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Do Cognitive Functions Belong in the Hierarchical Taxonomy of Psychopathology Model? A Meta-Analysis

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Abstract

Cognitive dysfunction is essential to conceptualizing, defining, and assessing much of psychopathology. Despite this prominence, cognitive abilities are not included in the prevailing empirically based classification system: the Hierarchical Taxonomy of Psychopathology (HiTOP). This gap exists because the factor-analytic literature the HiTOP is based on has solely used reporter measures rather than neuropsychological tests needed to measure cognitive ability. Given HiTOP's influence on research and clinical practice, the omission of cognitive functions from the model is consequential. This study aimed to determine how cognitive abilities fit into the empirical structure of psychopathology with a meta-analytic joint factor analysis. We pooled data from three published meta-analyses into a single correlation matrix of eight disorders from the *Diagnostic and Statistical Manual of Mental Disorders* and seven cognitive functions. We then fit a series of models to the meta-analytic correlation matrix using exploratory factor analysis and correlated factors across levels to estimate the hierarchical structure. The highest level of the model included a general factor with strong loadings of all disorders and cognitive functions (median $\lambda = |.51|$, range = $|.30|$ to $|.64|$). At the lowest level were three superspectra: psychosis and cognitive dysfunction, externalizing, and emotional dysfunction. Our results show cognitive abilities can be integrated into the HiTOP model and point to actionable next steps in research to accomplish this goal.

Keywords

nosology, classification, neuropsychological, mental disorders, factor analysis

Cognitive dysfunction is a hallmark of psychopathology. Many clinical theories posit a central role of cognitive deficits to psychopathology (Baskin-Sommers & Foti, 2015; Carver et al., 2017; Caspi & Moffitt, 2018; Green et al., 2019; Zelazo, 2020). Cognitive deficiencies often co-occur with mental disorders (Abramovitch et al., 2021; East-Richard et al., 2020), are strongly predictive of prognosis (McCutcheon et al., 2023), and are the primary target of treatments for multiple diagnoses (Nardo et al., 2022; Thérond et al., 2021; Vita et al., 2021). In fact, some forms of psychopathology are defined almost exclusively by impairments in cognition (e.g., dementia, learning disorders, intellectual disability; Andrews et al., 2009; Sachdev et al., 2009). Cognition is considered so essential to psychopathology that it is one of the domains in the National Institutes of Mental Health's Research Domain Criteria (RDoC) initiative (Morris & Cuthbert, 2012). Despite the prominence of cognitive dysfunction in how mental disorders are conceptualized, defined, and often treated, they are not included in the prevailing empirically based classification system: the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017, 2021).

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Omission of Cognitive Functions From the Empirical Structure of Psychopathology

The HiTOP model defines psychopathology in terms of hierarchically organized dimensions with narrow signs, symptoms, and behaviors at the base that combine into increasingly broad constructs up to the superspectra level (i.e., emotional dysfunction, externalizing, and psychosis). This model represents the consensus among a vast literature on the empirical structure of psychopathology and reflects a broader shift in clinical science away from *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) diagnoses from and toward transdiagnostic, dimensional models (Dalgleish et al., 2020). Because the HiTOP provides empirically validated phenotypes, this framework has advanced research on etiology, risk factors, mechanisms, and outcomes of psychopathology (Kim et al., 2021; Kotov et al., 2020; Krueger et al., 2021; Perkins et al., 2020; Waszczuk et al., 2020; Watson et al., 2022). Furthermore, clinicians favor the HiTOP over the *DSM* for case formulation, treatment planning, and clinical communication (Balling et al., 2023), and increasing evidence supports the model's clinical utility (Ruggero et al., 2019).

Cognitive dysfunction can be a symptom of psychopathology, and a long-standing goal of the HiTOP has been to incorporate them into the model (Forbes et al., 2024; Kotov et al., 2021; Michelini et al., 2024). Like other symptoms, cognitive abilities are continuously distributed in the population, with the extreme ends of the distribution reflecting dysfunction. Cognitive dysfunction appears in the *DSM* criteria for multiple diagnoses. For example, Criterion A for mild neurocognitive disorder is substantial impairment in cognitive performance in domains of executive function, attention, or memory, and so on; Criterion A for intellectual developmental disorder is deficits in intellectual functions; Criterion A for attention-deficit/hyperactivity disorder (ADHD) includes difficulty with sustained attention; and intellectual impairment is a specifier for autism.

The main difference between cognitive dysfunction and other forms of psychopathology that has prevented them from being included in the HiTOP is the assessment method. To date, the HiTOP has been based exclusively on factor analyses of symptoms assessed by reporters (i.e., via questionnaires and interviews; Kotov et al., 2021). Reporter methods are well suited to measure symptoms of psychopathology that involve maladaptive patterns of thoughts, feelings, and behaviors in everyday life. In contrast, cognitive abilities are a person's performance under optimal conditions and are best assessed with neuropsychological tests. These two sources of information—how a person tends to think, feel, and behave (or recent tendencies that deviate from their usual) and their abilities—are each relevant to psychological functioning (Keefe, 1995; Paulhus & Martin, 1987). Thus, because of the reliance on reporter-based symptom measures rather than neuropsychological tests, the HiTOP provides an incomplete account of mental health and omits major forms of disorders from the *DSM*.

