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Review

The C Factor: Cognitive dysfunction as a transdiagnostic dimension in psychopathology

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ABSTRACT

Research into cognitive functions across psychological disorders suggests that cognitive deficiencies may be present across multiple disorders, potentially pointing to a transdiagnostic phenomenon. More recently, a single dimension model of psychopathology, the p factor, has been proposed, in which cognitive deficits are thought to be an intrinsic construct, assumed to be transdiagnostic. However, no systematic investigation to date tested this hypothesis. The aim of the present study was to systematically review meta-analyses to assess the hypothesis that the C factor (cognitive dysfunction) is transdiagnostic in psychopathology and review potential moderators that may account for such a phenomenon. We conducted a systematic review of meta-analyses examining cognitive function across all disorders for which data were available. Included meta-analyses (n = 82), comprising 97 clinical samples, yielded 1,055 effect sizes. Twelve major disorders/categories (e.g., bipolar disorder, substance use disorders) were included, comprising 29 distinct clinical entities (e.g., euthymic bipolar disorder; alcohol use disorder). Results show that all disorders reviewed are associated with underperformance across cognitive domains, supporting the hypothesis that the C factor (or cognitive dysfunction) is a transdiagnostic factor related to p. To examine moderators that may explain or contribute to c, we first consider important interpretative limitations of neuropsychological data in psychopathology. More crucially, we review oft-neglected motivational and emotional transdiagnostic constructs of p, as prominent contributing constructs to the C factor. These constructs are offered as a roadmap for future research examining these constructs related to p, that contribute, and may account for cognitive dysfunctions in psychopathology.

1. Introduction

1.1. Neuropsychological testing and psychopathology

Neuropsychology emerged in the 1940s and was recognized as a distinct discipline in the 1960s (Bilder, 2011) at a time when X-rays and electroencephalography (EEG) were the only imaging techniques available to identify brain damage. Neuropsychological measures were used then to aid in localizing damage or insult to brain regions prior to brain surgery (Ruff, 2003). However, even today, neuropsychological testing may be the only "…*objective evaluation of behavior*…" (Keef, 1995, p. 7), and perhaps the only means of objective assessment of brain-behavior relationships available to psychologists, neurologists, and psychiatrists. Alongside the development of increasingly sophisticated functional brain imaging technologies in the late 1980s and early 1990s, neuropsychological measures have become increasingly incorporated in studies endeavoring to confirm, rather than explore, regional differences in brain activity between clinical and non-clinical samples (Abramovitch & Schweiger, 2015). This trend was followed by studies exploring cognitive functions in clinical samples without imaging, but with the same aim in mind, namely, the confirmation of localized brain deficiencies as the neuroanatomical substrates of psychopathology, and reaffirmation of contemporary neurobiological models of DSM disorders. In fact, most neuropsychological studies in the context of psychopathology discuss their results in light of purported disorder-specific neurobiological models, and very rarely in the context of general psychopathological mechanisms and prevailing psychological models. Nevertheless, the utilization of neuropsychological tests to localize specific 'deficient' brain regions has been criticized for decades and perceived as fundamentally misguided use and interpretation of such test data (e.g., Farah, 1994).

This misconception may be particularly relevant in the context of

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higher order executive functions (Ruff, 2003), a domain that suffers the most from task impurity and multicollinearity (Miyake & Friedman, 2012). This criticism notwithstanding, the focus on localization and specification continued unabated, as research in Cognitive Neuroscience thrived, benefitting from refined assessment of cognitive functions and the ever more widespread use of imaging technology. This trend, which in the context of MRI has been recently criticized as 'neo-phrenology' (Uttal, 2011), promoted extensive utilization of cognitive tests in psychopathology research. And as contemporary psychiatry intensified the search for neurophysiological and neuroanatomical bases of psychopathology, neuropsychology stepped into the fray with the goal of discovering cognitive/behavioral syndromes which could be associated with specific psychiatric disorders. The identification of such syndromes would allow then, it is hoped, for improved classification, early detection and subsequently even novel therapeutic interventions.

1.2. Neuropsychological assessment: A new hope?

The next step in the evolution of utilizing neuropsychological testing in psychopathology was to coopt contemporary imaging and genetic technologies in an effort to identify *disorder-specific* biological and cognitive markers of psychopathology. The drive to find such biologically based markers (i.e., 'biomarkers') that are adequately sensitive and specific to a particular disorder, lies at the heart of psychopathology research since the DSM-IV, as psychopathology research became increasingly influenced by the biomedical model (Deacon, 2013). Whereas the search for biomarkers of psychopathology began in the 1970s and the 1980s with studies assessing such candidates as cortisol levels and rapid eye movement latency (Lilienfeld & Treadway, 2016), the interest in disorder-specific cognitive markers emerged sometime later and became prominent in the past two decades. It is noteworthy that half a century later, no disorder-specific biomarker, or cognitive marker, for psychopathology has been found to have satisfactory levels of specificity, sensitivity, and predictive validity, or to have a diagnostic or therapeutic utility.

The consideration of neuropsychological test results as means of discovering biomarkers, equivalent to physiological and neurobiological measurements, has been bolstered by the present-day vision of the United States National Institute of Mental Health (NIMH), Research Domain Criteria (RDoC) endeavor, explicitly conceptualizing cognitive functions, assessed by neuropsychological tests, as objective as functional imaging data (Insel, 2014). This reinvigorated pursuit of disorder-specific cognitive endophenotypes led to one of the more ironic aspects in the field of neuropsychology, where based on familial studies, 'response inhibition', has been suggested as a *disorder-specific etiological* endophenotypic marker of at least eight different DSM disorders, including attention deficit/hyperactivity disorder (ADHD; Slaats-Willemse, Swaab-Barneveld, de Sonneville, van der Meulen, & Buitelaar, 2003), borderline personality disorder (BPD; McCloskey et al., 2009), bipolar disorder (BP; Bora, Yucel, & Pantelis, 2009a), schizophrenia (SCZ; Turetsky et al., 2007), obsessive-compulsive disorder (OCD; Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005), autism spectrum disorder (ASD; Schmitt et al., 2019), trichotillomania (Odlaug, Chamberlain, Derbyshire, Leppink, & Grant, 2014) and schizotypal personality disorder (SCZPD; Cadenhead, Light, Geyer, McDowell, & Braff, 2002).

However, this vast body of literature did not advance our understanding of the etiology, course, comorbidity, prognosis, diagnosis or the best intervention of any specific psychopathology in a meaningful way (Iacono, 2018; Kapur, Phillips, & Insel, 2012; Lilienfeld & Treadway, 2016; Scarr et al., 2015; Venkatasubramanian & Keshavan, 2016). In fact, it has been suggested that the field of neuropsychiatry has exhausted its search for diagnostic/etiological biomarkers as means of generating a biological driven psychiatric classification system, and that currently the field should shift its attention to prediction of treatment response (Boksa, 2013). The fact that the search for the Holy Grail of distinct specific neuropsychological profiles in DSM disorders (be it biomarkers or the search for familial endophenotypes) has not been fruitful is not surprising given that underperformance on neuropsychological tests has been identified in multiple disorders. Cumulative evidence emerged in the last decade suggesting that in the context of any psychopathology, cognitive deficiencies across domains may be the rule rather than the exception (Bloemen et al., 2018; Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari, & Lonnqvist, 2008; Doyle et al., 2018; East-Richard, Alexandra, Nadeau, & Cellard, 2019; Millan et al., 2012; Snyder, Miyake, & Hankin, 2015). Underperformance on a host of neuropsychological tests and corresponding domains has been documented in multiple disorders including, but not limited to, ADHD (Pievsky & McGrath, 2018), bipolar disorder I (BD-I) and bipolar Disorder II (BD-II; Bora, Yucel, Pantelis, & Berk, 2011; R. S. Lee et al., 2014) SCZ (Schaefer, Giangrande, Weinberger, & Dickinson, 2013), schizoaffective disorder (Bora, Yucel, & Pantelis, 2009b), major depressive disorder (MDD; Ahern & Semkovska, 2017), OCD (Abramovitch, Abramowitz, & Mittelman, 2013), post-traumatic stress disorder (PTSD; J. C. Scott et al., 2015), panic disorder (PD; O'Sullivan & Newman, 2014), eating disorders (EDs) including anorexia nervosa and bulimia nervosa (Hirst et al., 2017), alcohol and non-alcohol related substance use disorders (SUDs; Smith, Mattick, Jamadar, & Iredale, 2014), Tourette's syndrome (TS; Morand-Beaulieu et al., 2017), ASD (Demetriou et al., 2018), and several personality disorders (Garcia-Villamisar, Dattilo, & Garcia-Martinez, 2017). Similarly, research utilizing dimensional operationalization of psychopathology reveals deficient performance on neuropsychological tests to be associated with elevated levels of schizotypy (Siddi, Petretto, & Preti, 2017), rumination (Yang, Cao, Shields, Teng, & Liu, 2017), psychosis (Bora et al., 2009b), suicidal behavior (Richard-Devantoy, Berlim, & Jollant, 2014), anxiety (Moran, 2016), and psychopathy (Ogilvie, Stewart, Chan, & Shum, 2011).

1.3. The transdiagnostic nature of cognitive deficits

A few studies conducted selective reviews of cognitive functions associated with psychopathology and found unequivocal support for the ubiquity of deficient cognitive functioning among clinical populations when compared with controls. For example, Wright, Lipszyc, Dupuis, Thayapararajah, and Schachar (2014) conducted a meta-analysis examining response inhibition as assessed by Go/No-Go tests across disorders. The authors found small to medium effect sizes across all eleven disorders examined, reflecting underperformance across clinical samples. Similar results were found by the same group in a meta-analysis examining inhibitory functions as assessed by the Stop Signal Task across eleven disorders (Lipszyc & Schachar, 2010). An earlier cross-diagnostic review of cognitive functions across a number of degenerative diseases, neurological disorders, and three psychiatric disorders has been published in a book by Zakzanis and colleagues (Zakzanis, Leach, & Kaplan, 1999), entitled *Neuropsychological Differential Diagnosis*. This remarkably thorough work systematically reviewed the literature on major cognitive functions in dementias, primary progressive aphasia, Parkinson's disease, Huntington's disease, multiple sclerosis, mild traumatic brain injury (mTBI), as well as MDD, OCD and SCZ. The authors reported cognitive deficiencies across conditions, with medium effect sizes for OCD, and MDD, large effect sizes in SCZ, and much larger effect sizes for most degenerative conditions. Interestingly, mTBI was associated with nearly identical effect sizes for cognitive dysfunction as MDD across domains. The authors concluded that they did not identify disorder-specific neuropsychological profiles and that ultimately diagnosis of psychiatric disorders based on neuropsychological test data is not viable (Zakzanis et al., 1999).

