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Domain-specific associations between psychopathology and neurocognitive functioning

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Original Article

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Abstract

Background. Neurocognitive dysfunction is a transdiagnostic finding in psychopathology, but relationships among cognitive domains and general and specific psychopathology dimensions remain unclear. This study aimed to examine associations between cognition and psychopathology dimensions in a large youth cohort.

Method. The sample ($N = 9350$; age 8–21 years) was drawn from the Philadelphia Neurodevelopmental Cohort. Data from structured clinical interviews were modeled using bifactor confirmatory factor analysis (CFA), resulting in an overall psychopathology ('p') factor score and six orthogonal psychopathology dimensions: dysphoria/distress, obsessive-compulsive, behavioral/externalizing, attention-deficit/hyperactivity, phobias, and psychosis. Neurocognitive data were aggregated using correlated-traits CFA into five factors: executive functioning, memory, complex cognition, social cognition, and sensorimotor speed. We examined relationships among specific and general psychopathology dimensions and neurocognitive factors.

Results. The final model showed both overall and specific associations between cognitive functioning and psychopathology, with acceptable fit (CFI = 0.91; TLI = 0.90; RMSEA = 0.024; SRMR = 0.054). Overall psychopathology and most psychopathology dimensions were negatively associated with neurocognitive functioning (phobias [$p < 0.0005$], behavioral/externalizing [$p < 0.0005$], attention-deficit/hyperactivity [$p < 0.0005$], psychosis [$p < 0.0005$ to $p < 0.05$]), except for dysphoria/distress and obsessive-compulsive symptoms, which were positively associated with complex cognition ($p < 0.05$ and $p < 0.01$, respectively).

Conclusion. By modeling a broad range of cognitive and psychopathology domains in a large, diverse sample of youth, we found aspects of neurocognitive functioning shared across clinical phenotypes, as well as domain-specific patterns. Findings support transdiagnostic examination of cognitive performance to parse variability in the link between neurocognitive functioning and clinical phenotypes.

Neurocognitive dysfunction is common in psychiatric disorders including schizophrenia (Reichenberg, 2010; Saykin et al., 1991), posttraumatic stress (Aupperle, Melrose, Stein, & Paulus, 2012; Scott et al., 2015), and mood disorders (Bredemeier, Warren, Berenbaum, Miller, & Heller, 2016; Merikangas et al., 2017). It is prevalent across diagnoses (Abramovitch, Short, & Schweiger, 2021; Weiser et al., 2004) and in those scoring high on internalizing symptoms dimensions (Chavez-Baldini et al., 2023; Zhu et al., 2019). Because neurocognitive deficits often precede the onset of symptoms (Dickson, Laurens, Cullen, & Hodgins, 2012; Gur et al., 2014), they may indicate risk for psychopathology (Caspi et al., 2014; Jonas et al., 2022; Moffitt et al., 2011; Rotstein, Fund, Levine, Reichenberg, & Goldenberg, 2023) and elucidate neurodevelopment and psychopathology.

Most studies report impaired cognitive functioning in those with mental illness or high levels of a symptom dimension (East-Richard, R. -Mercier, Nadeau, & Cellard, 2020; Weiser et al., 2004), yet results for distinct neurocognitive profiles associated with specific dimensions of psychopathology vary. This heterogeneity may relate to diverse approaches in transdiagnostic studies (Astle, Holmes, Kievit, & Gathercole, 2022). One approach has compared cognitive functioning across traditional categorical diagnoses with shared characteristics. For example, studies have examined generalized and disorder-specific executive functioning across multiple disorders with social impairment, including social anxiety, autism, and early psychosis (Demetriou et al., 2018). Others have examined differential and shared patterns of neurocognitive performance across mood disorders (Merikangas et al., 2017). Individuals with bipolar

I disorder performed more accurately in complex cognition compared to controls, while individuals with major depressive disorder performed more accurately in emotion recognition, and patients across disorders showed similar speed performance across domains. While transdiagnostic, such findings still rest on categorical classification of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013) and reflect the shortcomings of these categories, including variability within diagnoses, comorbidity, and exclusion of subclinical populations with arbitrary thresholds (Astle et al., 2022).

An alternative to categorical approaches is dimensional models of psychopathology, such as NIMH's Research Domain Criteria (RDoC; Insel, 2014) and the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017) frameworks. Mental disorders are conceptualized as reflecting interactions among continuous parameters, with interacting symptom dimensions or factors (Caspi et al., 2014). These models often incorporate a general psychopathology factor accounting for shared variance across dimensions (Caspi et al., 2014; Lahey et al., 2012; Lahey, Krueger, Rathouz, Waldman, & Zald, 2017), and specific factors reflecting distinct psychopathology dimensions. Dimensional models offer one method to model psychiatric comorbidity, and the structure of these models has been examined (Kim & Eaton, 2015; Michelini et al., 2019; Ringwald, Forbes, & Wright, 2023; Sunderland et al., 2021). One promising approach is bifactor models (Moore et al., 2020), which specify a general factor of psychopathology and orthogonal individual factors, where scale items are allowed to load on both the general psychopathology factor and one specific factor (Reise, Moore, & Haviland, 2010). Thus, bifactor models can assess dimensionality of psychopathology by isolating the shared variance explained by general psychopathology from the specific variance explained by individual symptom dimensions (Reise, 2012).