Prior Work on Relations Between Psychopathology and Cognitive Functions

Research relating psychopathology and cognitive abilities is extensive in scope, but this literature has not sufficiently addressed the possibility of integrating cognitive dysfunction into the HiTOP. Most of this research has been on single *DSM* disorders, with results suggesting the presence of widespread cognitive deficiencies. Meta-analyses have concluded there is evidence for dysfunction in nearly all major domains of cognition (e.g., executive functioning, memory, processing speed, visuospatial abilities) for major depressive disorder (Rock et al., 2014; Snyder, 2013), anxiety disorders (Moran, 2016; Shi et al., 2019), eating disorders (Smith et al., 2018; Stedal et al., 2021), obsessive-compulsive disorder (Abramovitch et al., 2013), alcohol and substance use disorders (Crowe et al., 2020; Lee et al., 2019), psychotic disorders (Au-Yeung et al., 2023; Schaefer et al., 2013; Ventura et al., 2010), and bipolar disorder (Bourne et al., 2013; Dickinson et al., 2017). It is difficult to draw firm conclusions from this research, however, because each individual study has typically focused on one or two disorders and often related to just one or two domains of cognition. This narrow focus is a problem because *DSM* diagnoses co-occur more often than not (Caspi et al., 2020; Kessler et al., 2005), which makes it unclear whether the observed impairments are specific to a certain form of psychopathology. Likewise, functioning across cognitive domains tends to be correlated, so it is unclear from this research whether a disorder relates to dysfunction in a specific area of cognition or whether the dysfunction is more pervasive. In sum, by failing to account for intercorrelations between disorders and between cognitive functions, it remains unknown whether cognitive deficits relate to psychopathology in general or specifically to some forms and whether cognitive deficits are global when they occur or some functions are more impaired than others in a given mental disorder.

A relatively small body of research addresses these problems of overlapping symptoms by examining cognitive functions in relation to HiTOP dimensions. Results from this literature are mixed and provide limited insight into how cognition fits into the empirical structure of psychopathology. Some findings suggest cognitive abilities relate more strongly to general versus specific aspects of psychopathology (Pettersson et al., 2021), whereas other findings suggest that general and specific psychopathology show a similar profile of cognitive deficits (Caspi et al., 2014; Southward et al., 2023). Other findings suggest only one dimension is primarily deficient, although in some studies that dimension was externalizing (Du Pont et al., 2019; Nigg et al., 2017; Pettersson et al., 2021) and in others it was psychoticism (Jonas et al., 2024; Stein et al., 2023). There is also some evidence in youth samples that each form of psychopathology associates with a different type of cognitive deficit (Bloemen et al., 2018; Rotstein et al., 2023; Zoupou et al., 2024). The source of discrepancies across these studies is unknown—it could be due to variation in the subsets of disorders that were modeled, the cognitive domains examined, the particular neurocognitive tests administered, or sampling variability. Moreover, because these studies modeled psychopathology and cognition separately, they still do not answer the fundamental question of whether cognitive functions can be integrated into existing HiTOP dimensions or if they form a separate domain(s).

Joint Structure of Psychopathology and Cognitive Functions

The most direct approach to understanding how cognition fits into the structure of psychopathology is to estimate their joint structure. The few studies that have taken this approach have produced mixed results. Inconsistencies in this work stem from at least two sources. The first is the inclusion of thought disorder symptoms, which are associated with some of the most severe cognitive deficiencies (Abramovitch et al., 2021; East-Richard et al., 2020). Studies that did not assess thought disorder symptoms have concluded cognition and psychopathology are separate domains on the basis of the superior global fit of a confirmatory factor model with separate cognitive/psychopathology factors compared with models with joint factors (Eadeh et al., 2021; Rotstein et al., 2023) and findings that cognitive abilities do not load onto a general factor alongside personality, personality disorders, and psychopathology (Littlefield et al., 2021). In contrast, research that did assess thought disorder symptoms supports the integration of cognitive and psychological functions, finding that cognitive abilities and wide-ranging psychopathology load onto a single higher order factor (Moore et al., 2024). Together, this work suggests thought disorder symptoms must be included in a model to integrate cognitive functions and hints at the possibility cognitive deficits fall within the thought disorder spectrum.

A second source of inconsistencies in this literature is a reliance on hypothesis-driven analyses testing only one or two candidate models. Limiting the search space in this way is premature for addressing the novel question of whether cognitive abilities fit into the HiTOP model, particularly when fundamental parts of the model are not represented (e.g., thought disorder). Data-driven, exploratory factor analyses would enable a more exhaustive search of cognition-psychopathology relations and stronger conclusions about the joint structure.

Current Study

The current study aimed to determine how cognitive abilities fit into the empirical structure of psychopathology with a meta-analytic joint factor analysis. We knitted together the fragmentary literature on *DSM* diagnoses and cognitive abilities by combining data from three published meta-analyses into a single correlation matrix. By pooling data across more than 100 studies, we were able to account for method variance (due to, e.g., different neuropsychological tests) and sampling variability that likely contributed to mixed results in previous studies. Furthermore, instead of comparing a small number of hypothesized structures as has been done in all prior joint factor models, we sought a data-driven solution with exploratory factor analysis.

Method

The code and data needed to reproduce our analyses are available on OSF (https://osf.io/mgxht).

Description of data sets

Data from three meta-analyses were combined to create a complete correlation matrix of *DSM* Disorder × Cognitive Functions. Detailed information about the data sets can be found in the original studies, but we describe them briefly below along with adjustments made for the current analysis.

