Do cognitive functions belong in the Hierarchical Taxonomy of Psychopathology (HiTOP) 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 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 8 *DSM* disorders and 7 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| - |.64|). At the lowest level were three superspectra including 1) psychosis and cognitive dysfunction, 2) externalizing, and 3) 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.

Do cognitive functions belong in the Hierarchical Taxonomy of Psychopathology (HiTOP) Model? A meta-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), they are strongly predictive of prognosis (McCutcheon et al., 2023), and they 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).

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, 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 *DSM* diagnoses and towards transdiagnostic, dimensional models (Dalgleish et al., 2020).

Because 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 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). Given HiTOP's growing influence on research and clinical practice, the omission of cognitive functions from the model is consequential.

The reason cognitive functions are not included in the HiTOP model is that it is based on factor analyses of signs, symptoms, and traits assessed by reporters. Reporters (e.g., self, informants, clinicians) are well-suited to observe a person's maladaptive patterns of thoughts, feelings, and behaviors in everyday life. In contrast, cognitive abilities reflect performance under optimal conditions and are typically assessed with objective 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 separable and each are relevant to psychological functioning (Keefe, 1995; Paulhus & Martin, 1987). Their distinctiveness means cognitive ability may remain intact in some forms of psychopathology, whereas other forms manifest almost exclusively in cognitive deficiencies. In further support of a distinction between cognitive functions and psychopathology, it has been shown that cognitive dysfunction tends to persist even after other symptoms remit with treatment (Leucht et al., 2013; Reppermund et al., 2009; Semkovska et al., 2019), suggesting there may be independent mechanisms at play.

While distinguishable in many respects, psychopathology symptoms and cognitive deficiencies are also interconnected. Low cognitive abilities can make someone more vulnerable

to psychopathology (e.g., by limiting coping resources)(Pruessner et al., 2020), psychopathology can lead to cognitive impairments (e.g., mood-induced memory problems)(Abramovitch et al., 2012; Everaert et al., 2022), and cognitive dysfunction and psychopathology could have shared etiology (e.g., genetics)(Smeland et al., 2020; Wootton et al., 2023). Thus, there are arguments in favor of conceptualizing cognitive dysfunction as separate from psychopathology or as a fundamental part of it like any other symptom. However, limitations of existing research have prevented an empirical answer to this question of how cognition fits into the structure of psychopathology.

Prior Work on Relations between Psychopathology and Cognitive Functions

Although extensive in scope, research relating psychopathology and cognitive abilities has major methodological limitations. 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 disorders (Bourne et al., 2013; Dickinson et al., 2017). It is difficult to draw firm conclusions from this research though 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 others find 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). 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. Only three studies have taken this approach. In two of these studies, it was concluded that cognition is best conceptualized as a separate domain based on the superior global fit of a confirmatory factor model with separate cognitive/psychopathology factors compared to models with joint factors (Eadeh et al., 2021; Rotstein et al., 2023). The third study arrived at the same conclusion based on the finding that cognitive abilities did not load onto a general factor alongside personality, personality disorders, psychopathology (Littlefield et al., 2021). These studies have at least two limitations that undermine the conclusion that cognition is separate from psychopathology. First, these studies did not assess schizophrenia symptoms, although they are associated with some of the most severe cognitive deficiencies (Abramovitch et al., 2021; East-Richard et al., 2020). Second, these results from hypothesis-driven analyses testing only one or two candidate models may have missed an integrated structure of psychopathology and cognition that fits the data best. Data-driven, exploratory factor analyses would enable a more exhaustive search of cognition-psychopathology relations and stronger conclusions about the joint structure.

Present study

The present study aimed to determine how cognitive abilities fit into the empirical structure of psychopathology with a meta-analytic joint factor analysis. We knit 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 a

hundred 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 like has been done in all prior joint factor models, we sought a data-driven solution with exploratory factor analysis.

Methods

Code and data needed to reproduce our analyses and supplementary materials can be found on the Open Science Framework page for this study (https://osf.io/mgxht/).

Description of datasets

Data from three meta-analyses were combined to create a complete correlation matrix of *DSM* disorders × cognitive functions. Detailed information about the datasets can be found in the original studies, but we describe them briefly below along with adjustments made for the present analysis.

Disorder × disorder correlations were drawn from Ringwald and colleagues' (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 and colleagues' (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 due to insufficient data. Specifically, due to requirements of their meta-analytic modeling approach, domains for which pairwise complete correlations were unavailable had to be excluded. For the present 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 normative dataset (eHRB; 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 the Agelink and colleagues' database that had our variables of interest (see the supplementary materials for a list of contributing studies) along with the eHRB sample using the same univariate random effects pooling approach employed for the disorder × disorder correlations. All correlations were Pearson's *r*.

Disorder × cognitive functions correlations were obtained from Abramovitch and colleagues' (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 non-psychiatric controls. Unweighted mean effect sizes were reported as Cohen's *d*. For the present study, we converted the *d*s to *r*s with a two-step procedure: We first converted the *d*s to point-biserial *r*s (Rice & Harris, 2005), then converted the point-biserial *r*s to biserial *r*s (Terrell, 1982). Biserial *r* was used to

[1] Data from the eHRB normative dataset was 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.