Despite extensive research on neurocognitive functioning in mental health disorders, few studies have examined associations among broad sets of cognitive domains and psychopathology symptom dimensions. Studies of transdiagnostic samples focused on associations with single cognitive domains: executive functioning (Bloemen et al., 2018; Demetriou et al., 2021, 2018; Romer & Pizzagalli, 2021; Shanmugan et al., 2016; White et al., 2017), processing speed (Kramer, Willcutt, Peterson, Pennington, & McGrath, 2023), or social cognition (Gur, Moore, Calkins, Ruparel, & Gur, 2017). Other studies have evaluated a broad range of cognitive domains and dimensional psychopathology but used DSM-based categorical diagnoses as inclusion criteria (Chavez-Baldini et al., 2023; Merikangas et al., 2017; Service et al., 2020; Zhu et al., 2019). Other transdiagnostic research has examined cognitive functioning across limited psychopathology dimensions, such as internalizing, externalizing, or attention deficit symptomatology (Bloemen et al., 2018; Brislin et al., 2022). Most studies in youth have focused on associations between psychopathology and global cognition or executive function (Demetriou et al., 2021; Kramer et al., 2023; Romer & Pizzagalli, 2021; White et al., 2017), limiting the ability to identify neurocognitive correlates of psychopathology phenotypes. A better understanding is needed regarding the nature of neurocognitive deficits, both shared and unique, across psychopathology dimensions.

The present study aimed to evaluate relationships among cognitive functioning, general psychopathology, and distinct dimensions of psychopathology in a large sample of children, adolescents, and young adults. We utilized data from the community-based Philadelphia Neurodevelopmental Cohort (PNC), which included a structured psychopathology interview and a neurocognitive assessment across domains (Calkins et al., 2015; Gur et al., 2012). Prior PNC studies examined links between psychopathology and cognitive functioning in individual cognitive domains, such as executive functioning (White et al., 2017) or social cognition (Gur et al., 2017). In a recent review, Jonas et al. (2024) provided a preliminary demonstration of cognitive performance deficits associated with symptom levels in externalizing, psychosis, fear (phobias), and anxious-misery domains in the PNC. However, no study to date has utilized the full neurocognitive battery from the PNC, applied a data-driven approach to model psychopathology, and examined transdiagnostic and distinct links between psychopathology and cognition. Here, we adopted a dimensional approach to assess variations in mental health symptoms using a bifactor model, with one general psychopathology factor and six domain-specific factors, and examined associations with domains of neurocognitive functioning using a well-validated test battery.

Methods

Participants

The sample was derived from the PNC ($N = 9498$), an NIMH-funded Grand Opportunity (GO) project (Calkins et al., 2015, 2014; Gur et al., 2012; Satterthwaite et al., 2014). The present study is a secondary analysis of clinical and neurocognitive data from this cross-sectional study. Briefly, the PNC is a community-based sample of individuals aged 8–21 years enrolled in genomic studies at the Children's Hospital of Philadelphia (CHOP) who provided permission to be recontacted. Of 50 293 individuals recruited by the CHOP Center for Applied Genomics, 19 161 potentially eligible participants met inclusion criteria based on Electronic Medical Records (EMRs): (a) ages 8–21, (b) English proficiency, (c) good general health allowing completion of study procedures, (d) written informed consent/assent for re-contacting for future studies. Of potentially eligible participants, 13 598 were invited to participate, the remaining unreachable or unable to schedule. Of those invited, 9498 (64%) were enrolled and 9350 had complete data for analyses. Data collection lasted from 2009 to 2013. The sample here were 51.8% female (mean age 13.3 years [S.D. = 3.7]), 56.1% White, 32.7% Black, 11.3% Other racial category.

Following an overview of the study, written consent from participants age >18 years and assent and parental/legal guardian consent for individuals <18 were obtained. The study protocol was approved by CHOP and University of Pennsylvania institutional review boards.

Psychopathology assessment

Clinical assessments were described previously (Calkins et al., 2015). Briefly, a computerized structured interview (GOASSESS), adapted from the NIMH Genetic Epidemiology Research Branch Kiddie-SADS for Affective Disorders and Schizophrenia (Merikangas, Avenevoli, Costello, Koretz, & Kessler, 2009), was administered to probands and collaterals (ages 8–17) or probands alone (ages 18–21). In addition to medical history and demographic information, the GOASSESS evaluates several psychopathology domains: mood (Major Depressive Episode, Manic Episode), anxiety (Generalized Anxiety, Separation Anxiety, Specific Phobia, Social Phobia, Panic, Agoraphobia, Obsessive-Compulsive,