Disorder × Disorder correlations were drawn from Ringwald et al.'s (2023) meta-analysis on the structure of *DSM* disorders. All samples from factor-analytic studies of interviewer-assessed *DSM* disorders in adult samples were included in this meta-analysis. Meta-analytic correlations were estimated by pooling study-level correlations and weighting by sample size with a univariate random-effects approach (Hedges & Vevea, 1998). Source studies included assessments of dichotomous diagnoses and symptom counts, so the correlations were a combination of tetrachoric and Pearson's *r*.

Cognitive Function × Cognitive Function correlations were obtained from Agelink et al.'s (2020) meta-analysis on the structure of cognitive functioning. All studies reporting the factor structure of neuropsychological tests in cognitively healthy adult samples were included. Some cognitive domains from the source studies could not be included in the meta-analytic structural model reported by Agelink and colleagues because of insufficient data. Specifically, because of the requirements of their meta-analytic modeling approach, domains for which pairwise complete correlations were unavailable had to be excluded. For the current study, we added data from another sample, which allowed us to include measures of nonverbal memory and visuospatial abilities that were not in the original meta-analysis.[1] The sample added was the expanded Halstead-Reitan Battery (eHRB) normative data set (Heaton et al., 1991), which is considered to be one of the most comprehensive neuropsychological test batteries administered to a large (N = 460), demographically diverse sample of cognitively healthy adults (Patt et al., 2018). We pooled the raw correlation matrices of samples available for public sharing from Agelink and colleagues' database that had our variables of interest (for a list of contributing studies, see Table S1 in the Supplemental Material available online) along with the eHRB sample using the same univariate random-effects pooling approach used for the Disorder × Disorder correlations. All correlations were Pearson's r.

Disorder × Cognitive Function correlations were obtained from Abramovitch et al.'s (2021) systematic review of meta-analyses on cognitive functioning related to *DSM* disorders. Inclusion criteria were meta-analyses comparing neuropsychological test performance of adults who met criteria for a *DSM* disorder versus nonpsychiatric controls. Unweighted mean effect sizes were reported as Cohen's d. For the current study, we converted all d values to r with a two-step procedure: We first converted d to point-biserial r (Rice & Harris, 2005); then we converted point-biserial r to biserial r (Terrell, 1982). Biserial r was used to appropriately represent associations between the continuous neuropsychological test scores and dichotomous group membership (i.e., clinical vs. control), which was assumed to be drawn from a normally distributed continuum of psychopathology. Conversions at both steps were adjusted for population base rates of the disorders (Hudson et al., 2007; Kessler et al., 2005; van Os & Reininghaus, 2016). Base rates are reported in Table S4.

After making the reported adjustments for our analyses, a total of 35 studies (N = 120,596), 45 studies (N = 54,686), and 63 meta-analyses (N = 144,324 clinical participants) contributed to the Disorder × Disorder, Cognitive Function × Cognitive Function, and Disorder × Cognitive Function correlations, respectively. The number of studies per correlation are reported in Table S3.

Data harmonization

Each meta-analysis contained partially overlapping constructs. Because our analyses required a complete correlation matrix, we could retain only constructs for which every pairwise correlation with other constructs in the model were available. In cases that a correlation was missing, we had to listwise delete one construct in the pair. Decisions about which construct to omit were guided by the goal of maximizing content coverage and recovering as many factors as possible. Specifically, we aimed to include at least two diagnoses per each of the HiTOP superspectra (i.e., externalizing, emotional dysfunction, psychosis; Jonas et al., 2024; Krueger et al., 2021; Watson et al., 2022) for well-defined factors to emerge and to include constructs representing most major domains of cognitive functioning (i.e., executive functioning, attention, memory, processing speed, and visuospatial abilities).

A total of eight *DSM* diagnoses were retained on the basis of an overlap between Abramovitch and Ringwald and colleagues' meta-analyses (Abramovitch et al., 2021; Ringwald et al., 2021). Externalizing disorders included were alcohol use and substance use disorders.[2] Psychoticism spectrum disorders were schizophrenia[3] and bipolar disorders. There were five emotional dysfunction diagnoses in common between the source meta-analyses. However, we omitted posttraumatic stress disorder because keeping it would require dropping a vital cognitive domain (i.e., set shifting). As a result, the emotional dysfunction disorders included were panic disorder, eating disorders (anorexia and bulimia), obsessive-compulsive disorder, and depression.

After exclusions were made on the basis of available disorder correlations, seven cognitive functions were retained. These functions included set shifting, working memory, and fluency within the executive functioning domain, verbal and nonverbal memory in the memory domain, processing speed, and visuospatial abilities. Neuropsychological tests used to assess cognitive functioning domains are in Table 1. As shown in the table, every cognitive domain was measured by multiple tests, ensuring minimal influence of task-specific variance.

