 40.9, 7.3, 6.9, 7.2, and 5.6, respectively. Table S2 presents the 25 highest loading items on each of the clinical domains.

Associations Between Psychopathology and Signal Detection Variables. The models containing the signal-detection theory EF subcomponents of PCPT discrimination accuracy, PCPT response bias, and Penn Letter N-Back Test (N-back) discrimination accuracy had the following fit indices: comparative fit index equal to 0.96, 0.90, 0.94, and 0.90, respectively; Tucker-Lewis Index equal to 0.96, 0.89, and 0.94, respectively; root mean-square error of approximation equal to 0.027 ± 0.001, 0.027 ± 0.001, and 0.027 ± 0.001, respectively. For the N-back response bias model, a Bayes estimator was used to achieve convergence. As such, model fit indices are not available for this model.

Results from these models (Table S2) showed that for all clinical domains except for externalizing and fear domains, lower discrimination accuracy across the 2 tasks predicted higher symptoms. For the fear domain, higher discrimination accuracy predicted higher symptoms, but only on the N-back task. Interestingly, for the response bias measures, the only significant main effect emerged in the fear domain. A negative response bias (i.e., cautious bias) on the PCPT predicted heightened fear symptoms.

SUPPLEMENTAL REFERENCES

1. Moore TM, Reise SP, Gur RE, Hakonarson H, Gur RC. Psychometric properties of the Penn Computerized Neurocognitive Battery. Neuropsychology [Internet]. 2015;29:235-246.
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TABLE S1 Factor Loadings From the General Execution Function Bi-Factor Model

General Psychopathology		Anxious-Misery		Fear		Externalizing		Psychosis	
Item	**Loading**	**Item**	**Loading**	**Item**	**Loading**	**Item**	**Loading**	**Item**	**Loading**
MAN	0.84	GAD	0.83	SOC	0.72	ADD	0.81	PSY	0.81
MAN	0.84	GAD	0.82	SOC	0.70	ADD	0.81	PSY	0.77
DEP	0.83	OCD	0.59	SOC	0.70	ADD	0.71	PSY	0.63
MAN	0.83	OCD	0.58	SOC	0.67	ADD	0.68	PSY	0.62
MAN	0.83	PAN	0.57	AGR	0.64	ADD	0.68	PSY	0.58
OCD	0.83	OCD	0.55	AGR	0.63	ODD	0.64	PSY	0.57
MAN	0.82	OCD	0.54	AGR	0.62	ADD	0.63	PSY	0.57
OCD	0.82	OCD	0.51	AGR	0.60	ODD	0.63	PSY	0.56
MAN	0.81	DEP	0.51	SOC	0.59	ADD	0.61	PSY	0.56
OCD	0.80	PAN	0.51	AGR	0.58	ADD	0.61	PSY	0.55
OCD	0.80	OCD	0.50	AGR	0.55	ODD	0.58	PSY	0.54
DEP	0.79	DEP	0.50	AGR	0.54	ADD	0.56	PSY	0.49
OCD	0.77	OCD	0.48	PHB	0.50	ODD	0.53	PSY	0.49
DEP	0.76	PAN	0.48	SEP	0.49	CD	0.52	PSY	0.46
MAN	0.74	OCD	0.46	PHB	0.47	ODD	0.50	PSY	0.46
PAN	0.73	OCD	0.45	SEP	0.43	CD	0.43	PSY	0.43
DEP	0.73	OCD	0.44	PHB	0.43	CD	0.42	PSY	0.35
PSY	0.72	OCD	0.44	PHB	0.42	CD	0.39	PSY	0.27
PSY	0.70	OCD	0.41	AGR	0.40	TX	0.38	PSY	0.23
PSY	0.70	OCD	0.41	PHB	0.40	CD	0.37	PSY	0.21
ODD	0.70	SUI	0.39	PHB	0.38	CD	0.36	PSY	0.06
OCD	0.70	OCD	0.39	SEP	0.38	CD	0.24	MAN	0.03
PSY	0.69	OCD	0.38	PHB	0.36	TX	0.21	MAN	−0.02
SUI	0.69	OCD	0.36	PHB	0.33	PSY	0.21	MAN	−0.03
OCD	0.69	PSY	0.36	SEP	0.30	CD	0.19	MAN	−0.04

Note: Loading refers to estimated factor loadings. ADD = attention hyperactivity; AGR = agoraphobia; CD = conduct; DEP = depression; GAD = generalized anxiety disorder; MAN = mania; OCD = obsessive-compulsive disorder; ODD = oppositional defiant; PAN = panic; PHB = phobia; PSY = psychosis; SEP = separation; SOC = social anxiety; SUI = suicide; TX = treatment.