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Post-traumatic Stress), Attention-Deficit/Hyperactivity, behavioral (Oppositional Defiant, Conduct), eating disorders (Anorexia, Bulimia), and suicidality. To evaluate negative/disorganized and positive psychosis symptoms, we used items from the Scale of Prodromal Symptoms (SOPS) from the Structured Interview for Prodromal Syndromes (McGlashan, Miller, & Woods, 2003) and the Prevention through Risk Identification, Management, and Education (PRIME) Screen-Revised (Kobayashi et al., 2008; Miller et al., 2003), respectively. Here, 115 screening items from the GOASSESS were used, but detailed probe items (e.g. duration of specific symptoms, functional impairment) were not considered because their inclusion resulted in unusual item properties due to very small variances. Assessments were conducted by Bachelor's and Master's level trained clinical research coordinators ($n = 55$) at the laboratory or the participant's home. Assessors underwent common training and certification protocols accompanied by periodic re-trainings, and all interview data underwent standardized post-administration review procedures (Calkins et al., 2015). Each interview required the presence of one assessor. Participants' responses to interview questions were recorded in the database.

Cognitive assessment

Neurocognitive functioning was assessed using the Penn Computerized Neurocognitive Battery (CNB), which includes tests covering a broad array of cognitive domains (Gur et al., 2012, 2014, 2010; Moore, Reise, Gur, Hakonarson, & Gur, 2015; Roalf et al., 2014). The CNB has been validated in populations with wide age ranges in clinical and community samples (Gur et al., 2006, 2012; Hartung et al., 2016; Iannacone et al., 2014; Irani et al., 2012; Roalf et al., 2014; Silver et al., 2006). All neurocognitive tests have been previously detailed (Gur et al., 2001, 2010, 2012; Moore et al., 2015) and are summarized in the Supplement by cognitive domain. The 14 CNB tests take approximately 1-h to complete and assess five neurocognitive domains: complex cognition, executive function, episodic memory, social cognition, and sensorimotor speed. Clinical research coordinators trained on a standard protocol administered the CNB on the web-based platform (Gur et al., 2010, 2012) and installed it on laptops for testing in participants' homes or the laboratory. Response times and responses were recorded and uploaded to the database.

Analyses

Demographic variables (age, sex) were obtained during the psychopathology assessment. Socioeconomic status (SES) was approximated by linking participants' addresses to census block-groups (neighborhoods) (Moore et al., 2016).

Factor analyses were conducted to construct measurement models and examine the relations between latent psychopathology and cognition factors. To model psychopathology, we used the item-wise clinical data. For participants in the 8–10 age group, we used the collateral report, and for ages 11–21, we used proband report. While original analyses of these items (Shanmugan et al., 2016) extracted four factors (and 'p'), here we applied a data-driven approach to examine whether items might 'split' further into additional sub-factors (beyond 4), allowing relationships between psychopathology and cognition to be more specific. Indeed, we found two additional factors. This addition of factors was supported by the CFA fit indices: while the 4-factor model produced acceptable conventional fit indices (CFI, SRMR, and RMSEA), replicating Shanmugan et al. (2016), the 'robust' fit indices produced by Lavaan were unacceptable for the 4-factor model. It was not until six factors were specified that the 'robust' fit indices reached acceptable levels. In the present analysis, we split the sample and conducted a series of preliminary exploratory factor analyses (EFA) in the first half of the sample to determine the best underlying structure for the symptom domains. The 6-factor solution was then used in confirmatory factor analyses (CFA), which were conducted on the second half of the sample, with a bifactor configuration. Age, sex, and SES were controlled for in the measurement model at the item level. Factor reliability indices (e.g. H, determinacy, etc.) commonly reported for bifactor models (Rodriguez, Reise, & Haviland, 2016) were estimated.

Cognition was modeled based on our previous work (Moore et al., 2015). Scores reflected efficiency, defined as the average of standardized speed and accuracy scores. Based on prior (2015) recommendation, our model placed abstraction and mental flexibility in the complex cognition factor instead of the executive control factor. The present model additionally differed from the Moore et al. (2015) model by including two additional indicators: motor (tapping) speed and sensorimotor (praxis) speed. These scores were modeled as their own factor, resulting in a 5-factor model (compared to 4 factors in previous work). A second *ad hoc* correction made to the cognition model was that the bifactor configuration was abandoned in favor of a correlated-traits configuration (Reise et al., 2010), because the H indices of factor reliability (Rodriguez et al., 2016) were too low for the specific factors in the bifactor model to justify relating them to the clinical factor in an SEM. The correlated-traits model fixed this problem, resulting in highly reliable latent cognitive factors in the CFA. Sex and SES were controlled for in all cognitive variables within the measurement model, as in the clinical model. Age was controlled for before analysis using age-normalization of scores. Sex, SES, and age were controlled for at the indicator-level (i.e. item- or test-level) within the confirmatory factor analysis; rather than relating the estimated factors to these covariates (regressing out of the factors), they were regressed out of the indicators of those factors.