Data analysis

To estimate relations among psychopathology and cognitive functions, we fit a series of structural models to the meta-analytic correlation matrix using exploratory factor analysis. Models were estimated in Mplus (Version 8.5; Muthén & Muthén, 2017). The pooled matrix was nonpositive definite because of the inclusion of tetrachoric correlations and because each meta-analytic correlation in the matrix was estimated separately. To

Table 1. Neurocognitive Tests Included in Each Portion of the Pooled Correlation Matrix

Cognitive domain	Neurocognitive tests
Set shifting	• CANTAB (attention-switching task, intra- and extradimensional set shift) • Object alternation task • Trail Making Test Part B • Wisconsin Card Sorting Task
Working memory	• CANTAB (delayed matching to sample, spatial recognition memory, spatial span, spatial working memory) • Corsi block test • Paced Auditory Serial Addition Test • Self-ordered pointing • WAIS-III/WAIS-IV/WAIS-R (arithmetic, digit span, letter-number sequencing) • WMS-III (digit span, letter-number sequencing, spatial span)
Fluency	• COWAT • Design fluency • WAIS-III/WAIS-IV/WAIS-R (similarities)
Verbal memory	• CANTAB (paired associations learning) • CVLT (immediate recall, short-delay free recall, short-delay cued recall, long-delay free recall, long-delay cued recall, long-delay recognition) • Hopkins Verbal Learning Test (delayed recall) • Repeatable Battery for the Assessment of Neuropsychological Status (list recall) • WMS-III/WMS-IV (logical memory immediate recall, logical memory delayed recall, verbal paired-associates immediate recall, verbal paired-associates delayed recall, Word List I, Word List II)
Nonverbal memory	• Benton Visual Retention Test—revised • Figure Memory Test (delayed recall) • ROCF (immediate recall, delayed recall) • WMS-III/WMS-IV (faces immediate recall, faces delayed recall, family pictures immediate recall, family pictures delayed recall, visual reproduction) • WMS-IV (spatial addition, visual reproduction)
Processing speed	• CANTAB (stop-signal reaction-time task) • Conners CPT (reaction time) • Go/no-go test (reaction time) • Stroop test (reaction time) • Trail Making Test Part A • WAIS-III/WAIS-IV/WAIS-R (digit symbol coding, symbol search, digit symbol substitution)
Visuospatial abilities	• Judgment of Line Orientation test • ROCF (copy) • WAIS-III/WAIS-IV/WAIS-R (block design, picture completion)

Note: CANTAB = Cambridge Neuropsychological Test Automated Battery; WAIS = Weschler Adult Intelligence Scale; WMS = Weschler Memory Scale; COWAT = Controlled Word Association Test; CVLT = California Verbal Learning Test; ROCF = Rey-Osterrieth Complex Figure Test; CPT = Continuous Performance Test.

accommodate these conditions, we used unweighted least-squares (ULS) estimation, which does not require positive definiteness (Li, 2016).

Our aim was to identify a hierarchical model with as many levels as possible. To accomplish this, we estimated models with one to eight factors. Given that global fit statistics and standard errors (and associated significance testing) are not available for models with ULS estimation, we evaluated models on the basis of the effect size of factor loadings and factor interpretability. We considered primary loadings for a factor to be those ≥ |.35|. This value is not a strict cutoff but a conservative guideline informed by work indicating loadings between .30 and .40 are meaningful (Floyd & Widaman, 1995). After determining the lowest level of the hierarchy, we correlated factors at subsequent levels to estimate the hierarchical structure (i.e., correlated the general factor with factors from the two-factor model and factors from the two-factor model with factors from the three-factor model). Typically, factor scores are correlated across levels with this modeling approach. However, because factor scores require individual-level data that were unavailable in the pooled correlation matrix, we used congruence coefficients instead following precedence of prior work (Ringwald et al., 2021).

Results

The meta-analytic correlation matrix is shown in Table 2. Several disorders were notably associated with deficits in certain cognitive functions (i.e., *r*s ≥ .30). Focusing on these relatively strong correlations, depression was associated with worse set-shifting and visuospatial abilities, obsessive-compulsive disorder with worse nonverbal memory, alcohol use disorder with worse set shifting, and bipolar disorders with worse verbal memory and processing speed. There were also a number of correlations just below .30. In contrast to these somewhat circumscribed deficits, psychotic disorders were associated with deficits in all cognitive functions.

The hierarchical model of psychopathology and cognitive abilities is shown in Figure 1. Factor loadings and factor intercorrelations for each level are shown in Table 3. All disorders and cognitive functions loaded strongly onto a general factor in the one-factor model (median $\lambda = |.51|$, range = $|.30|$ to $|.64|$). Only eating disorders did not have a primary loading on this factor (i.e., $\lambda < .35$). The median size of disorder factor loadings was slightly lower than cognitive functions ($\lambda = |.44|$ vs. $|.53|$). In the two-factor model, emotional dysfunction disorders split off into a separate factor, with cognitive dysfunction, psychotic disorders, and externalizing disorders marking the first factor. In the three-factor model, externalizing disorders formed a separate factor, whereas psychotic disorders remained on the first factor alongside all cognitive functions. Note that the factor loading for eating disorders fell slightly below our primary loading threshold in the three-factor models ($\lambda = .29$). We considered eating disorders to be a marker of emotional dysfunction nonetheless because of the established empirical basis for this placement in the HiTOP model (Forbush et al., 2017; Watson et al., 2022). Solutions with four or more factors were not interpretable and contained evidence of overextraction (i.e., Heywood cases, singlet factors). Thus, the lowest level of the hierarchy in our final model consisted of three superspectra: psychoticism and cognitive dysfunction, externalizing, and emotional dysfunction. The top marker of the first factor was psychotic disorders, alcohol use disorders for externalizing, and panic disorder for emotional dysfunction.