Focusing on depressive and anxiety disorders, Castaneda et al. (2008) systematically reviewed the literature on cognitive dysfunction and concluded that evidence for deficient neuropsychological test performance was found across anxiety and depressive disorders. Snyder et al. (2015) conducted a selective review of meta-analyses examining

executive functions across disorders. The authors found executive function deficiencies across all seven disorders reviewed (i.e., ADHD, BP, SUDs, MDD, OCD, PTSD, and SCZ). Echoing previous findings, medium effect sizes were reported across disorders, except for SCZ for which a large effect size was found (Snyder, Miyake et al., 2015). More recently, East-Richard et al. (2019) conducted a review of 36 meta analyses (publication years 2002-2016) examining cognitive function in SCZ, ADHD, ASD, BP, MDD, OCD, and PTSD. The authors determined that deficient cognitive functioning is present across all the included disorders, and across age groups and concluded that deficient executive functions (and episodic memory) may be considered transdiagnostic markers of psychopathology (East-Richard et al., 2019). In light of the foregoing, we introduce the term 'C factor' to denote cognitive dysfunction in psychopathology and explore its nature below.

1.4. The C factor and p factor

Additional evidence for the non-specific nature of the association between cognitive dysfunction and psychopathology appears in cross-sectional and longitudinal studies. A number of studies demonstrated that cognitive dysfunction is associated with the presence of psychopathology and with overall psychopathology-related severity/burden, but not with a specific diagnosis or disorder-specific symptom severity, in both adults and youth samples (David, Zammit, Lewis, Dalman, & Allebeck, 2008; Doyle et al., 2018; McGrath et al., 2016; Stordal et al., 2005). These studies are in line with the conceptual framework of the p factor. The p factor model, as proposed by Caspi et al. (2014), suggests a superordinate factor of psychopathology that has stronger explanatory power than disorder-specific factors, which together with a more specific internalizing-externalizing subfactor is largely viewed as a powerful contemporary model of psychopathology. However, although the validity of the p factor model has been demonstrated across multiple studies, including genetic research (Allegrini et al., 2020), it has also been subjected to some criticism (Fried, Greene, & Eaton, 2021). In their seminal study, Caspi et al. (2014) analyzed neuropsychological test data and demonstrated how cognitive dysfunction was associated with the p factor. Specifically, the authors utilized data from neuropsychological tests to examine the association between cognitive functions and the p factor. Their results demonstrated that performance scores across all cognitive domains assessed were significantly and negatively correlated with the p factor score (small effect sizes). This finding led the authors to conclude that cognitive dysfunction may be transdiagnostic and should be considered a part of the p factor (Caspi et al., 2014). Subsequent studies replicated these findings, both confirming the strength of p' as a model of psychopathology (Snyder, Young, & Hankin, 2017), as well as lending support to the conclusion that cognitive dysfunction may be closely related to p'. These findings were identified in clinical (Martel et al., 2017), as well as in community samples (M. Wagner et al., 2018). However, it remains to be determined whether the C factor is ubiquitous in psychopathology, and the nature of the association between p and c are yet to be systematically examined.

1.5. The present investigation

To our knowledge, no comprehensive systematic review pertaining to all cognitive functions across all disorders has been published to date. Furthermore, to our knowledge there is no integrative discussion as to the putative factors accounting for this phenomenon vis a vis a transdiagnostic perspective. That is, explanations for the presence of cognitive dysfunction in psychopathology are almost always disorder-specific and tend to focus on neurobiological correlates or disorder-specific psychopathological mechanisms. To address this gap in the literature, the primary aim of the present study was to systematically review all available meta-analyses comparing cognitive functions across DSM disorders to non-clinical controls, and to provide detailed information to researchers and clinicians regarding the reported magnitude of cognitive dysfunctions for each disorder.

The secondary aim of this study was to facilitate future research by providing an integrative discussion as to the putative etiological accounts for the ubiquity of cognitive dysfunction in psychopathology, including factors that were not previously discussed in this context such as motivation, stigma and self-efficacy. We hypothesize that such a review will result in reconsideration of cognitive dysfunction in psychopathology not as merely reflecting specific brain pathology related to a priori reduced availability of resources. Rather, it may well emerge that these deficits are meaningfully related to transdiagnostic features of psychopathology such as reduced motivation, emotional states, and pharmacotherapy issues that are inherent to psychopathology.

Finally, given the prevalence of misconceptions regarding neuropsychological testing, this review provides important contextual grounds outlining some major methodological and conceptual limitations of neuropsychological cognitive testing in the extant literature, including the oft neglected topic of magnitude of cognitive dysfunction and its interpretations, the lack of working definition of cognitive deficit or impairment in the context of psychopathology, and the ecological validity of neuropsychological tests in psychopathology.

2. Methods

A systematic literature search identifying meta-analyses of cognitive functioning in DSM disorders was conducted using NCBI-PubMed, ISI Web of Science, and PsycInfo databases as well as individual publication reference lists through October 2020. Keywords included 'meta-analysis', 'review', 'systematic review', and 'quantitative review', together with keywords including terms such as 'disorder', 'psychopathology', and individual disorders (e.g., 'depression', 'ADHD', 'OCD'). Keywords relating to cognitive functions included general terms such as 'cognitive function', 'neuropsych*', 'assessment', 'deficit', 'impairment', as well as specific terminology related to neuropsychological domains (e.g., 'executive function', 'memory', 'attention'). Two research assistants were trained by the first author on literature search and data extraction. All stages of the literature search and data extraction were closely supervised by the first author. Inclusion criteria included a meta-analysis assessing neuropsychological test performance of adult clinical samples diagnosed with a primary DSM disorder, versus non-psychiatric control samples. Included manuscripts had to be in English, published in a peer-reviewed journal, examining at least one cognitive domain or neuropsychological test. Meta-analyses that provided information regarding effect sizes exclusively for indexes (e.g., processing speed) but not individual tests, were included only if the individual sub-domains/outcome measures comprising the index were reported. In a handful of cases where indexes included different subdomains under the same index, and no information for individual outcome measures or subdomains were reported, such indexes were excluded. Effect sizes related to modified neuropsychological measures (e.g., emotional Stroop), or cognitive tasks that were not subject to psychometric work and/or for which the construct validity was not known or not clear, were not recorded.

Given that decision-making tasks do not meet criteria for classic neuropsychological tasks and that decision-making tasks incorporate multiple intercorrelated and overlapping cognitive domains (e.g., working memory, attention, planning, inhibition) these tasks were determined to be outside the scope of the present investigation. In cases where studies presented only index scores that included neuropsychological tasks, together with tasks that were subject to exclusion, such studies were excluded if information regarding the valid neuropsychological measure was not available separately. Studies that did not use a formal screening procedure to establish diagnoses, or that included a sample that was comprised of participants for whom diagnosis was established together with participants for whom inclusion was determined using other means (e.g., cut off scores on a symptomatic measure), were excluded. Studies that included youth and adults without

providing separate effect sizes for the adult samples were excluded. In both of the latter cases, if attempts to obtain separate data by contacting authors failed, these studies were excluded. Meta analyses focusing on developmental disorders, degenerative disorders, and neurological/medical disorders were excluded. We excluded pediatric meta analyses, and meta analyses focusing solely on older adult samples with psychopathology. Meta analyses comparing two clinical groups with no non-psychiatric control groups, or ones that compared clinical samples to non-standard non-psychiatric control samples (e.g., specific at-risk populations, subclinical, fully remitted) were excluded. Although studies reviewing specific disorder stages (e.g., first episode, euthymic status) were included, highly selective meta analyses (e.g., meta analyses that exclusively investigated incarcerated individuals) were excluded. Our initial search yielded 197 meta analyses, of which 115 were excluded, resulting in 82 meta analytic studies (Fig. 1). Reasons for exclusion included the study being a qualitative systematic review (n = 23), lack of non-clinical comparison group (n = 22), studies including exclusively pediatric samples (n = 16), non-representative highly selective clinical samples (n = 10), mixed pediatric & adult samples from which separate data from authors was not available (n = 11), studies including exclusively non-neuropsychological/non-standard tasks (n = 10), no clinical group (n = 5), lack of formal diagnostic procedure (n = 4), no cognitive tasks (e.g., cognitive functions assessed via self-report questionnaires; n = 5), mixed clinical samples (n = 3), longitudinal (within group) analyses (n = 4), and insufficient data that was not obtainable via study authors (n = 1). From the 82 studies included, meta analytic information from 97 clinical samples was available, with a total of 1,055 effect sizes recorded. Effect size magnitude was interpreted based on guidelines provided by Cohen (1988). Specifically, small, medium, and large effects correspond to standardized mean difference (Cohen's d) values of .2, .5, and .8.

The following variables were recorded: year of publication, primary disorder (e.g., MDD) or disorder category (e.g., affective disorders), specifier/status (e.g., affective disorders with psychotic features; euthymic bipolar), overall number of studies included in the meta-analysis, overall clinical n, overall control n, neuropsychological domain (e.g., executive function), neuropsychological sub-domain (e.g., set-shifting), neuropsychological test name (e.g., WCST[1]), outcome measures (e.g., number of words, number of seconds), confidence interval, and information regarding heterogeneity. To be able to accurately report effect sizes for major domains and subdomains, we recorded the original domain name from each study, and then reviewed all category titles for accuracy given the test or tests included. In cases where we identified errors, such as where authors would term working memory tests as memory tests, or visuomotor skills for a test of processing speed, we aligned those effect sizes with the correct terminology to be included under the appropriate domains. The major domains categories were EF, memory, processing speed, attention, visuospatial functions, and sub domains for EF were response inhibition, working memory, set shifting, planning, fluency, and verbal and non-verbal memory.