TABLE S2 Signal Detection Variable (Discrimination Accuracy and Response Bias) Predicting Clinical Domains

	Overall Psychopathology		Anxious-Misery		Fear		Externalizing		Psychosis	
	β	*p*	β	*p*	β	*p*	β	*p*	β	*p*
PCPT P_r										
P_r	**−0.110**	.000	**−0.249**	.000	−0.095	.002	−0.046	.105	**−0.325**	.000
Age	**0.294**	.000	**−0.205**	.000	**−0.183**	.000	**−0.154**	.000	**−0.252**	.000
Sex	**−0.112**	.000	**0.331**	.000	**0.328**	.000	**−0.059**	.000	**0.084**	.000
Race	**−0.229**	.000	**0.242**	.000	0.032	.024	0.013	.351	0.023	.131
$P_r \times$ age	**0.518**	.000	**−0.402**	.000	**−0.515**	.000	**−0.433**	.000	**−0.229**	.000
$P_r \times$ sex	**−0.430**	.000	**0.859**	.000	**0.654**	.000	**0.394**	.000	**0.571**	.000
PCPT B_r										
B_r	0.032	.013	0.010	.493	**−0.093**	.000	0.016	.212	−0.026	.062
Age	**0.274**	.000	**0.125**	.000	−0.036	.012	**−0.125**	.000	**−0.079**	.000
Sex	0.011	.403	**0.225**	.000	**0.222**	.000	**−0.170**	.000	**−0.057**	.000
Race	**−0.181**	.000	**0.190**	.000	**−0.094**	.000	**−0.071**	.000	**−0.065**	.000
$B_r \times$ age	0.038	.002	0.013	.400	**−0.088**	.000	0.012	.368	−0.025	.066
$B_r \times$ sex	0.028	.028	0.003	.837	**−0.061**	.000	0.032	.014	−0.039	.006
N-back P_r										
P_r	**−0.181**	.001	**−1.025**[a]	.000	**0.697**	.000	−0.138	.035	**−0.465**	.000
Age	**0.288**	.000	**−0.228**	.000	0.015	.494	**−0.161**	.000	**−0.193**	.000
Sex	**−0.094**	.000	**0.357**	.000	**0.273**	.000	**−0.060**	.000	**0.083**	.000
Race	**−0.229**	.000	**0.149**	.000	0.044	.006	−0.003	.818	−0.021	.178
$P_r \times$ age	**0.499**	.000	**0.409**	.000	**−1.162**[a]	.000	**−0.307**	.000	−0.021	.766
$P_r \times$ sex	**−0.356**	.000	**0.863**	.000	**0.423**	.000	**0.380**	.000	**0.510**	.000
N-back B_r										
B_r	0.135	.002	−0.032	.282	−0.051	.172	0.126	.008	0.068	.148
Age	**0.273**	.000	**0.147**	.000	−0.022	.075	**−0.121**	**.000**	**−0.044**	**.001**
Sex	**−0.064**	.000	**0.237**	.000	**0.219**	**.000**	**−0.137**	**.000**	−0.007	.308
Race	**−0.189**	.000	**0.174**	.000	**−0.062**	**.000**	**−0.057**	**.000**	**−0.112**	**.000**
$B_r \times$ age	−0.132	.002	0.054	.154	0.036	.247	−0.085	.046	−0.059	.171
$B_r \times$ sex	0.004	.403	−0.004	.414	−0.007	.346	−0.030	.043	−0.018	.193

Note: Boldface indicates significant effects (p ≤ .001). B_r = response bias; N-back = Penn Letter N-Back Test; PCPT = Penn Continuous Performance Task; P_r = discrimination accuracy.

[a]*Although model estimation terminated normally, coefficients with an absolute value higher than 1.0 were possible because of non-positive–definite residual variance and covariance matrices.*