Discussion

This study used meta-analytic joint factor analysis to unify the piecemeal literature on *DSM* diagnoses and cognitive abilities and answer the question of how cognitive abilities fit into the empirical structure of psychopathology. Our results show that deficiencies in executive functioning, memory, processing speed, and visuospatial abilities in the current HiTOP model are part of the psychoticism superspectrum. These findings lay the foundation for a more comprehensive classification system of psychopathology that includes cognitive abilities, and we provide concrete, actionable recommendations for future research.

Summary of findings and connections to prior work

The principal finding of this study is that cognitive functions can be integrated into the structure of psychopathology. This adds critical knowledge about nosology that was made possible only by combining meta-analyses, building substantially on prior meta-analyses on bivariate associations of *DSM* disorders and cognitive functions (e.g., Abramovitch et al., 2021). In our one-factor model, we found a balanced construct composed of roughly equal parts psychopathology and cognitive abilities. Moreover, a separate cognitive factor never emerged, even after extracting up to eight factors. At the lowest level of our hierarchical model, psychoticism and cognitive abilities formed a single factor, which adds more direct evidence for speculation that cognitive deficits are especially pronounced in the psychosis superspectrum (Jonas et al., 2024; Michelini et al., 2021). This conclusion has been based primarily on indirect comparisons of studies on single *DSM*-defined disorders (e.g., Abramovitch et al., 2021) and a few studies comparing cognitive functioning of psychotic disorders to one or two nonpsychotic disorders (e.g., Reichenberg et al., 2009; Sheffield et al., 2018). Our study goes beyond speculation by incorporating meta-analyses of within-domain associations, analyzing all variables in a joint structural model, and statistically isolating the common variance to show there is indeed a privileged relation of psychoticism to cognitive dysfunction.

Our results support a different conclusion than the few prior studies that have tested a joint factor model of psychopathology and cognitive abilities (Eadeh et al., 2021; Littlefield et al., 2021; Rotstein et al., 2023). Existing evidence suggests they are separate constructs, and none have placed cognition in the psychoticism superspectrum. These studies are not directly comparable to our meta-analysis given that two were in youth samples (Eadeh et al., 2021; Rotstein et al., 2023), and different operationalizations of cognitive dysfunction/psychopathology were used (e.g., self-report psychopathology, a cognitive ability index including standardized college admission test scores; Littlefield et al., 2021). Aside from these sources of variability, there are three other explanations for the divergence in results.

Table 2. Meta-Analytic Correlation Matrix of Disorders and Cognitive Functions

	Cognitive functions							*DSM* disorders						
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Set shifting														
2. Working memory	**.34**													
3. Fluency	**.33**	**.33**												
4. Verbal memory	.26	.22	.24											
5. Nonverbal memory	**.33**	.19	.18	.28										
6. Processing speed	**.57**	.20	.23	.21	.19									
7. Visuospatial abilities	**.42**	.29	.25	.20	**.39**	.30								
8. Eating disorders	−.13	−.13	−.06	−.21	−.21	−.13	−.20							
9. Depression	**−.34**	−.21	−.29	−.24	−.29	−.27	**−.31**	.23						
10. OCD	−.17	−.14	−.16	−.16	**−.31**	−.20	−.16	.19	.30					
11. Panic disorder	−.14	−.13	−.20	−.25	−.14	−.04	−.17	.22	**.39**	.29				
12. AUD	**−.36**	−.28	−.24	−.22	−.23	−.22	−.24	.10	.19	.11	.12			
13. SUD	−.15	−.26	−.08	−.25	−.24	−.18	−.24	.09	.22	.11	.20	**.52**		
14. Bipolar disorders	−.29	−.25	−.28	**−.32**	−.26	**−.30**	−.15	.09	.25	.29	.26	.21	.27	
15. Psychotic disorders	**−.34**	**−.35**	**−.39**	**−.43**	**−.36**	**−.41**	**−.32**	.14	.16	.22	.13	.09	.14	**.38**

Note: Cognitive functions are scored with higher values indicating better functioning. Correlations > |.30| are indicated in bold. Disorder × Disorder correlations include tetrachoric and Pearson's *r*s, Cognitive Function × Cognitive Function correlations are Pearson's *r*, and Disorder × Cognitive Function correlations are biserial *r*. *DSM* = *Diagnostic and Statistical Manual of Mental Disorders*; OCD = obsessive-compulsive disorder; AUD = alcohol use disorder; SUD = substance use disorder.