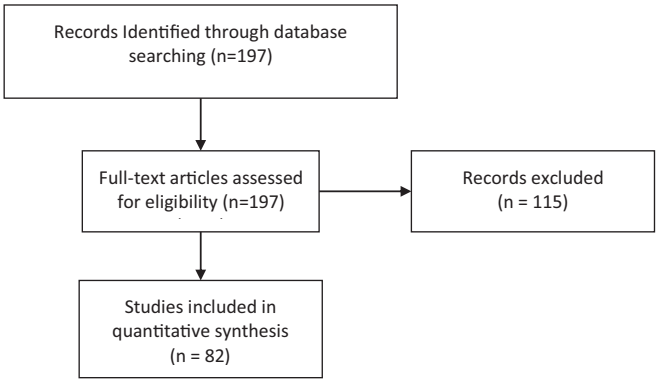


Fig. 1. PRISMA flow diagram of literature search and screening process

[1] Given the scope of the present investigation, results are reported on the domain (e.g., memory), and subdomains (e.g., verbal memory) levels. Performance on individual neuropsychological tests or outcomes are not reported.

3. Results

The 97 meta-analyses included in the present study, the conditions assessed, the number of studies included in each meta-analysis, the number of clinical participants and the scope of each of the 97 meta-analyses included are presented in Table 1. These studies examined 12 primary disorder categories: ADHD (k=7), ASD (k=2), BD (k=18), eating disorders (k=11), depression (k=12), OCD (k=5), PD (k=1), personality disorders (k=3), PTSD (k=2), SCZ (k=18), substance related and addictive disorders including gambling disorder (GD; k=17), and Tourette's syndrome (k=1), comprising a total of 29 separate distinct clinical populations, including specifiers/status (e.g., 'first episode', 'drug naïve', 'euthymic', specific SUDs). Out of the 97 meta-analyses included, 53% (k=51) were comprehensive studies assessing all available neuropsychological information; 14% (k=14) focused on executive functions; 31% (k=30) focused on specific executive functions including inhibitory functions (k=14), set shifting/cognitive flexibility/verbal fluency (k=9), and working memory (k=3); 4% (k=4) assessed memory functions, 1% (k=1) examined processing speed, and 1% (k=1) examined attention functions (Table 1). Publications years ranged from 1997 to 2020, with only five studies (6%) published before the year 2000, including one on depression, and four studies on SCZ.

Results of this review are presented in 3 different ways. First, we present results for major neuropsychological domains and subdomains for primary disorders. Table 2 presents unweighted mean effect sizes for all 10 neuropsychological subdomains across disorders. These were generated by calculating simple unweighted mean scores across effect sizes for each domain or subdomain. We also provide a series of graphs depicting effect sizes for each available domain and subdomain for each disorder category (Fig. 2a to 2m). Finally, a series of figures was generated each depicting a neuropsychological domain trans-diagnostically (Fig. 3a to 3j).

3.1. Major conditions, major domains

3.1.1. Executive function

Across disorders, small effect sizes for executive function (d range .33-.49) were found for ADHD, TS, OCD, PD, PTSD, ED, ASPD, and SUD. Medium effect sizes were found for ASD, Gambling Disorder (GD), BD, depression, and BPD (d range .52-.70). A large effect size (d =.85) was found for schizophrenia.

3.1.1.1. Set shifting. Small effect sizes were found for ADHD, OCD, PD, ED, ASPD, BPD, and SUD (d range .27-.42). Medium effect sizes for set shifting were found for ASD, BD, and depression (d range .50-.66). A large effect size was found for SCZ (d=.80). Information regarding set shifting was not available for TS, GD, and PTSD.

3.1.1.2. Response inhibition. Small effect sizes were found for ADHD, TS, ASD, OCD, ED, and SUD (d range .33-.49). Medium effect sizes for response inhibition were found for GD, BD, ASPD, and SCZ (d range .51-.69). A large effect size was found for depression (d=.83). Information regarding response inhibition was not available for PD, BPD, and PTSD.

3.1.1.3. Working memory. Small effect sizes were found for ADHD,

Table 1
Meta-analyses of cognitive function in DSM disorders included in the present study.

Disorder/Category	Meta-analysis	Scope/focus	Specifier	*K*	Clinical sample n
ADHD	Alderson, Kasper, Hudec, & Patros, 2013	Working memory	ADHD	38	980
	Bálint et al., 2009	Attention	ADHD	25	1,711
	Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005	Executive functions	ADHD	13	641
	Hervey, Epstein, & Curry, 2004	Comprehensive	ADHD	33	–
	Lijffijt, Kenemans, Verbaten, & van Engeland, 2005	Inhibition	ADHD	6	127
	Schoechlin and Engel, 2005	Comprehensive	ADHD	24	867
	Skodzik et al., 2017	Long-Term memory	ADHD	19	780
ASD	Demetriou et al., 2018	Executive functions	Autism Spectrum Disorder	235[a]	6,816[a]
	Westwood, Stahl, Mandy, & Tchanturia, 2016	Set Shifting	Autism Spectrum Disorder	24	–
Bipolar Disorder	Dickinson, Becerra, & Coombes, 2017	Executive Function	Bipolar Disorder I	30	3,792
	Lee et al., 2014	Comprehensive	First episode Bipolar Disorder	12	341
	Arts, Jabben, Krabbendam, & Van Os, 2008	Comprehensive	Euthymic Bipolar Disorder	28	679
	Bora et al., 2009a	Comprehensive	Euthymic Bipolar Disorder	45	1,446
	Bourne et al., 2013	Comprehensive	Euthymic Bipolar Disorder	31	1,276
	Cotrena et al., 2020	Comprehensive	Euthymic Bipolar Disorder	107	6,424
	Kurtz and Gerraty, 2009	Comprehensive	Euthymic Bipolar Disorder	42	1,197
	Mann-Wrobel, Carreno, & Dickinson, 2011	Comprehensive	Euthymic Bipolar Disorder	28	1,026
	Robinson et al., 2006	Comprehensive	Euthymic Bipolar Disorder	26	689
	Samamé, Martino, & Strejilevich, 2013	Comprehensive	Euthymic Bipolar Disorder	11	382
	Torres et al., 2007	Comprehensive	Euthymic Bipolar Disorder	39	948
	Kurtz and Gerraty, 2009	Comprehensive	Manic/mixed Bipolar Disorder	13	314
	Kurtz and Gerraty, 2009	Comprehensive	Depressed Bipolar Disorder	5	96
	Bora et al., 2011	Comprehensive	Bipolar Disorder II	9	263
	Cotrena et al., 2020	Comprehensive	Bipolar Disorder II	19	702
	Dickinson et al., 2017	Executive Function	Bipolar Disorder II	36	3,792
	Raucher-Chéné, Achim, Kaladjian, & Besche-Richard, 2017	Verbal Fluency	Mixed Bipolar Disorder	39	1,423
	Stefanopoulou et al., 2009	Comprehensive	Mixed Bipolar Disorder	53	2,508
Eating Disorders	Dobson & Dozois, 2004	Interference Control (Stroop)	Anorexia Nervosa	2	37
	Hirst et al., 2017	Executive Function	Anorexia Nervosa	32	1,606
	Westwood et al., 2016	Set Shifting (WCST)	Anorexia Nervosa	18	1,554
	Zakzanis et al., 2010	Comprehensive	Anorexia Nervosa	27	608
	Cury et al., 2020	Executive Function	Binge Eating Disorder	15	–
	Lavagnino, Arnone, Cao, Soares, & Selvaraj, 2016	Inhibition (SST)	Binge Eating Disorder	3	115
	Wu, Hartmann, Skunde, Herzog, & Friederich, 2013	Inhibition	Binge Eating Disorder	2	82
	Dobson & Dozois, 2004	Interference Control (Stroop)	Bulimia Nervosa	6	141
	Wu et al., 2013	Inhibition	Bulimia Nervosa	19	563
	Zakzanis et al., 2010	Comprehensive	Bulimia Nervosa	14	347
	Roberts, Tchanturia, Stahl, Southgate, & Treasure, 2007	Set Shifting	Anorexia & Bulimia Nervosa	15	360
Depression	Christensen, Griffiths, MacKinnon, & Jacomb, 1997	Comprehensive	Major Depressive Disorder	154	–
	Lim et al., 2013	Comprehensive	Major Depressive Disorder	22	955
	Rock, Roiser, Riedel, & Blackwell, 2014	Comprehensive	Major Depressive Disorder	24	784
	Stefanopoulou et al., 2009	Comprehensive	Major Depressive Disorder	53	1,189
	Wagner, Doering, Helmreich, Lieb, & Tadić, 2012	Executive Function	Major Depressive Disorder	110	3,162
	Ahern and Semkovska, 2017	Comprehensive	Major Depressive Disorder - First episode	30	994
	Lee, Hermens, Porter, & Redoblado-Hodge, 2012	Comprehensive	Major Depressive Disorder - First episode	13	644
	Bora, Harrison, Yücel, & Pantelis, 2013	Comprehensive	Euthymic Major Depressive Disorder	27	895
	Semkovska et al., 2019	Comprehensive	Euthymic Major Depressive Disorder	252	11,882
	Epp, Dobson, Dozois, & Frewen, 2012	Interference control (Stroop)	Depression	14	487
	Henry and Crawford, 2005	Verbal Fluency	Depression	42	1,077
	Snyder, 2013	Executive Function	Depression	113	3,936
OCD	Abramovitch et al., 2013	Comprehensive	Obsessive-Compulsive Disorder	115	3,452
	Fradkin, Strauss, Pereg, & Huppert, 2018	Cognitive Flexibility	Obsessive-Compulsive Disorder	75	2,677
	Henry, 2006	Fluency, Set Shifting	Obsessive-Compulsive Disorder	32	785
	Shin et al., 2014	Comprehensive	Obsessive-Compulsive Disorder	88	3,070
	Snyder, Kaiser et al., 2015	Executive Function	Obsessive-Compulsive Disorder	110	3,162
Panic Disorder	O'Sullivan and Newman, 2014	Comprehensive	Panic Disorder	14	439
Personality Disorders	Ruocco, 2005	Comprehensive	Borderline Personality Disorder	10	225
	Unoka and Richman, 2016	Comprehensive	Borderline Personality Disorder	27	835
	Ogilvie et al., 2011	Executive Function	Antisocial Personality Disorder	301	5,847
PTSD	Johnsen & Asbjørnsen, 2008	Verbal Memory	Post-Traumatic Stress Disorder	28	667
	Scott et al., 2015	Comprehensive	Post-Traumatic Stress Disorder	60	1,779
Schizophrenia	Aleman et al., 1999	Memory	Schizophrenia	70	–