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 the supplementary materials.

After making the reported adjustments for our analyses, the total number of studies contributing to the disorder × disorder correlations was 35 (N = 120,596), 45 studies (N = 54,686) for the cognitive function × cognitive function correlations, and 63 meta-analyses (N = 144,324 clinical participants) for the disorders × cognitive functions correlations. Number of studies per correlation are reported in the supplementary materials.

Data harmonization

Each meta-analysis contained partially overlapping constructs. Because our analyses required a complete correlation matrix, we could only retain 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, visuospatial abilities).

A total of 8 *DSM* diagnoses were retained based on 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 5 emotional dysfunction diagnoses in common between the source meta-analyses. However, we omitted PTSD 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, 7 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 . The pooled matrix was non-positive definite because each meta-analytic correlation in the matrix was estimated separately and due the inclusion of

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[2] Results for alcohol and non-alcoholic substance use disorders were combined in Abramovitch and colleagues' meta-analysis. We re-analyzed 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 present study were therefore based on four meta-analyses for alcohol use disorder and 7 meta-analyses for other substance use disorders. Studies included in the analyses are listed in the supplementary materials.

[3] Schizophrenia and composites of psychotic symptoms (e.g., psychotic episodes, delusional disorder, schizoaffective disorder) were combined in Ringwald and colleagues' meta-analysis.

tetrachoric correlations. To accommodate these conditions, we used unweighted least squares (ULS) estimator, 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 1-8 factors. Given that global fit statistics and standard errors (and associated significance testing) are not available for models with ULS estimation, we evaluated models based on the effect size of factor loadings and factor interpretability. We considered primary loadings for a factor to be those $\geq |.35|$. 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. But since 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., 2023).

Results

The meta-analytic correlation matrix is in Table 2. Several disorders were notably associated with deficits in certain cognitive functions (i.e., *r*s $\geq$ .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 in Table 3. All disorders and cognitive functions loaded strongly onto a general factor in the one-factor model (median λ= |.51|, range = |.30| - |.64|). Only eating disorders did not have a primary loading on this factor (i.e., $\lambda < .35$). The median size of disorders factor loadings was slightly lower than cognitive functions (λ= |.44| versus |.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. 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: 1) psychoticism and cognitive dysfunction, 2) externalizing, and 3) 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 in the current HiTOP model, deficiencies in executive functioning, memory, processing speed, and visuospatial abilities 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. Bivariate associations for several disorders and cognitive abilities were substantial with correlations of up to r = -.43. Also, 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 and a few studies comparing cognitive functioning of psychotic disorders to one or two non-psychotic disorders (e.g., Reichenberg et al., 2009; Sheffield et al., 2018). Our meta-analysis goes beyond speculation by pooling these studies together into a single 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 suggested 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 are variability, there are three other explanations for the divergence in results. 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 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 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 over 300,000 participants offers unprecedented evidence that cognitive abilities can, in fact, be integrated into the structure of psychopathology.

Implications and Future Directions for Nosology, Research, and Clinical Practice

Our meta-analysis suggests that HiTOP's psychoticism superspectrum could be revised to include impaired executive functioning, processing speed, and visuospatial abilities. This revision would align with the ICD-11 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 in the lower-order structure of psychoticism, most cognitive deficiencies 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 representation of psychopathology than were available for the present 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., in press). 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 relates 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 heterogenous *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 on to 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 to 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 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, 2021). Most germane to the current study, reporter methods typically do not (or cannot) differentiate symptoms arising from deficit ability versus psychological factors like 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 HiTOP to include task-based assessments need not be limited to cognitive abilities– many other psychopathology-relevant behaviors measured by non-neuropsychological 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 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 like spitefulness associated with antagonism (Edershile, 2022). Defining HiTOP constructs based on multi-method 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 et al., 2014; Keefe, 2004), but our results reinforce a need to develop more abbreviated and valid instruments for this purpose. Another way to make neuropsychological testing more feasible would be to administer single tasks in a modular

fashion when certain symptoms are endorsed in the same way optional subscales of some clinical interviews or self-reports are currently used. 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. Finally, we used heterogenous DSM disorders as markers rather than homogenous trait dimensions or narrow symptoms. Due to 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 HiTOP can incorporate cognitive abilities specifically, and non-reporter measures of psychopathology generally.

Conclusions

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 HiTOP towards a more complete and useful classification system that expands beyond reporter measures.

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Table 1. Neurocognitive tests included in each portion of the pooled correlation matrix.

Cognitive Domain	Neurocognitive Tests
Set-shifting	▪ CANTAB (Attention Switching Task, Intra-Extra Dimensional 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 Task Reaction Time) ▪ 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	▪ Line Orientation Test ▪ ROCF (Copy) ▪ WAIS III/WAIS-IV/WAIS-R (Block Design, Picture Completion)

Note. CANTAB = Cambridge Neuropsychological Test Automated Battery; COWAT = Controlled Word Association Test; CVLT = California Verbal Learning Test; WMS = Weschler Memory Scale; WAIS = Weschler Adult Intelligence Scale.