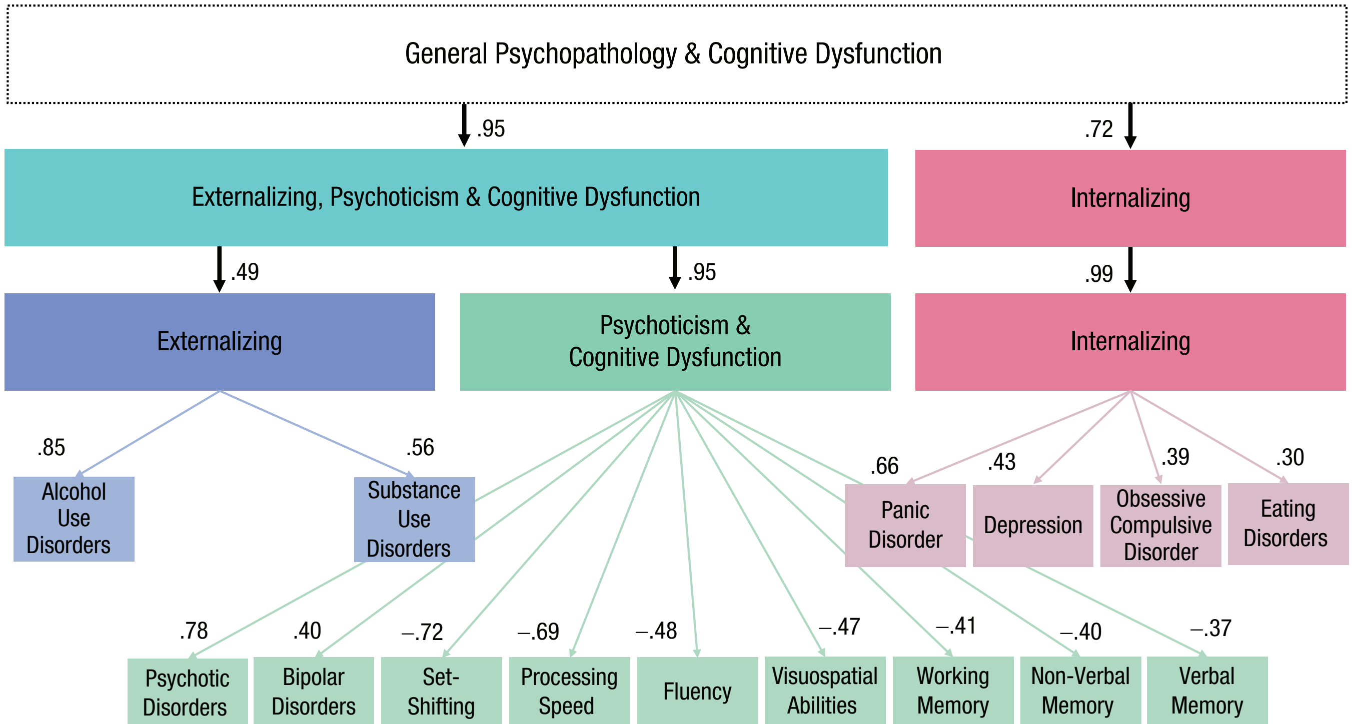

Fig. 1. Meta-analytic hierarchical model of psychopathology and cognitive abilities. Correlations between factors at different levels are congruence coefficients. Standardized factor loadings are shown next to the observed variables.

One reason previous studies failed to support the integration of cognitive functions into psychopathology is that those models may have been more influenced by method variance. Because these studies used one instrument for psychopathology and one for cognition (or a disproportionate number of reporter measures to neuropsychological tests), the separate factors may reflect different measures rather than different constructs. Our meta-analysis pooling across many assessment instruments and cognitive tasks limited measure-specific variance to produce estimates that may be closer to the "true" associations.

A second possible reason for the divergence in findings is the difference in samples studied. The Disorder × Cognition correlations in our study were obtained from a meta-analysis of case-control studies, whereas prior work on the joint structure used unselected samples. Case-control studies, which sample people at the most severe (i.e., diagnosed cases) and least severe ends (i.e., healthy controls) of the psychopathology distribution, may have amplified the differences in cognitive abilities (Fisher et al., 2020; Preacher et al., 2005). This potential effect of sampling may be most pronounced for psychoticism-related disorders given the low base rates in unselected samples. Unselected samples represent psychopathology dimensions better by capturing the middle of the distribution and may provide a more accurate estimate of associations. On the other hand, unselected samples with mostly unimpaired cognition and subclinical levels of psychopathology (e.g., students, Prolific workers, youths sampled before the age of risk for psychosis)—and particularly low representation of psychoticism—may have insufficient variance to detect their associations. Future research can address these issues by examining the joint structure in samples representing a fuller range of psychopathology severity (Stanton et al., 2020).

A third way our study differs from prior work is that we took a fundamentally different, and arguably more rigorous, approach to testing the placement of cognitive abilities in the psychopathology structure. All prior studies used confirmatory factor analysis to test a small number of hypothesized solutions that imposed stringent and unrealistic assumptions of simple structure (i.e., no disorder cross-loadings). In contrast, our exploratory factor-analytic approach relaxed the assumptions of simple structure and allowed for essentially any configuration of cognitive and psychopathology factors to emerge that best fit the data. Thus, our data-driven solution summarizing findings of 80 independent studies and 63 meta-analyses encompassing more than 300,000 participants offers unprecedented evidence that cognitive abilities can, in fact, be integrated into the structure of psychopathology.

Table 3. Factor Loadings and Factor Correlations for the Meta-Analytic Joint Structure of Cognitive Functioning and Psychopathology