(*continued on next page*)

Table 1 (*continued*)

Disorder/Category	Meta-analysis	Scope/focus	Specifier	*K*	Clinical sample n
	Bokat and Goldberg, 2003	Verbal Fluency	Schizophrenia	13	526
	Dickinson, Ramsey, & Gold, 2007	Comprehensive	Schizophrenia	37	1,961
	Doughty & Done, 2009	Semantic Memory	Schizophrenia	91	–
	Forbes, Carrick, McIntosh, & Lawrie, 2009	Working Memory	Schizophrenia	187	17,152
	Johnson-Selfridge and Zalewski, 2001	Executive Function	Schizophrenia	71	–
	Heinrichs and Zakzanis, 1998	Comprehensive	Schizophrenia	204	7,420
	Henry and Crawford, 2005	Comprehensive	Schizophrenia	84	2,947
	Knowles, David, & Reichenberg, 2010	Processing Speed	Schizophrenia	47	4,135
	Laws, 1999	WCST	Schizophrenia	29	1,169
	Lee and Park, 2005	Working Memory	Schizophrenia	124	3,531
	Schaefer et al., 2013	Comprehensive	Schizophrenia	100	9,048
	Stefanopoulou et al., 2009	Comprehensive	Schizophrenia	53	1,067
	Westerhausen, Kompus, & Hugdahl, 2011	Interference control (Stroop)	Schizophrenia	36	1,081
	Bora and Murray, 2014	Comprehensive	Schizophrenia - First Episode	25	905
	Mesholam-Gately et al., 2009	Comprehensive	Schizophrenia - First Episode	47	2,204
	Rajji, Ismail, & Mulsant, 2009	Comprehensive	Schizophrenia - First Episode	110	5,010
	Fatouros-Bergman, Cervenka, Flyckt, Edman, & Farde, 2014	Comprehensive	Schizophrenia - Drug Naive	23	1,106
SUD	Hall et al., 2018	Comprehensive	Cocaine abuse/dependence	54	1,718
	Jovanovski, Erb, & Zakzanis, 2005	Comprehensive	Cocaine abuse/dependence	15	481
	Smith et al., 2014	Inhibition	Cocaine abuse/dependence	19	416
	Hall et al., 2018	Comprehensive	Methamphetamine abuse/dependence	41	1,297
	Potvin et al., 2018	Comprehensive	Methamphetamine use disorder	44	1,592
	Scott et al., 2007	Comprehensive	Methamphetamine abuse/dependence	17	471
	Smith et al., 2014	Inhibition	Methamphetamine abuse/dependence	4	71
	Crowe, Cammisuli, & Stranks, 2020	Comprehensive	Alcohol Dependence	17	734
	Smith et al., 2014	Inhibition	Alcohol abuse/dependence	18	852
	Stephan et al., 2017	Executive Function	Alcohol Dependence	77	2,620
	Stavro, Pelletier, & Potvin, 2013	Comprehensive	Alcoholism-short term abstinence	62	-
	Smith et al., 2014	Inhibition	Opioid abuse/dependence	5	161
	Wollman et al., 2019	Comprehensive	Opioid abuse/dependence	61	2,580
	Chowdhury, Livesey, Blaszczynski, & Harris, 2017	Executive Function	Gambling Disorder	12	507
	Ioannidis, Hook, Wickham, Grant, & Chamberlain, 2019	Executive Function	Gambling Disorder	50	-
	Smith et al., 2014	Inhibition	Gambling Disorder	8	262
	Lovell, Akhurst, Padgett, Garry, & Matthews, 2020	Comprehensive	Marijuana abuse/dependence	30	849
Tourette Syndrome	Morand-Beaulieu et al., 2017	Inhibition	Tourette Syndrome	61	1,717

Note. ADHD = Attention Deficit/Hyperactivity Disorder; PTSD = Post-Traumatic Stress Disorder; SUD = Substance Use Disorders; OCD = Obsessive-Compulsive Disorder; ASD = Autism Spectrum Disorder.

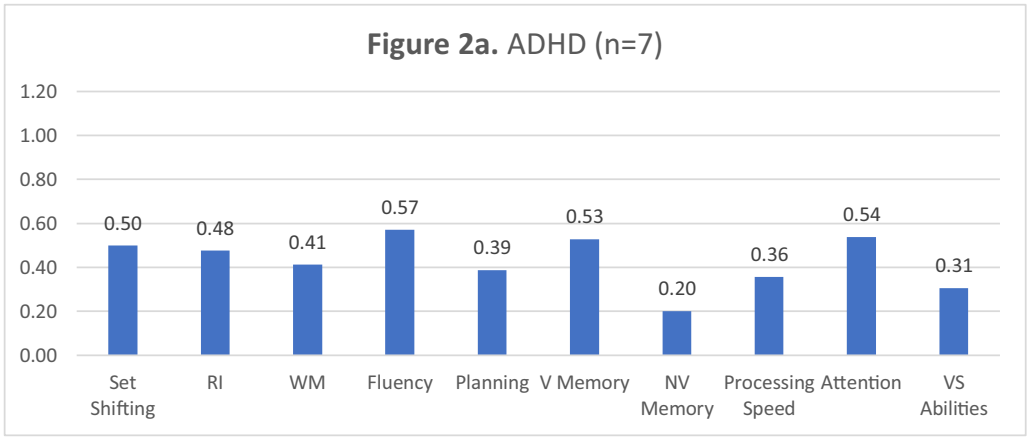
[a] Total K and total N available for youth and adults combined.

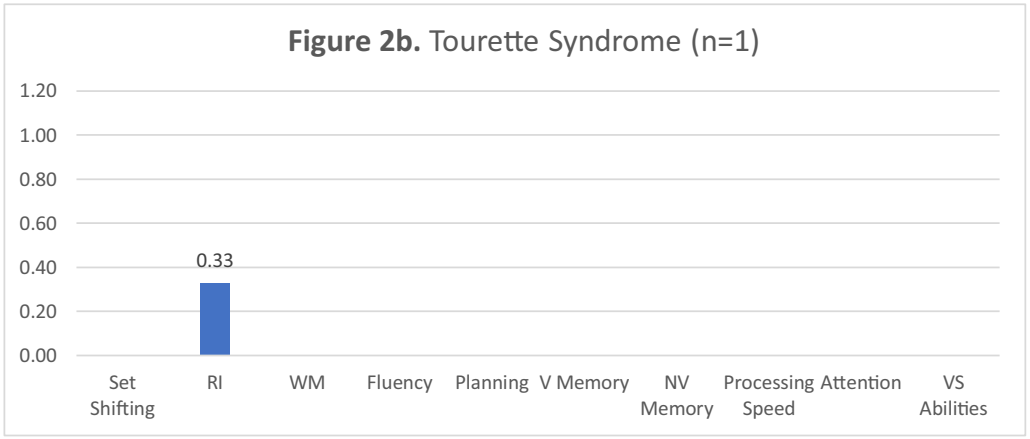
Table 2
Unweighted mean effect sizes for 10 cognitive functions across disorder categories.

	Executive Functions					Attention	Memory		Processing speed	Visuospatial Abilities	Unweighted Mean ES
	Set Shifting	Response Inhibition	Working Memory	Fluency	Planning		Verbal Memory	Non-Verbal Memory			
Eating Disorders	0.27	0.29	0.26	0.13	0.43	0.25	0.42	0.42	0.27	0.40	0.31
Tourette Syndrome	–	0.33	–	–	–	–	–	–	–	–	0.33
Panic Disorder	0.31	–	0.29	0.43	0.29	–	0.55	0.31	0.08	0.38	0.33
Gambling	–	0.57	–	–	–	0.12	–	–	0.33	–	0.34
Substance Abuse	0.38	0.41	0.49	0.29	0.72	0.29	0.48	0.47	0.29	0.48	0.43
ADHD	0.50	0.48	0.41	0.57	0.39	0.54	0.53	0.20	0.36	0.31	0.43
ASD	0.65	0.49	0.40	0.44	0.27	–	–	–	–	–	0.45
OCD	0.42	0.49	0.33	0.38	0.59	0.48	0.38	0.75	0.48	0.40	0.47
PTSD	–	–	0.50	0.43	–	–	0.65	0.31	0.59	0.38	0.48
Depression	0.62	0.83	0.38	0.54	0.45	0.55	0.44	0.53	0.50	0.57	0.54
Personality Disorders	0.28	0.51	0.33	0.39	0.84	0.40	0.45	1.59	0.47	0.41	0.57
Bipolar Disorders	0.64	0.66	0.56	0.62	0.60	0.61	0.71	0.57	0.68	0.33	0.60
Schizophrenia	0.80	0.65	0.82	0.93	0.86	0.78	1.02	0.85	0.96	0.76	0.84
Unweighted Mean ES	0.49	0.52	0.43	0.47	0.54	0.45	0.56	0.60	0.46	0.44	0.47

ADHD = Attention Deficit/Hyperactivity Disorder; ASD = Autism Spectrum Disorder; OCD = Obsessive-Compulsive Disorder; PTSD = Post-Traumatic Stress Disorder.