Data imputation and EFAs were conducted in R v4.2.2 (R Core Team, 2022), using the missForest (Stekhoven, 2022; Stekhoven & Bühlmann, 2012) and Lavaan (Rosseel, 2012) packages, respectively. CFAs were conducted in Mplus version 8.4 (Muthén & Muthén, 1998–2019) using the mean- and variance-adjusted weighted least squares (wlsmv) estimator (Muthén, du Toit, & Spisic, 1997). Model fit was evaluated based on recommendations from previous research (Hu & Bentler, 1998; Hu & Bentler, 1999). Bifactor-specific indices were calculated in R, using the BifactorIndicesCalculator package (Dueber, 2021). The effects of interest and the two models were estimated concurrently. Effects of interest were the relationships between clinical and cognitive factors. Factors within the clinical model were treated orthogonally, while cognitive factors were allowed to covary. All analyses covaried for age, sex, and SES.

Results

Exploratory factor analysis: clinical model

Supplementary Table 1 shows EFA results from one-half of the sample ($n = 4674$). The 6 factors represented the following domains: dysphoria/distress, obsessive-compulsive (OC), behavioral/externalizing, attention-deficit/hyperactivity (ADH), phobias, and psychosis. Next, CFA with a bifactor configuration to include an overall psychopathology ('p') factor was conducted

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in the second half of the sample, and model fit was acceptable. The comparative fit index (CFI) was 0.91 and the Tucker-Lewis index (TLI) was 0.90; the root mean-square error of approximation (RSMEA) was 0.025 and the standardized root mean square residual (SRMR) was 0.063. The strongest indicators of each factor are detailed in Supplement.

Compared to Shanmugan et al. (2016) 4-factor structure, our model revealed further separation in the previously defined 'anxious-misery' factor and the behavioral factor. Specifically, the anxious-misery (mood-anxiety symptoms) factor split, with OC diverging as its own factor. We labeled the remaining symptoms 'dysphoria/distress', aligning with HiTOP nomenclature (Jonas et al., 2024; Kotov et al., 2017). The ADH symptoms diverged from the previous behavioral factor into a distinct factor, and we labeled the remaining symptoms as 'behavioral/externalizing' factor, aligning with HiTOP nomenclature. The fear (phobias) and the psychosis factors remained.

Confirmatory factor analysis

To model cognition, we ran a correlated-traits model with the five domains: complex cognition, executive function, episodic memory, social cognition, and sensorimotor speed. The fit indices were adequate (CFI = 0.96; TLI = 0.92; RMSEA = 0.048; SRMR = 0.025).

The final confirmatory model, tested on the entire sample, included the cognitive correlated-traits model (Table S2) and the clinical bifactor model (Table S3), showing acceptable fit (CFI = 0.91; TLI = 0.90; RMSEA = 0.024; SRMR = 0.054). Significant associations among domains of the psychopathology and cognitive models are shown in Fig. 1. Cognitive factors were specified to correlate with clinical factors. Within the context of the final confirmatory model, the psychopathology bifactor model showed good internal consistency, supporting the presence of an overall psychopathology ('p') factor (general factor $\omega = 0.98$; $\omega_H = 0.88$, $H = 0.97$), with sufficient reliability remaining in each specific factor (Hs = 0.80–0.86). While the bifactor is the least parsimonious measurement model (Reise et al., 2010), given the borderline fit of the bifactor model (e.g. CFI = 0.91), that added complexity appears to be necessary. See Supplementary Methods for greater detail and Supplementary Table S4 for bifactor reliability indices.

Associations between clinical and cognitive domains

Cognition and psychopathology dimensions showed both factor-specific and overall significant associations (Table 1). The p-factor was associated with poorer complex cognition ($p < 0.0005$), but no other domain-specific deficit. All cognitive domains were negatively associated with ADH (all ps < 0.0005), phobias (all ps < 0.0005), and psychosis (ps<0.0005 for complex cognition, episodic memory, and social cognition; $p = 0.006$ for executive function; $p = 0.048$ for sensorimotor speed) factors, indicating that greater psychopathology symptom burden was associated with poorer cognitive performance. The behavioral/externalizing factor was negatively associated with complex cognition and executive functioning (ps < 0.0005), but with no other neurocognitive domain. Positive associations were observed between dysphoria/distress ($p = 0.043$) and OC ($p = 0.005$) factors and complex cognition, indicating that those with greater symptom burden had better complex reasoning abilities. However, these psychopathology dimensions did not show significant associations with other cognitive domains.

Differences in the magnitude of the associations between cognitive functioning and psychopathology dimensions also emerged. To assess the statistical significance of the differences among estimates, a 95% confidence interval (CI) for each estimate was calculated. Since some overlap in CIs does not necessarily indicate lack of significant differences between groups, we have indicated the significance of differences by asterisks in Fig. 2 and specified them in Supplementary Table S5. As can be seen in Fig. 2 and Table S5, psychopathology dimensions showed some specificity of association with cognitive performance. Overall, phobias, ADH, and psychosis were associated with deficits across domains. However, ADH showed the greatest deficit in executive functions, phobias had the greatest deficits in episodic memory, complex cognition, and sensorimotor speed, and psychosis had the greatest deficits in social cognition. The behavioral/externalizing factor was associated with the poorest performance specifically in executive functioning and complex cognition domains, and both OC and dysphoria/distress were associated with better than average complex cognition performance.