Factor loadings	F1		F1	F2		F1	F2	F3
Set shifting	**−.64**	Set shifting	**−.77**	.14	Psychotic disorders	**.78**	.25	.00
Psychotic disorders	**.58**	Processing speed	**−.68**	.18	Set shifting	**−.72**	.07	.12
Visuospatial abilities	**−.54**	Psychotic disorders	**.61**	.01	Processing speed	**−.69**	.04	.15
Processing speed	**−.53**	Visuospatial abilities	**−.51**	.07	Fluency	**−.48**	.03	.07
Nonverbal memory	**−.53**	Working memory	**−.48**	.04	Visuospatial abilities	**−.47**	.07	.08
Bipolar disorders	**.53**	Fluency	**−.46**	.07	Working memory	**−.41**	.13	.03
Depression	**.53**	Nonverbal memory	**−.42**	.20	Bipolar disorders	**.40**	.00	.22
Verbal memory	**−.51**	AUD	**.41**	.09	Nonverbal memory	**−.40**	.03	.21
Working memory	**−.49**	Bipolar disorders	**.41**	.22	Verbal memory	**−.37**	.01	.23
Fluency	**−.49**	Verbal memory	**−.38**	.23	AUD	.03	**.85**	.01
AUD	**.45**	SUD	**.30**	.22	SUD	.00	**.56**	.20
SUD	**.42**	Depression	.29	**.42**	Panic disorder	.01	.01	**.66**
OCD	**.40**	OCD	.18	**.37**	Depression	.26	.02	**.43**
Panic disorder	**.37**	Panic disorder	.01	**.66**	OCD	.23	.10	**.39**
Eating disorders	**.30**	Eating disorders	.13	.29	Eating disorders	.14	.03	**.30**
Factor correlations			F1	F2		F1	F2	F3
		F1	—		F1	—		
		F2	.38	—	F2	.46	—	
					F3	.38	.23	—

Note: Loadings ≥ |.30| are indicated in bold. F1 = one-factor model; F2 = two-factor model; F3 = three-factor model; OCD = obsessive-compulsive disorder; AUD = alcohol use disorder; SUD = substance use disorder.

Interpreting joint factors of cognitive functions and disorders

It is important to note that, like the HiTOP model generally, the model in the current study is descriptive. That is, the factors we found reflect cross-sectional covariance between psychiatric disorders and cognitive functions, agnostic to the sources of covariance. Consequently, our results do not speak to whether (a) cognitive dysfunction is a trait-like predisposing factor for other psychopathology symptoms, (b) cognitive dysfunctions are a state-dependent consequence of other symptoms (e.g., mood-induced memory problems; Abramovitch et al., 2012; Everaert et al., 2022) or its treatment (e.g., antipsychotics impacting verbal learning; Husa et al., 2014), or (c) cognitive dysfunction and other symptoms have shared etiology (e.g., genetics; Smeland et al., 2020; Wootton et al., 2023). Clarifying which of these mechanisms explain the joint cognition-psychoticism factor we found will require longitudinal designs that disentangle state and trait components of cognitive functioning and symptoms and specify their temporal ordering.

Implications and future directions for nosology, research, and clinical practice

Our meta-analysis suggests that the HiTOP's psychoticism superspectrum could be revised to include impaired executive functioning, processing speed, and visuospatial abilities. This revision would align with the 11th edition of the *International Classification of Diseases and Related Health Problems* (World Health Organization, 2019), in which cognition is already a qualifier for a diagnosis of schizophrenia (Gaebel, 2012), and with the perspective of some scholars that cognitive impairments are actually the core pathology of psychoticism (Kahn & Keefe, 2013; Rapoport et al., 2012). However, before this revision can be made, the results need to be replicated in a population-based sample to ensure they are not unduly affected by case-control studies, as noted in the previous section. Additionally, although cognitive abilities fell within the psychosis superspectrum, it remains unclear how they relate to its component psychoticism and detachment spectra. We expect that most cognitive deficiencies in the lower order structure of psychoticism will be in the detachment spectrum. This hypothesis is based on consistent findings indicating that cognitive dysfunction is most closely linked to negative symptoms and disorganization but not reality-distortion symptoms (Dibben et al., 2009; Dominguez et al., 2009; Ventura et al., 2010). It is also plausible that a tripartite structure akin to schizotypy (i.e., trait psychoticism) would emerge (Kwapil & Barrantes-Vidal, 2015; Tandon et al., 2009), with some or all cognitive deficits forming a separate factor alongside detachment (i.e., negative symptoms) and psychoticism (i.e., positive symptoms) spectra. Once structural evidence for joint psychoticism and cognitive dysfunction factors are established, it would require validation before conclusions can be made about its superiority to the alternatives (Forbes et al., 2024).

It is also possible that cognitive abilities will fall in domains other than psychoticism when modeled with wider representations of psychopathology than were available for the current study. In particular, we expect that many cognitive deficiencies will be related to neurocognitive and neurodevelopmental disorders—which are disorders not currently in the HiTOP model. Adding these disorders to the model has been a high priority (Forbes, in press; Michelini et al., 2024). However, one of the biggest barriers to this goal has been the reliance on reporter measures in structural research rather than neuropsychological tests that are best suited to assess the cardinal symptoms of cognitive dysfunction. We also expect that some cognitive abilities that were unavailable for our analyses will link most strongly to externalizing and emotional dysfunction constructs. Namely, deficits in attention, planning, and response inhibition are consistently observed in externalizing psychopathology, and, although the evidence is less consistent, poor episodic memory, response inhibition, and performance monitoring relate to subfactors of emotional dysfunction (Michelini et al., 2021). In sum, future research can build on the precedent we set by modeling a wider range of psychopathology and cognitive abilities, which we anticipate will eventually lead to changes throughout the HiTOP model.