Mean Neuropsychological Subdomain Effect Sizes Across Disorders





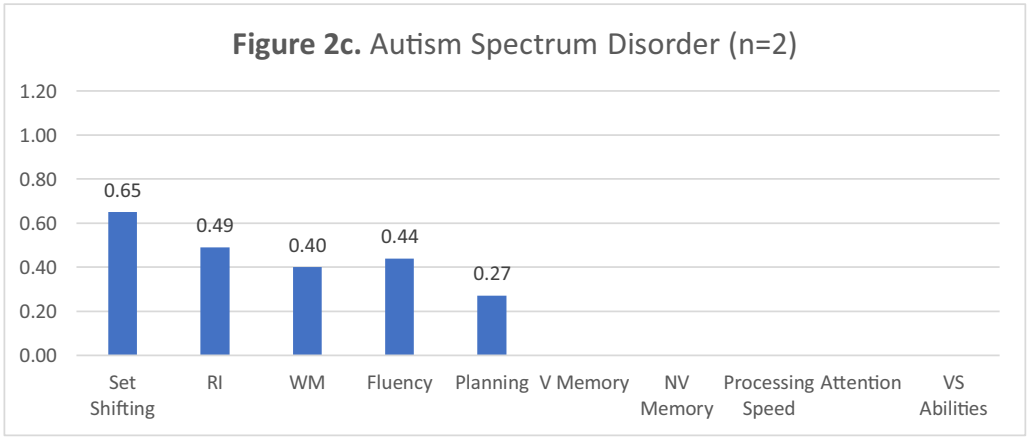


Fig. 2. Mean neuropsychological subdomain effect sizes across disorders

ASD, OCD, PD, ED, depression, ASPD, and SUD (*d* range .24-.49). Medium effect sizes for working memory were found for PTSD, and BD (*d*=.50 and .56, respectively). A large effect size was found for SCZ (*d*=.82). Information regarding working memory was not available for TS, BPD, and GD.

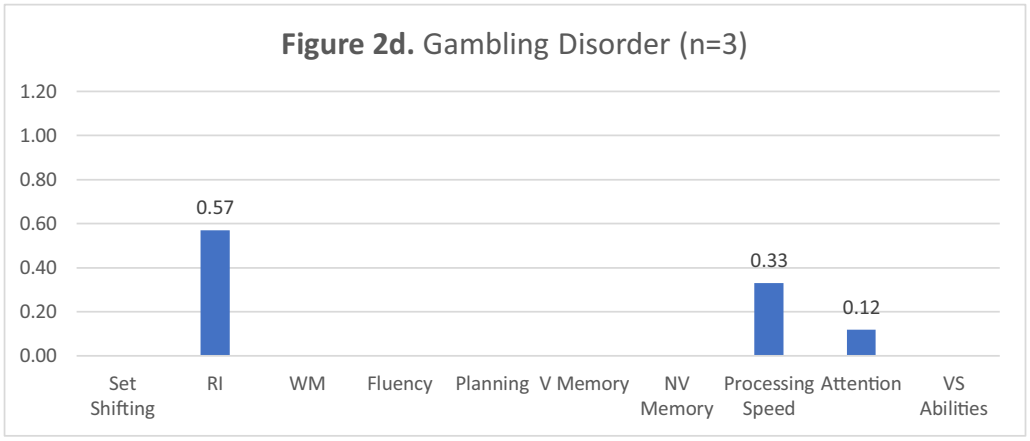
3.1.1.4. Fluency. Small effect sizes were found for ASD, OCD, PD, PTSD, ED, ASPD, and SUD (*d* range .13-.44). Medium effect sizes for fluency were found for ADHD, BD, and depression (*d* range .56-.62). A large effect size was found for SCZ (*d*=.93). Information regarding fluency was not available for TS, BPD, and GD.

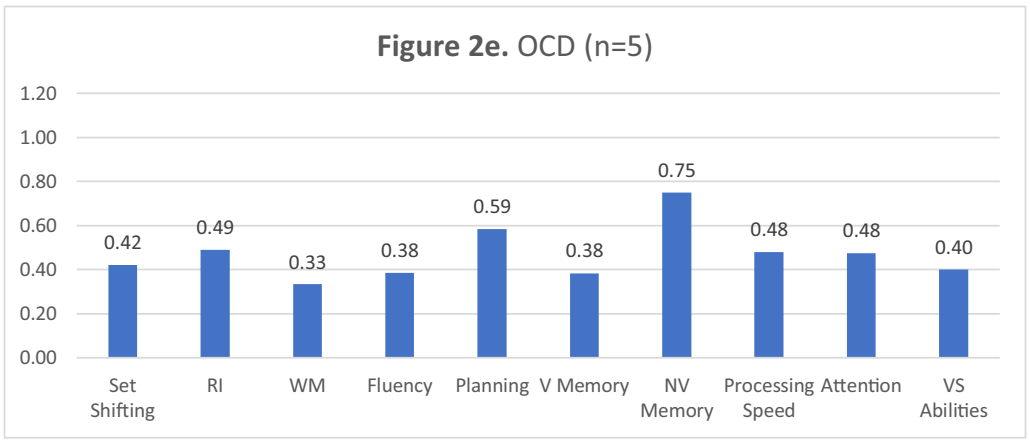
3.1.1.5. Planning. Small effect sizes were found for ADHD, ASD, PD, depression, ASPD, and ED, (*d* range .24-.48). Medium effect sizes for fluency were found for OCD, BD, and SUD (*d* range .59-.72). A large effect size was found for BPD and SCZ (*d*=1.43, and .86, respectively). Information regarding planning was not available for TS, GD, and PTSD.

3.1.2. Memory

Small effect sizes were found for ADHD, PD, Depression, ED, and

Mean Neuropsychological Subdomain Effect Sizes Across Disorders – Continued





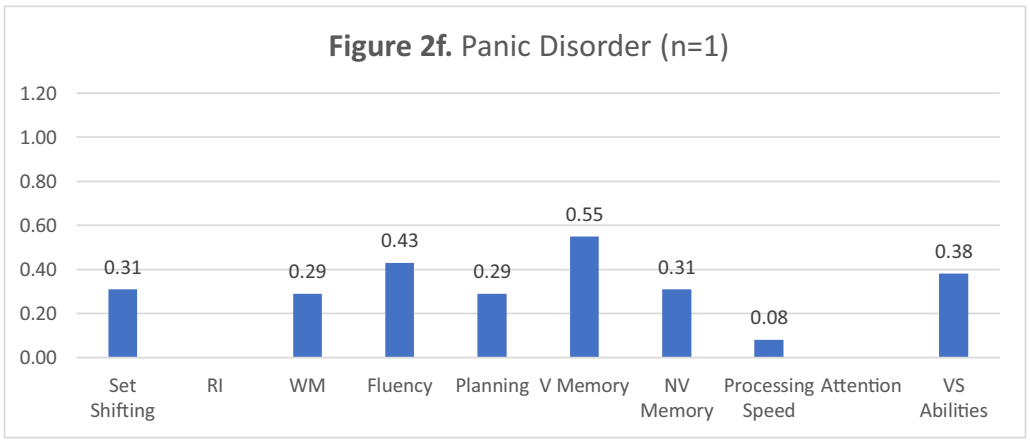


Fig. 2. (*continued*).

SUD (*d* range .41-.48). Medium effect sizes for were found for OCD, PTSD, BD, and BPD (*d* range .59-.74). A large effect size was found for SCZ (*d*=.98). Information regarding memory was not available for TS, ASD, ASPD, and GD.

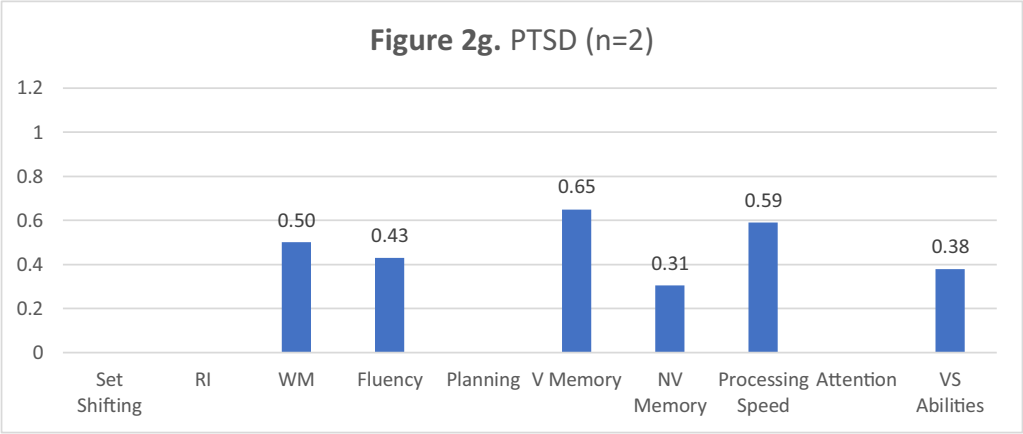
3.1.2.1. Verbal memory. Small effect sizes were found for OCD, Depression, ED, BPD, and SUD (*d* range .38-.48). Medium effect sizes were found for ADHD, PD, PTSD and BD (*d* range .53-.74). A large effect size was found for SCZ (*d*= 1.02). Information regarding verbal memory was not available for TS, ASD, ASPD, and GD.