Discussion

Prior research has shown that neurocognitive deficits are transdiagnostic in nature (Abramovitch et al., 2021). However, it has been challenging to precisely characterize the relationships between neurocognitive functioning and psychopathology dimensions to determine the specificity of such associations. Using bifactor and correlated-traits models, the present study evaluated relationships among domains of cognition and dimensions of psychopathology in a large, community-based youth cohort. The latent structure of the psychopathology model uncovered an overall psychopathology ('p') factor and 6 orthogonal symptom dimensions – dysphoria/distress, OC, behavioral/externalizing, ADH, phobias, and psychosis. We found both unique and shared patterns of associations between these psychopathology dimensions and five domains of neurocognitive functioning: executive function, episodic memory, complex cognition, social cognition, and sensorimotor speed. While our findings support the existence of transdiagnostic cognitive deficits, they also identify some specificity in associations with psychopathology dimensions, including dimensions associated with better neurocognitive performance.

Our results are consistent with extensive literature demonstrating a relationship between general psychopathology ('p') and cognition (Bloemen et al., 2018; Brislin et al., 2022; Caspi et al., 2014; Castellanos-Ryan et al., 2016; Kramer et al., 2023; Martel et al., 2017; Romer & Pizzagalli, 2021). However, our analyses revealed that only the complex cognition domain was associated with general psychopathology. Notably, most prior research linked executive functioning to the p-factor (Bloemen et al., 2018; Castellanos-Ryan et al., 2016; Martel et al., 2017; Romer & Pizzagalli, 2021; White et al., 2017). Difference in results may reflect different definitions of cognitive constructs. Kramer et al. (2023) found that higher overall psychopathology was linked to poorer performance on processing speed tasks. However, certain tasks in their battery measure aspects of complex cognition. For example, the Colorado Speed Test (Decker, 1989) and the Penn Verbal Reasoning Test (PVRT) relate to abstraction and language. Similarly, White et al. (2017) assessed executive functioning using the Penn Conditional Exclusion Test (PCET), included in our complex cognition domain to align with prior findings (Moore et al., 2015). The effect sizes we found are consistent with prior studies that also found small correlations. The extensive original

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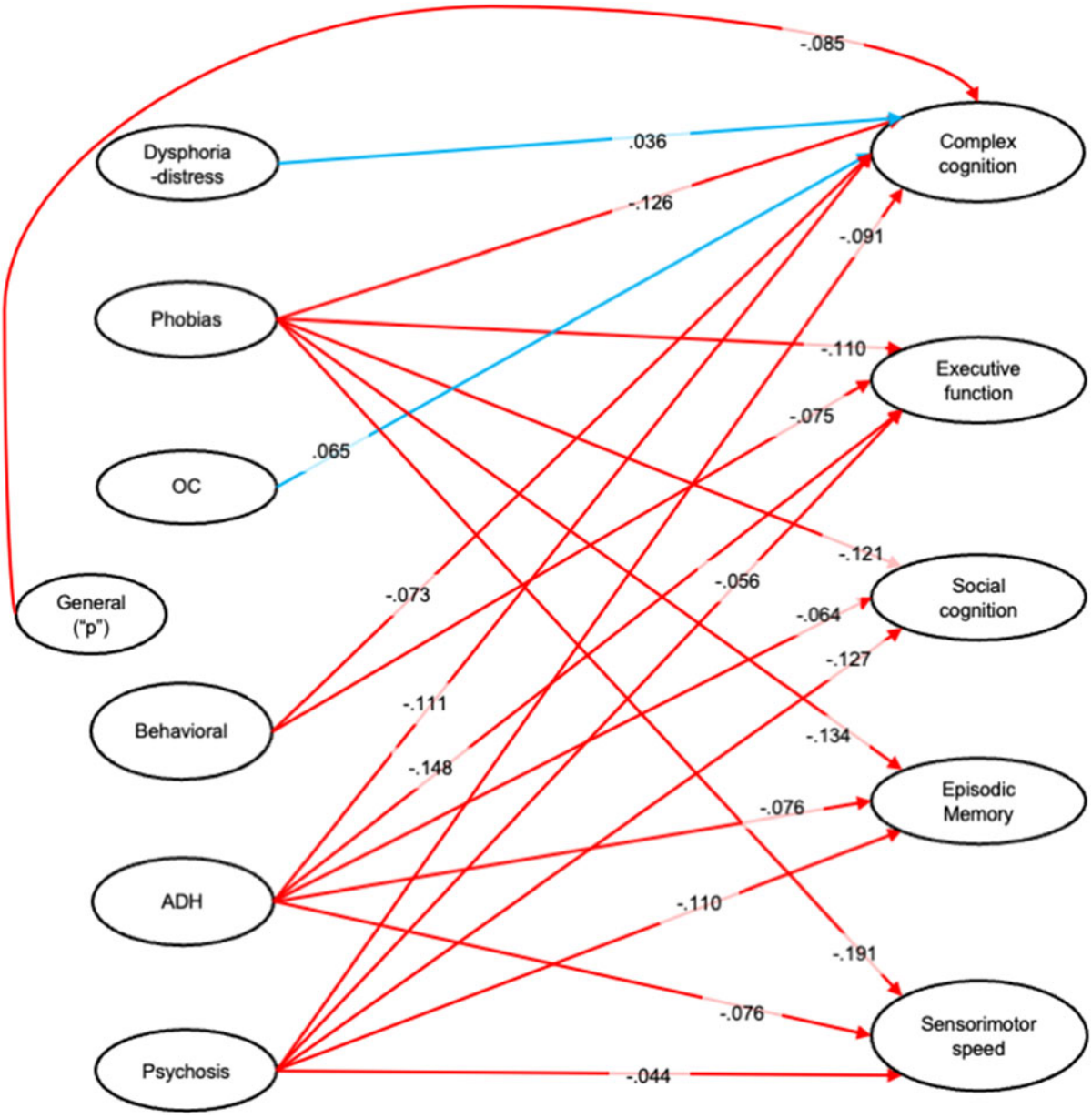