Merging cognitive functions and psychopathology into a unified HiTOP framework can stimulate new hypotheses and accelerate research on their relations. Because the bulk of prior research has been on heterogeneous *DSM*-defined diagnoses, often in isolation and in relation to one or two domains of cognition, it has been difficult to make progress in understanding which cognitive deficits are general or specific to a given form of psychopathology. Mapping cognitive abilities onto more precise phenotypes (i.e., HiTOP dimensions instead of *DSM* diagnoses) can help resolve this issue. For example, locating various cognitive abilities in the psychosis superspectrum would be a more parsimonious way to test whether the cognitive deficits seen across psychoticism-related disorders reflect variation in severity or kind compared with the usual approach of comparing the cognitive abilities of groups with different psychotic disorders (Gotra et al., 2020; Hill et al., 2013; Tamminga et al., 2014). Including cognitive abilities in the HiTOP would also allow us to build on well-established theory and research from cognitive science about the basic processes underlying neuropsychological test performance (Keefe, 1995). Such a bridge between basic science and clinical science would also create a more direct interface between the HiTOP and RDoC, enabling a synergistic approach to understanding psychopathology (Michelini et al., 2021).

Adding cognitive abilities measured by neuropsychological tasks to the HiTOP model could address some problems created by reliance on reporter methods. One of these problems is symptom equifinality. That is, there are symptoms that appear superficially similar but have essential differences that are not captured by reporter measures (Forbes, in press). Most germane to the current study, reporter methods typically do not (or cannot) differentiate symptoms arising from deficit ability versus psychological factors such as motivation or mood. To take one example, although concentration problems are observed in many disorders, they often associate with executive functioning deficits when they co-occur with externalizing symptoms (e.g., ADHD) but less so when they co-occur with emotional dysfunction symptoms (e.g., depression), suggesting distinct underlying mechanisms (Fasmer et al., 2016; Paucke et al., 2021). Incorporating cognitive abilities into the structure could therefore clarify the nature of symptoms in a way that is not currently possible.

Furthermore, the expansion of the HiTOP to include task-based assessments need not be limited to cognitive abilities—many other psychopathology-relevant behaviors measured by nonneuropsychological laboratory tasks could be added to the model. For instance, incentive delay tasks measure variation in reward processing relevant to disinhibited externalizing (Balodis & Potenza, 2015; Plichta & Scheres, 2014), the effort expenditure for rewards task measures behavior related to anhedonia (Treadway et al., 2009), theory-of-mind tasks assess social-cognitive deficits observed in psychoticism or autism (Bora et al., 2009; Senju, 2012), and economic games tap interpersonal tendencies such as spitefulness associated with antagonism (Edershile, 2022). Defining HiTOP constructs on the basis of multimethod data would enrich their nomological networks, resulting in a more valid and useful classification system.

A clinical implication of expanding the definition of psychopathology writ large to include cognitive abilities is that neuropsychological tests should be regularly administered alongside reporter measures—not just when a neurodevelopmental/neurocognitive disorder is suspected, as is often the case. Unfortunately, neuropsychological tests are resource intensive. There are abbreviated and automated tests available that may be more feasible for clinical practice (Berry et al., 2022; Gur, 2001; Keefe, 2004), but most assess a narrow range of content. Another approach is computerized adaptive testing, which could fulfill the need for breadth of efficiency, although only one such assessment of neurocognitive and psychopathology has been introduced to date (Moore et al., 2023). Our results indicate that the continued development and validation of such measures should be a priority. A more accurate and precise mapping of symptom-cognition correlations achieved by further research on their joint structure will allow us to identify such brief, neuropsychological screeners for other psychiatric conditions.

Limitations

Our meta-analysis has some limitations. First, correlations in the model were drawn from different populations (e.g., cognitively healthy samples, epidemiological samples, clinical samples) and therefore may not be invariant. Second, as noted, the inclusion of case-control studies in the Disorder × Cognitive Functions portion of the matrix may have inflated the correlations, especially with psychoticism. Third, and also noted previously, our models were limited by the disorders and cognitive domains available for inclusion. Cognitive domains with strong links to psychopathology that were not included in the current study will be especially critical to examine within the HiTOP model, including attention, planning, and response inhibition. Finally, we used heterogeneous disorders from the *DSM* as markers rather than homogeneous trait dimensions or narrow symptoms. Because of these limitations, our results do not necessarily imply a definitive placement of cognitive abilities in the HiTOP model. Instead, we view our results primarily as proof of concept that the HiTOP can incorporate cognitive abilities specifically and nonreporter measures of psychopathology generally.

Conclusion

This study provides meta-analytic evidence that cognitive abilities can be integrated into the empirical structure of psychopathology. We hope our study is one small step that becomes a giant leap for the HiTOP toward a more complete and useful classification system that expands beyond reporter measures.

Transparency

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Supplemental Material

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Notes

1. Data from the expanded Halstead-Reitan Battery normative data set were not included in the meta-analysis because no studies had reported factor analyses using this sample during the time interval the literature review was conducted.
2. Results for alcohol and nonalcoholic substance use disorders were combined in Abramovitch and colleagues' meta-analysis. We reanalyzed the meta-analyses of alcohol and substance use disorders separately to have two indicators for externalizing. To ensure reliable estimates, effect sizes based on < 200 cases were not included. Effect sizes for the current study were therefore based on four meta-analyses for alcohol use disorder and seven meta-analyses for other substance use disorders. Studies included in the analyses are listed in Table S2 in the Supplemental Material.
3. Schizophrenia and composites of psychotic symptoms (e.g., psychotic episodes, delusional disorder, schizoaffective disorder) were combined in Ringwald and colleagues' meta-analysis.

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