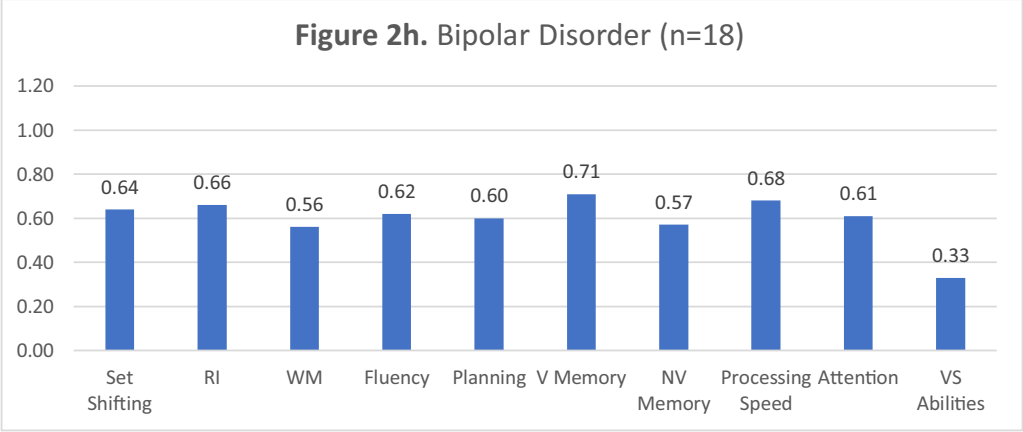
3.1.2.2. Non-verbal memory. Small effect sizes were found for ADHD, PD, PTSD, ED, and SUD (*d* range .20-.42). Medium effect sizes were found for OCD, BD, and Depression. (*d* range .51-.75). A large effect size was found for SCZ and BPD (*d*=.85, and 1.59, respectively). Information regarding verbal memory was not available for TS, ASD, ASPD, and GD.

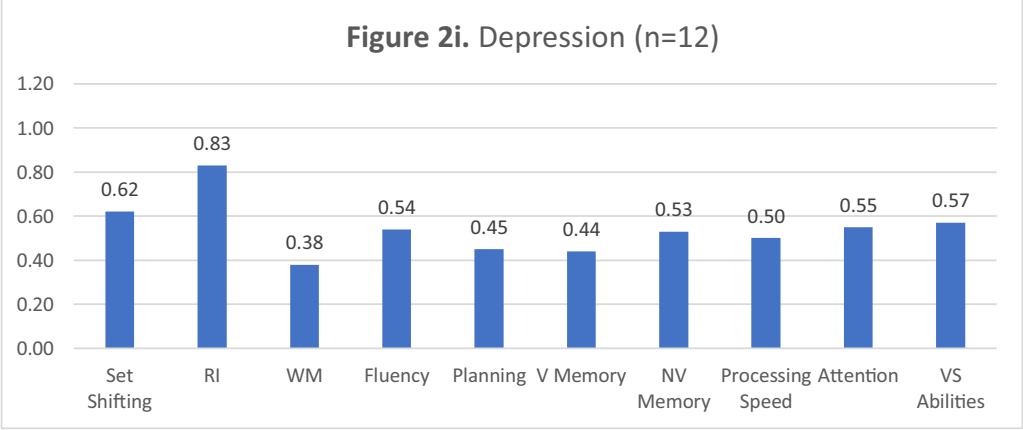
3.1.3. Processing speed

Small effect sizes were found for ADHD, GD, PD, ED, OCD, BPD, and SUD (*d* range .08-.48). Medium effect sizes for processing speed indices, were found for PTSD, BD, ASPD, and Depression (*d* range .50-.68). A large effect size was found for SCZ (*d*=.96). Information regarding processing speed was not available for TS, and ASD.

Mean Neuropsychological Subdomain Effect Sizes Across Disorders – Continued







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3.1.4. Attention

Small effect sizes were found for, GD, OCD, ED, ASPD, BPD, and SUD (*d* range .12-.49). Medium effect sizes for attention were found for ADHD, BD, Depression and SCZ (*d* range .55-.78). Information regarding attention was not available for TS, ASD, PD, and PTSD.

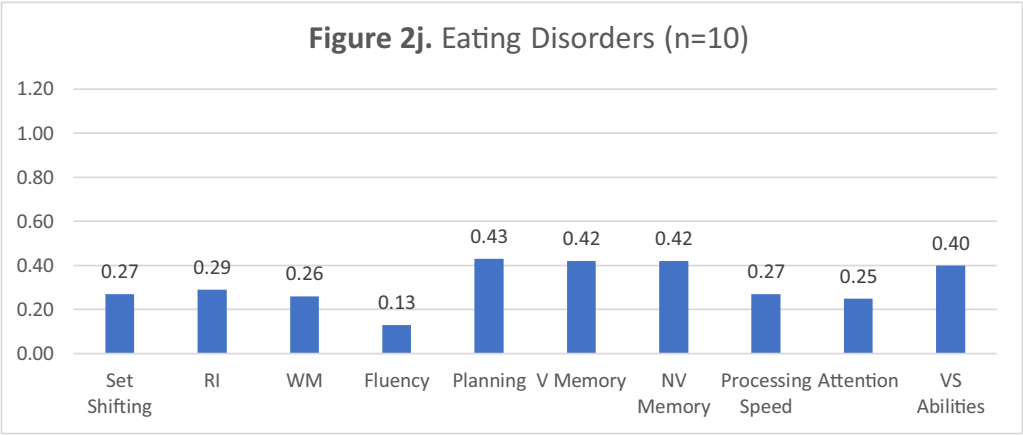
3.1.5. Visuospatial function

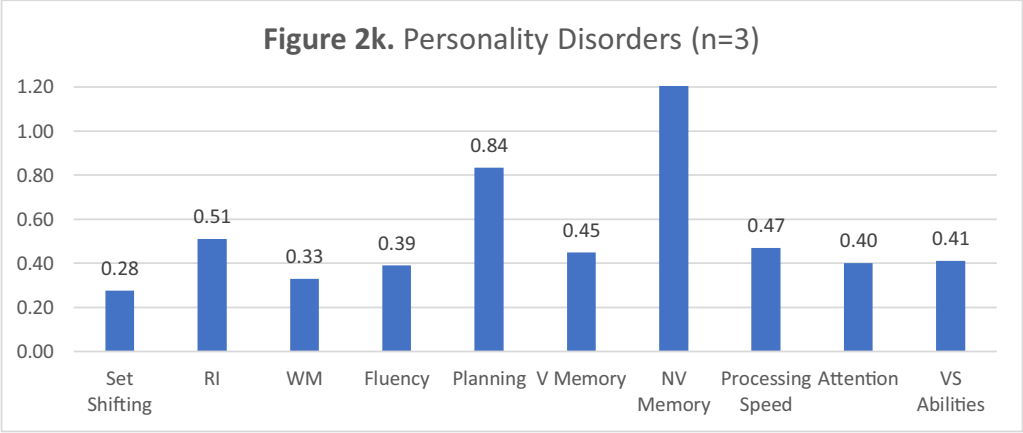
Small effect sizes were found for ADHD, OCD, PD, PTSD, BD, ED, BPD, and SUD (range .31-.48). Medium effect sizes for visuospatial functions, were found in Depression, and SCZ (.57, and .76, respectively). Information regarding visuospatial function was not available for TS, ASD, ASPD, and GD.

3.2. Notable differences between primary disorders

Examination of effect sizes for the five major neuropsychological domains between disorders categories revealed that apart from SCZ, no other disorder was associated with a large effect size on any of the five domains. In fact, SCZ was found to be associated with large effect sizes for EF, Memory, and Processing speed (d = .85, .98, and .96, respectively), and two effects approaching large magnitude for Attention and VS abilities (d = .78, and .77 respectively). In terms of neuropsychological subdomains of EF, large effect sizes were found for SCZ across subdomains, apart from RI. In fact, RI in SCZ was found to have a medium effect size (d = .65), but BD-I and depression were found to have

Mean Neuropsychological Subdomain Effect Sizes Across Disorders – Continued





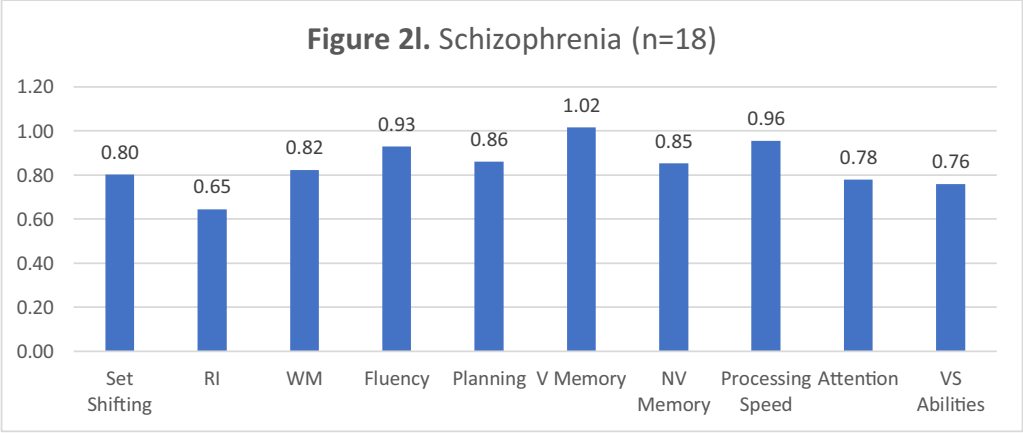


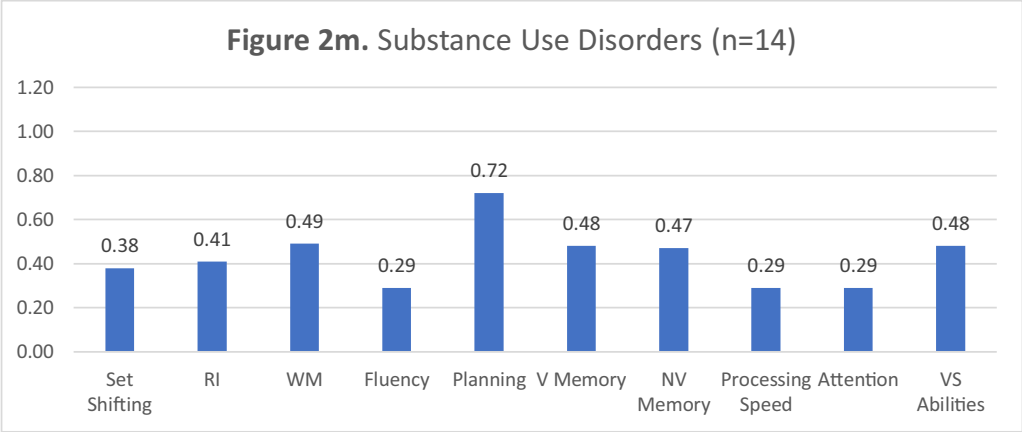
Fig. 2. (*continued*).

large effect sizes for RI (.98, .90, respectively). Interestingly, effect sizes for RI in ADHD were found to be small, approaching medium magnitude ($d = .48$). A very large effect size was also found for planning in BPD ($d = 1.43$). Verbal memory was associated with very large effect sizes for Bipolar-I ($d = 1.05$) and depressed BD (d=1.23). BPD was associated with a very large effect size for non-verbal memory (d=1.59), which was the highest effect size found in this study. BD-I was also associated with a large effect size for processing speed (d=.84). Across disorders, most diagnostic categories were generally associated with small effect sizes, with the exceptions of SCZ, BPD and BD. Indeed, across meta-analyses comparing test performance of ADHD, PTSD, and PD to non-clinical groups, 60%, 50%, and 88% of the effects were small, respectively. For all other disorders reviewed (apart from SCZ, BPD & BD mentioned above), our findings indicate a high proportion (>70%) of small effect sizes.