Figure 1. Structural equation model with psychopathology bifactor and cognitive correlated-traits models.
Note: The structural equation model depicts the significant associations ($p < 0.05$) between the latent psychopathology factors of the bifactor model and the latent cognitive factors in the correlated-traits model. Blue arrows indicate positive associations; red arrows indicate negative associations. OC, obsessive-compulsive; ADH, attention-deficit/hyperactivity.

study (Caspi et al., 2014) found relatively weak relationships between 'p' and cognition/IQ (e.g. −0.19 for WAIS FSIQ, −0.13 for verbal comprehension, −0.13 for perceptual reasoning, −0.18 for working memory, etc.), and other studies (e.g. Castellanos-Ryan et al., 2016) had similarly small effects (r = 0.07–0.14). The small effect sizes in the present study are perhaps due to use of efficiency rather than accuracy as the main cognitive dimension, and correction for several covariates (SES, gender, age) at the test and item level (compared to uncorrected bivariate correlations in Caspi et al., 2014).

Though prior reviews have suggested that psychopathology is associated with generalized cognitive deficits (Abramovitch et al., 2021), our findings suggest some specificity beyond transdiagnostic cognitive dysfunction. Consistent with prior work, higher levels of phobias, psychosis, behavioral/externalizing, and ADH symptoms were associated with lower performance in executive functioning and complex cognition (Bloemen et al., 2018; Gur et al., 2014; Jonas et al., 2024; Kramer et al., 2023; White et al., 2017). Higher levels of phobias, psychosis, and behavioral symptoms were also associated with lower performance in episodic memory. Lower performance in social cognition tasks was likewise reported for psychosis (Gur et al., 2006, 2017; Jonas et al., 2024), phobias (Jonas et al., 2024; Plana, Lavoie, Battaglia, & Achim, 2014), and ADH dimensions (Parke et al., 2021; Uekermann et al., 2010). Regarding sensorimotor speed, we replicated an association between slower sensorimotor speed and psychosis symptoms (Osborne, Walther, Shankman, & Mittal, 2020). We also found that the phobias and ADH factors

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Table 1. Associations between neurocognitive and psychopathology domains

	Neurocognitive factors									
	Complex cognition		Executive function		Episodic memory		Social cognition		Sensorimotor speed	
Psychopathology factors	*β*	*P*	*β*	*p*	*β*	*p*	*β*	*p*	*β*	*p*
Dysphoria/distress	**0.036**	**0.043**	−0.002	0.920	−0.002	0.925	−0.003	0.848	−0.014	0.519
OC	**0.065**	**0.005**	**−0.023**	**0.352**	−0.012	0.575	0.000	0.996	−0.025	0.380
Behavioral/externalizing	**−0.073**	**<0.0005**	**−0.075**	**<0.0005**	−0.011	0.555	−0.019	0.315	−0.018	0.454
ADH	**−0.111**	**<0.0005**	**−0.148**	**<0.0005**	**−0.076**	**<0.0005**	**−0.064**	**<0.0005**	**−0.076**	**<0.0005**
Phobias	**−0.126**	**<0.0005**	**−0.110**	**<0.0005**	**−0.134**	**<0.0005**	**−0.121**	**<0.0005**	**−0.191**	**<0.0005**
Psychosis	**−0.091**	**<0.0005**	**−0.056**	**0.006**	**−0.110**	**<0.0005**	**−0.127**	**<0.0005**	**−0.044**	**0.048**
Overall P	**−0.085**	**<0.0005**	−0.024	0.185	0.017	0.271	−0.018	0.221	−0.017	0.374

OC, obsessive-compulsive; ADH, attention-deficit/hyperactivity.
Boldface indicates significant effects ($p < 0.05$).

were associated with slower motor speed, but the nature of this relationship is less clear (Farran et al., 2020). Finally, OC and dysphoria/distress dimensions were associated with better performance in complex cognition (Brislin et al., 2022; Jonas et al., 2024). Such specificities may have been masked in prior studies due to smaller samples, limited psychopathology dimensions, or a reduced neurocognitive battery. Our model's parsing of psychopathology factors and examination of a broad range of cognitive functions may offer more sensitivity to domain-specific associations with cognition.