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						
1. 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**

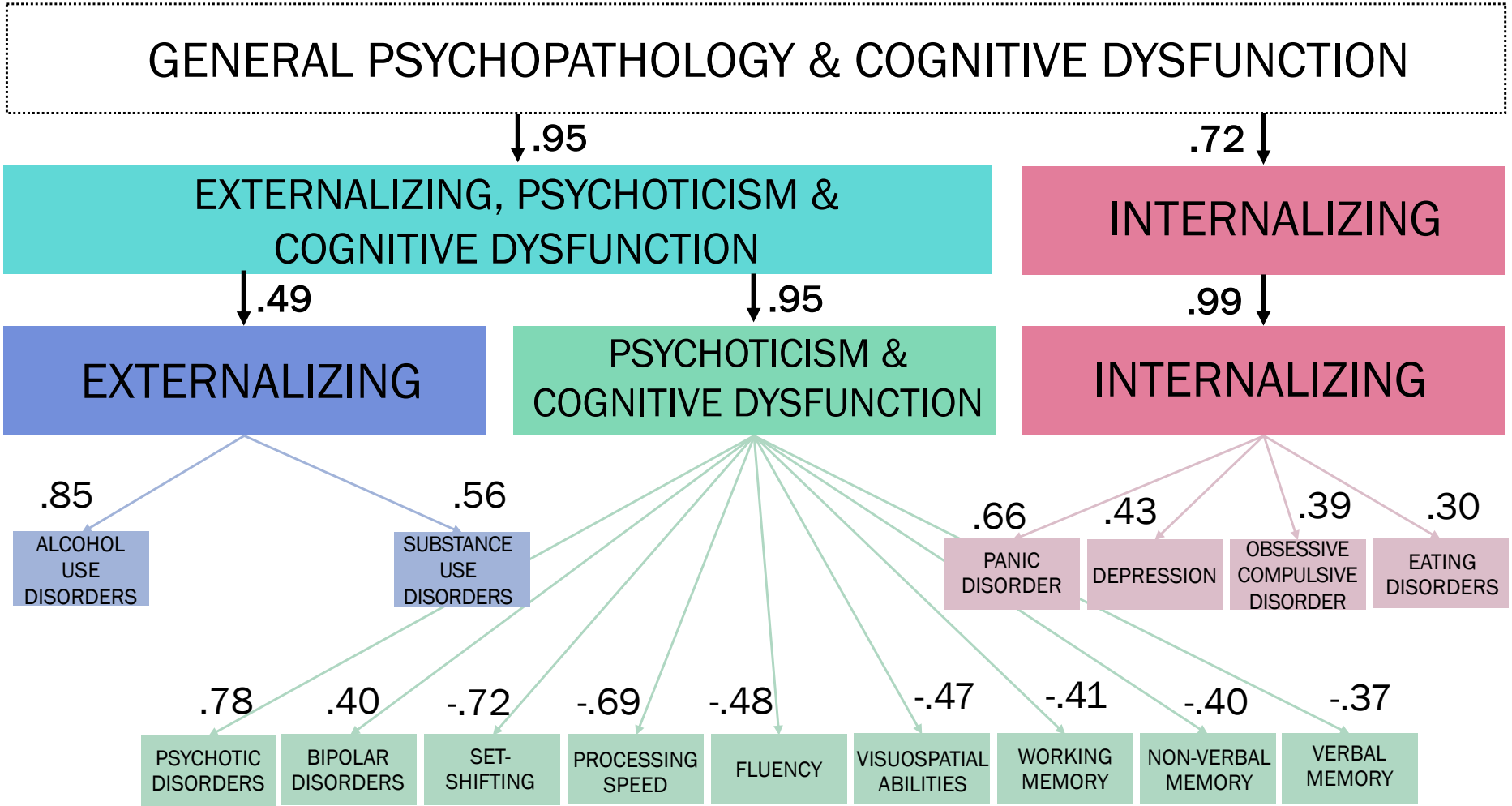
Note. Cognitive functions are scored with higher values indicating better functioning. Correlations > .30 are underlined. Disorder × disorder correlations include tetrachoric and Pearson's *r*s, cognitive function × cognitive function correlations are Pearson's *r*, disorders × cognitive functions correlations are biserial *r*. OCD = Obsessive compulsive disorder, AUD = alcohol use disorders, SUD = substance use disorders.

Table 3. Factor loadings and factor correlations for the meta-analytic joint structure of cognitive functioning and psychopathology

	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 ≥ |.35| are bolded. OCD = Obsessive compulsive disorder, AUD = alcohol use disorders, SUD = substance use disorders.

Figure 1. Meta-analytic hierarchical model of psychopathology and cognitive abilities.



Note. Correlations between factors at different levels are congruence coefficients. Standardized factor loadings are shown next to the observed variables.