4. Discussion

The present review is the first to systematically and critically examine the transdiagnostic association between psychopathology and cognitive functions, utilizing all available meta-analyses to provide a comprehensive review of the field. The results of this systematic review indicate conclusively that, compared with non-psychiatric controls, any form of psychopathology examined is associated with poorer performance across cognitive domains, subdomains, and tests. Transdiagnostically, small to medium effect sizes were found for

Mean Neuropsychological Subdomain Effect Sizes Across Disorders – Continued



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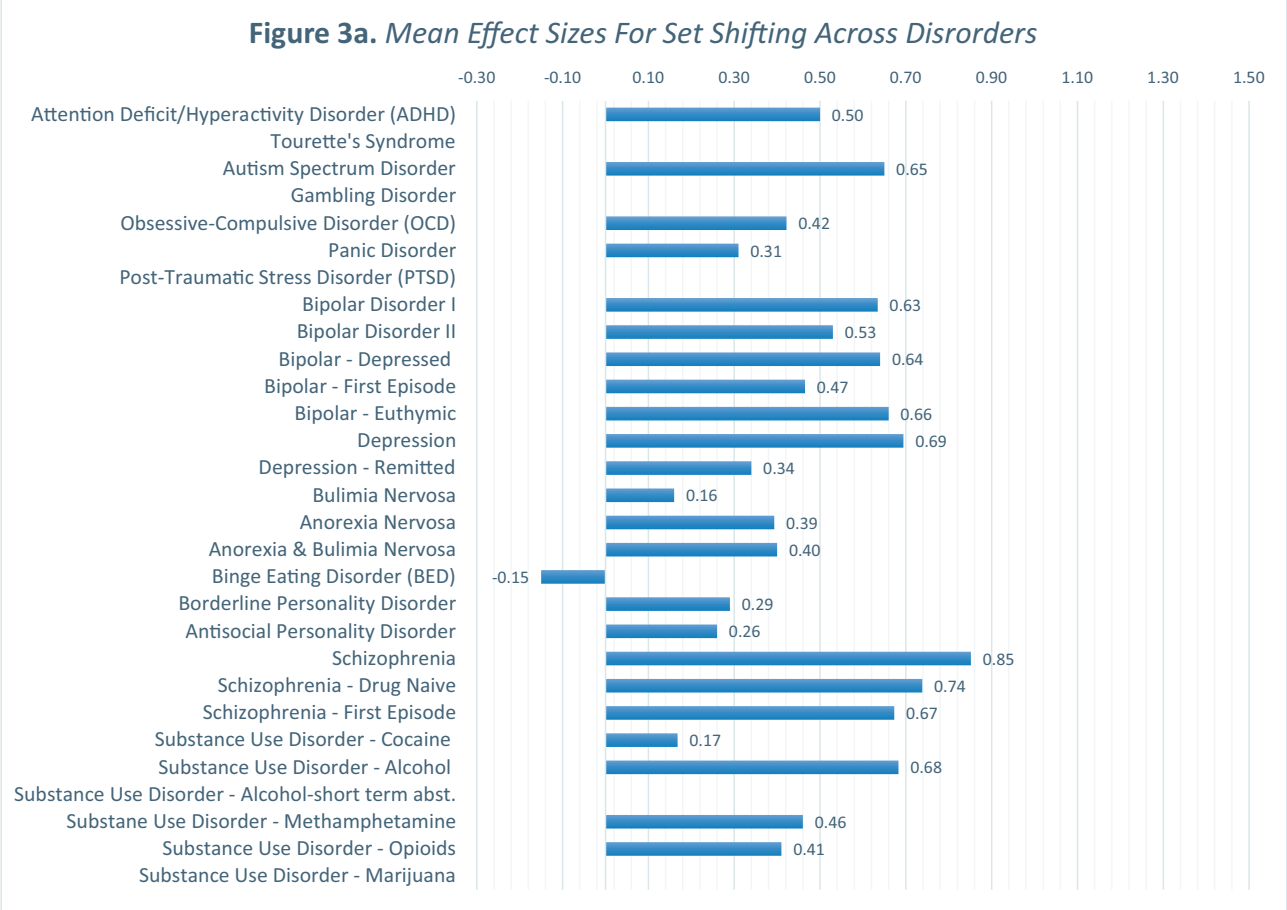

Fig. 3a. Mean effect sizes for set shifting across disrorders.

neuropsychological subdomains, ranging from ~.4 to ~.6 (except for SCZ). When considering major cognitive domains only, effect sizes revealed even smaller differences across disorders. Hence, we use the C factor to denote the ubiquity of cognitive dysfunction associated with practically every psychological disorder.

Although our findings indicate that the C factor is transdiagnostic in psychopathology, some expected notable differences were noticeable between disorders. On average, disorders characterized by psychotic symptoms (i.e., SCZ and BD- I), were found to be associated with increased effect sizes. This is not surprising since, as suggested by Caspi and Moffitt (2018), the severity of the p factor is correlated with the severity of the C factor. Conversely, EDs, and most SUDs, were found to be associated with smaller effect sizes. Notable exceptions for this general trend were found across domain and subdomains. For example, SCZ tended to be associated with the highest effect sizes across domains, but in some cases equivalent or larger effect sizes were found for other

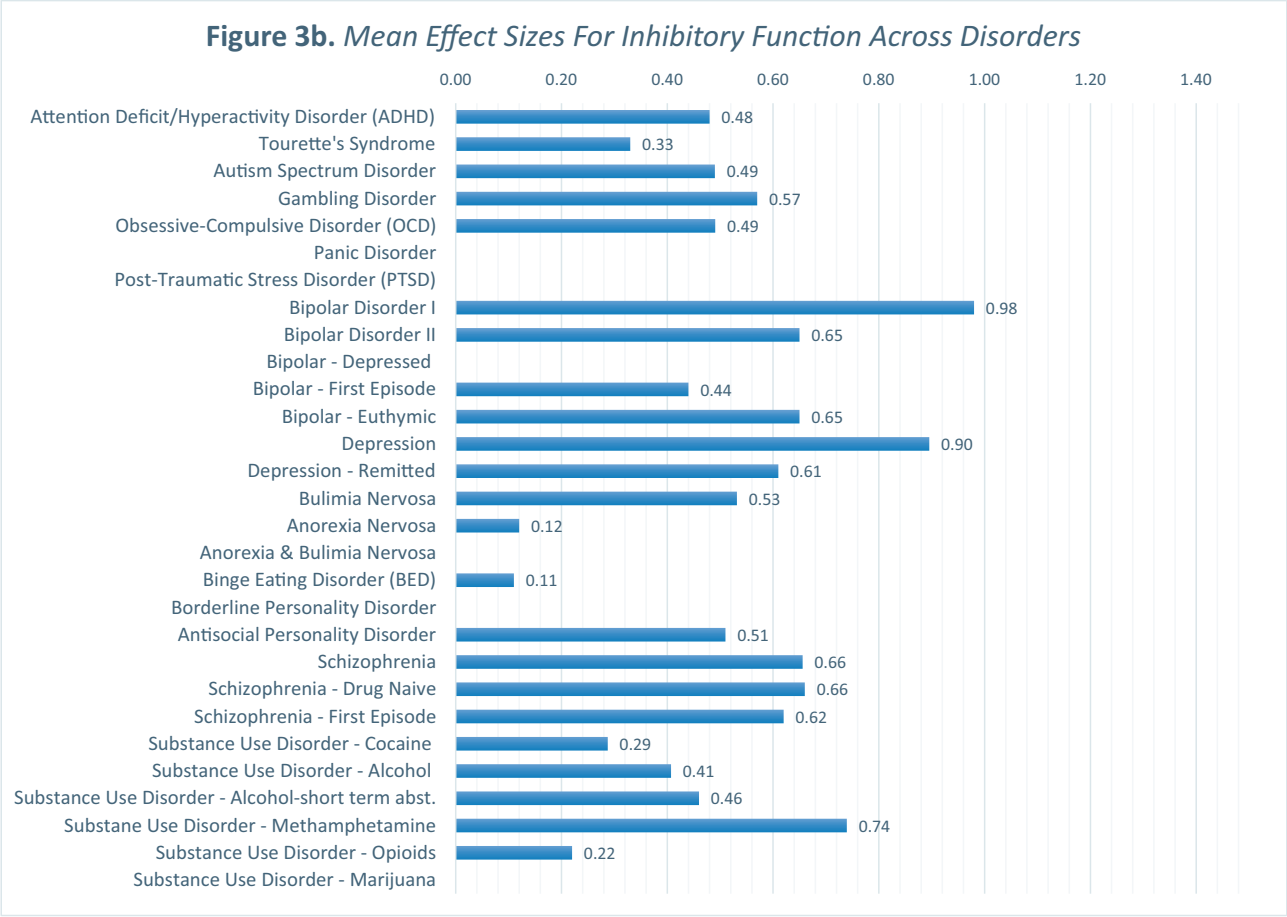

Fig. 3b. Mean effect sizes for inhibitory function across disorders.

disorders (e.g., larger effect size for response inhibition in MDD). Of note, the three disorders associated with largest effect sizes, namely SCZ, BD, and BPD, are the disorders found most commonly in inpatients settings (NASMHPD, 2017) and are associated with the most prolonged pharmacotherapy, and with significantly elevated rates of polypharmacy (De las Cuevas & Sanz, 2004; Mojtabai & Olfson, 2010). Along this line, research findings suggest that in the context of psychotic disorders, better cognitive functioning was associated with reduce likelihood of polypharmacy (Chakos et al., 2006).