Psychosis was linked more strongly to poorer social cognition than ADH, consistent with prior PNC research showing that psychosis is associated with lower social cognition scores compared to other dimensions (Gur et al., 2017). On the other hand, phobias and ADH were associated with even poorer executive functioning than psychosis, diagnostic domains that need comparative examination (Service et al., 2020; White et al., 2017). Consistent with prior research (Clark, Prior, & Kinsella, 2000; Ezpeleta & Granero, 2015; Jarrett, 2016), ADH was more strongly linked to executive function than the phobias or behavioral/externalizing dimensions. The phobias factor was significantly associated with all neurocognitive domains. Furthermore, it was more strongly linked to poorer complex cognition than the behavioral dimension and overall psychopathology, and more strongly associated with poorer episodic memory and social cognition than ADH. We also found that phobias had a more robust association with sensorimotor speed compared to ADH and psychosis. Among all cognitive domains, sensorimotor speed was most strongly linked to phobias, a finding that could be pursued in future studies. Prior work has reported mixed associations between phobias and cognitive functioning (Demetriou et al., 2021; Gustavson & Miyake, 2016; White et al., 2017). Further work may be needed to disentangle the covariation between phobias/fear and dimensions of personality or psychopathology, such as neuroticism or negative emotionality (Watson, Clark, Simms, & Kotov, 2022).

The dimensions significantly associated with poorer neurocognitive functioning across all domains were psychosis and ADH – the latter collapsed in the behavioral factor in prior studies (Calkins et al., 2015; Shanmugan et al., 2016). Both psychosis and behavioral/ADH are often extracted in dimensional studies of psychopathology (Caspi et al., 2014; Martel et al., 2017; Parkes et al., 2021; Sunderland et al., 2021). Additionally, they can be related to components of the *p*-factor identified by Southward, Cheavens, and Coccaro (2023). Specifically, ADH has been linked to impulsivity (Leffa, Caye, & Rohde, 2022; Walerius, Reyes, Rosen, & Factor, 2018), while psychosis-related disorders can be conceptualized as thought disorders (Kotov et al., 2017). Impulsivity has been proposed as a main subcomponent of the *p*-factor, and thought dysfunction has shown a strong link with the *p*-factor (Southward et al., 2023). Examining how dimensions intersect with cognitive performance across various domains could allow a more precise characterization of at-risk individuals and uncover potential avenues for targeted interventions.

Dysphoria/distress and OC factors were associated with better performance on complex cognition tasks, similar to prior work and a recent demonstration of cognitive functioning associated with anxious-misery symptoms in the PNC (Jonas et al., 2024). Increased internalizing symptoms, typically including anxious/depressed and somatic complains, have been linked to higher general cognitive functioning (Brislin et al., 2022) and cognitive

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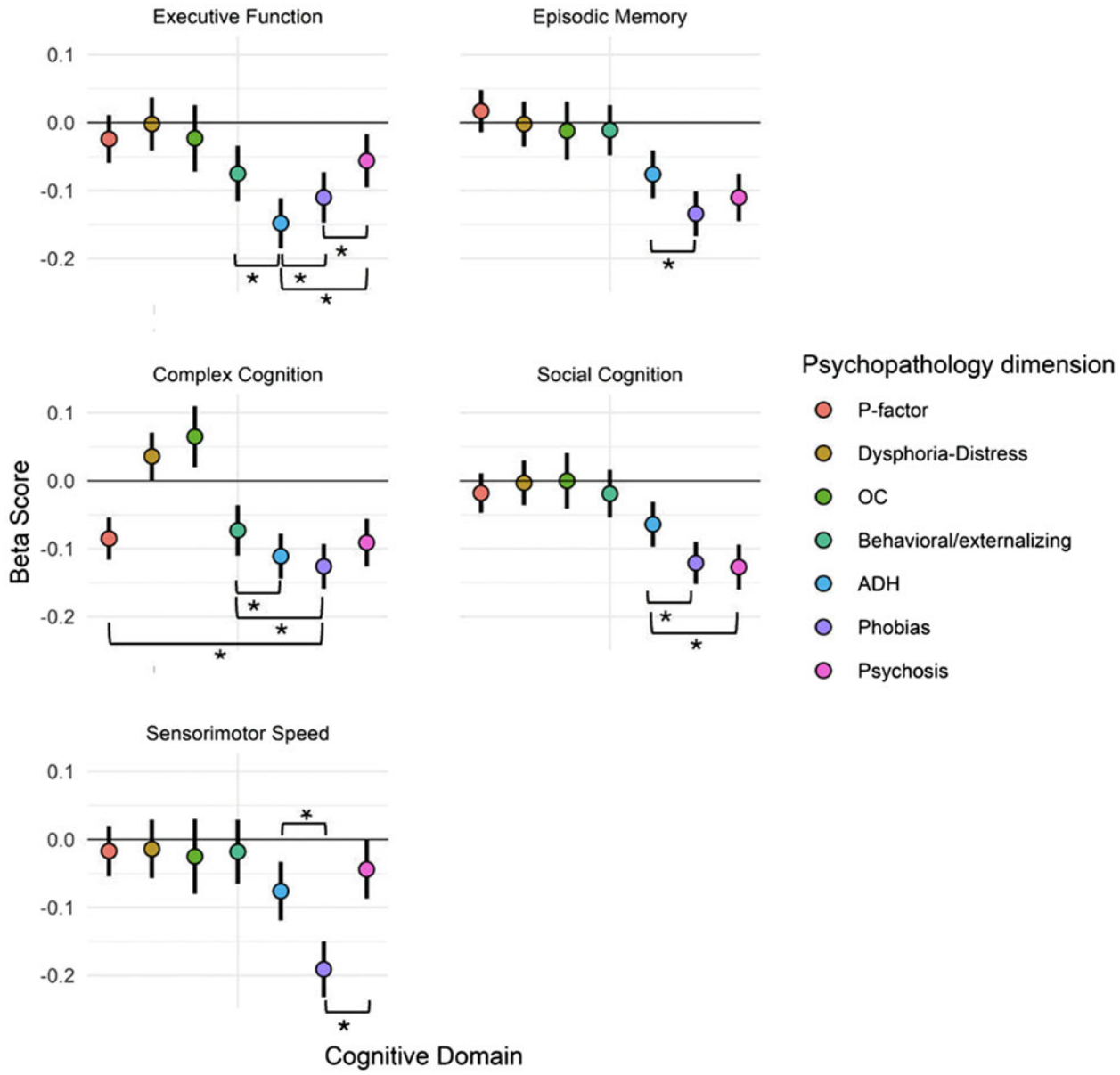

Figure 2. Effect sizes for psychopathology-cognitive domain associations profiles of psychopathology domain beta scores for each cognitive domain.
Note: Error bars represent 95% confident intervals. ADH, attention-deficit/hyperactivity; OC, obsessive-compulsive. * indicates significant difference between compared groups.

flexibility (Bloemen et al., 2018). Of note, White et al. (2017), who included the PCET in the executive function domain in the PNC, also reported a positive association between executive functioning and the anxious-misery symptom dimension. Our findings also replicate Service et al.'s (2020) transdiagnostic study of the Paisa population in Colombia. The major depression group was the least impaired across neurocognitive domains, and the 'anxious-misery' factor, across diagnoses, was positively associated with performance, especially on social cognition.

Limitations of the present study should be acknowledged. The sample was restricted to individuals aged 8–21, affecting generalizability to other age groups. Relatedly, clinical data for the psychopathology models were drawn from collateral information for participants aged 8–10 years and proband information for those aged 11–21 years, similar to prior work. However, this discrepancy could affect the validity and reliability of results, given that divergence between proband and collateral reports have been reported across psychopathology domains, race, and SES (e.g. Belendiuk, Clarke, Chronis, and Raggi, 2007; Curhan, Rabinowitz, Pas, and Bradshaw, 2020; Jones et al., 2017; Salbach-Andrae, Klinkowski, Lenz, and Lehmkuhl, 2009; Xavier et al., 2022). In addition, the PNC evaluated lifetime diagnoses in a community-based, non-clinical sample, which might assess psychopathology traits rather than states. This is perhaps why higher *p*-factor scores significantly associated only with poorer complex cognition performance, the most stable, trait-like factor in the CNB (analogous to IQ; Beaver et al., 2013). The focus on lifetime diagnoses rather than current symptoms also might not have fully captured the temporal aspect of certain conditions. Certain domains of psychopathology, such as personality disorders and substance use disorders, were not assessed. The use of cross-sectional data hinders establishing the directionality of

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the relationships between cognitive domains and symptom dimensions (Abramovitch et al., 2021; Bredemeier et al., 2016; Letkiewicz et al., 2014; Romer & Pizzagalli, 2021). The sub-factors in the cognition model might be 'contaminated' with the 'g' factor since we did not use a bifactor model. We used efficiency scores in these analyses rather than examining performance accuracy and speed separately, which prevents specification of specific associations of psychopathology with accuracy or speed individually. Given that our sample spanned ages marked by significant neurodevelopment, it is possible that the associations between cognitive and clinical factors are not consistent across the age range, or that factors used are not invariant across this age range; future work should examine this possibility. Finally, to model psychopathology we used a bifactor model, which has been criticized for overfitting data and undermining model validity (Watts, Lane, Bonifay, Steinley, & Meyer, 2020).

Notwithstanding these limitations, the present study offers insights into the transdiagnostic nature of cognitive deficits in youth. Findings support both transdiagnostic cognitive deficits and variability in the link between clinical phenotypes and neurocognitive functioning. Future research should explore these relationships in a sample with a broader age range and more severe clinical profiles, including elevated current symptoms. Longitudinal studies exploring the temporal link between psychopathology and cognitive deficits could also inform treatment and early interventions targeting cognition to prevent, detect, or treat mental health conditions.

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Competing interests. The authors declare none.

Ethical standards. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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