Given that not all major DSM disorders were covered by the meta-analyses included in this systematic review, we supplemented our review by examining the literature on neuropsychological functions in large samples of participants with specific disorders, for which meta-analyses are not available, as well as on dimensional psychopathology (i.e., elevated traits) and their association with cognitive function. This search yielded a body of literature that is in line with the results of the present investigation, providing supporting and complementary evidence to our findings. Underperformance on several neuropsychological domains was reported in depersonalization disorder (Guralnik, Giesbrecht, Knutelska, Sirroff, & Simeon, 2007), hoarding disorder (Grisham & Baldwin, 2015; Tolin, Villavicencio, Umbach, & Kurtz, 2011), hair pulling disorder (Trichotillomania) and body dysmorphic disorder (Jefferies-Sewell, Chamberlain, Fineberg, & Laws, 2017; Toh, Castle, & Rossell, 2015), and OCPD (Fineberg et al., 2015). Similarly, studies examining dimensional psychopathological traits and their association with cognitive functions consistently report underperformance on cognitive tests in the context of schizotypy (Siddi et al., 2017), dissociation (Schurle Bruce, Ray, Bruce, Arnett, & Carlson, 2007), psychopathy/antisocial behavior (Morgan & Lilienfeld, 2000), rumination (Yang et al., 2017), both extraversion and introversion (Campbell, Davalos, McCabe, & Troup, 2011) and neuroticism (Murdock, Oddi, & Bridgett, 2013).

These results cement our findings that deficient performance on cognitive tests (i.e., the C factor) is a transdiagnostic phenomenon, as well as one that transcends DSM taxonomy. Furthermore, these findings are in support of the hypothesis that c characterizes and positively associated with p' (Caspi & Moffitt, 2018), and is transcending specific symptomatic phenotypes and phenomenology. Consequently, the ubiquity of the C factor in psychopathology raises an all-important question: *What is the underlying reason that disorders that differ substantially in terms of their clinical presentation, phenomenology, treatment, and purported neurobiological alterations, all share deficient cognitive performance?* Here we review and discuss several plausible explanations, which may be utilized as a roadmap for further research into the association between cognitive function and psychopathology. However, to provide an important interpretive context to our results, we first address specific methodological and conceptual problems inherent to neuropsychological testing in psychopathology.

4.1. Psychometric and interpretive issues in neuropsychology

Although deficient neuropsychological test performance was found across disorders, various construct-specific effect sizes vary within and between disorders. Given that different neuropsychological constructs are thought to reflect real life cognitive and behavioral functions, it is tempting to infer from these results about phenomenological, functional, and clinical variations between syndromes that may account for such differences. However, extreme caution should be taken when interpreting these results along these lines.

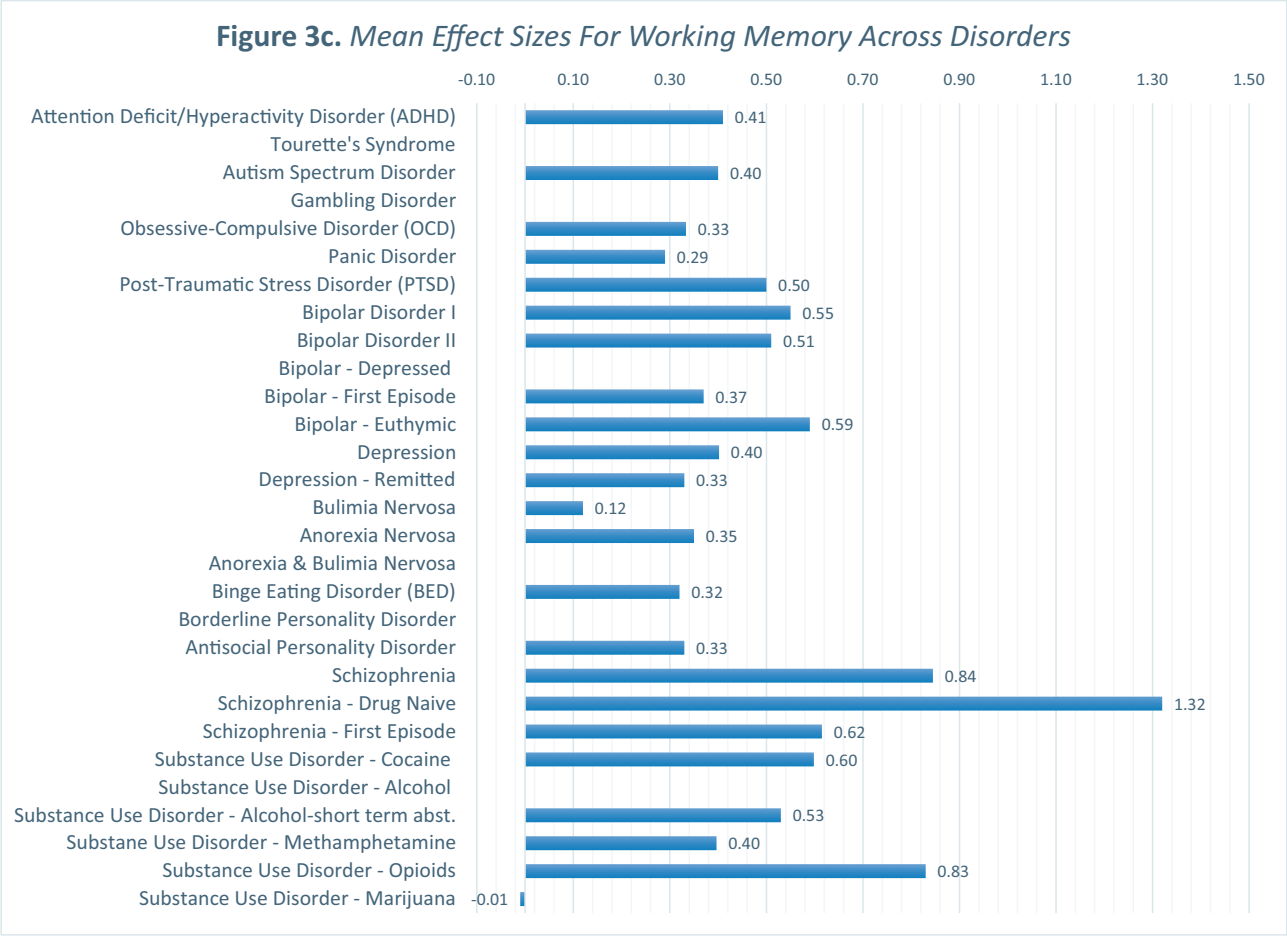

Fig. 3c. Mean effect sizes for working memory across disorders

4.1.1. Ecological validity

In its current form neuropsychological assessment is used to infer a person's ability to perform tasks in the real world (Spooner & Pachana, 2006). Thus, the past three decades saw an ever-increasing interest in ecological validity, the "*functional and predictive relationship between the patient's performance on a set of neuropsychological tests and the patient's behavior in a variety of real-world settings*" (Sbordone, 1996, p. 16). The two main approaches to assess ecological validity in neuropsychological testing are veridicality, and verisimilitude. Veridicality, which is the dominant method, is the degree to which existing neuropsychological tests relate empirically to a measure of everyday function. Verisimilitude entails the degree in which a test's demands resemble the demands of the everyday environment (Chaytor & Schmitter-Edgecombe, 2003). The latter requires development of novel tests, in which tasks mimic or simulate everyday functions. Tests such as the Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson, Alderman, Burgess, Emslie, & Evans, 1996) that simulate real life situation (e.g., planning a visit to the zoo, searching for ones keys) exemplify the verisimilitude approach.

The vast majority of research into ecological validity of neuropsychological testing centers on veridicality and is conducted on neurological patients. This body of literature yields moderate levels of ecological validity in terms of everyday functions/cognitive functions. However, better ecological validity is accomplished using the verisimilitude approach (Chaytor & Schmitter-Edgecombe, 2003). Threats to ecological validity include: 1) test environment, which is controlled and quiet in order to accomplish best performance, as opposed to the complexity of real life situations; 2) sampling circumscribed aspects of behavior in a specific time; 3) compensatory strategies: people utilize different cognitive strategies (e.g., mnemonics) that they may not use in real life settings; 4) disagreement regarding a tests' construct validity where neuropsychologists may utilize a test that is thought to be related to construct X, whereas others would assume that the same test relates to construct Y, which will correspond with a different target real world behavior (for a review see Chaytor & Schmitter-Edgecombe, 2003).

Very little systematic research on ecological validity of neuropsychological tests in the context of psychopathology has been conducted, with the exception of SCZ. One of the main reasons is that most measures of everyday function assess 'activities of daily living' which assess very basic tasks. Indeed, researchers have been calling for ecological validity research focusing on neuropsychological assessment of 'neurologically intact populations' (Spooner & Pachana, 2006). However, such examination was not found to be rewarding. In fact, where researchers attempt to examine the association between neuropsychological tests that are thought to predict a specific construct such as impulsivity, results do not point to such an association in both clinical and non-clinical populations (Cyders & Coskunpinar, 2011; L. Sharma, Markon, & Clark, 2014). Indeed, reviewing the real life value of attention tasks, Kingstone, Smilek, Ristic, Kelland Friesen, and Eastwood (2003) concluded that "*... the evidence suggests that laboratory studies that have lost touch with real-life context may generate fundamental misunderstandings of the principles of human attention and behavior.*"(Kingstone et al., 2003, p. 179).

In the context of psychopathology it is important to note that neuropsychological tests were designed under the assumption that the assessment situation would facilitate examinees' best performance (Lezak, Howieson, Bigler, & Tranel, 2012). However, common symptoms of stress, anxiety, and depression—let alone full-blown DSM disorders—negatively impact performance on neuropsychological testing.

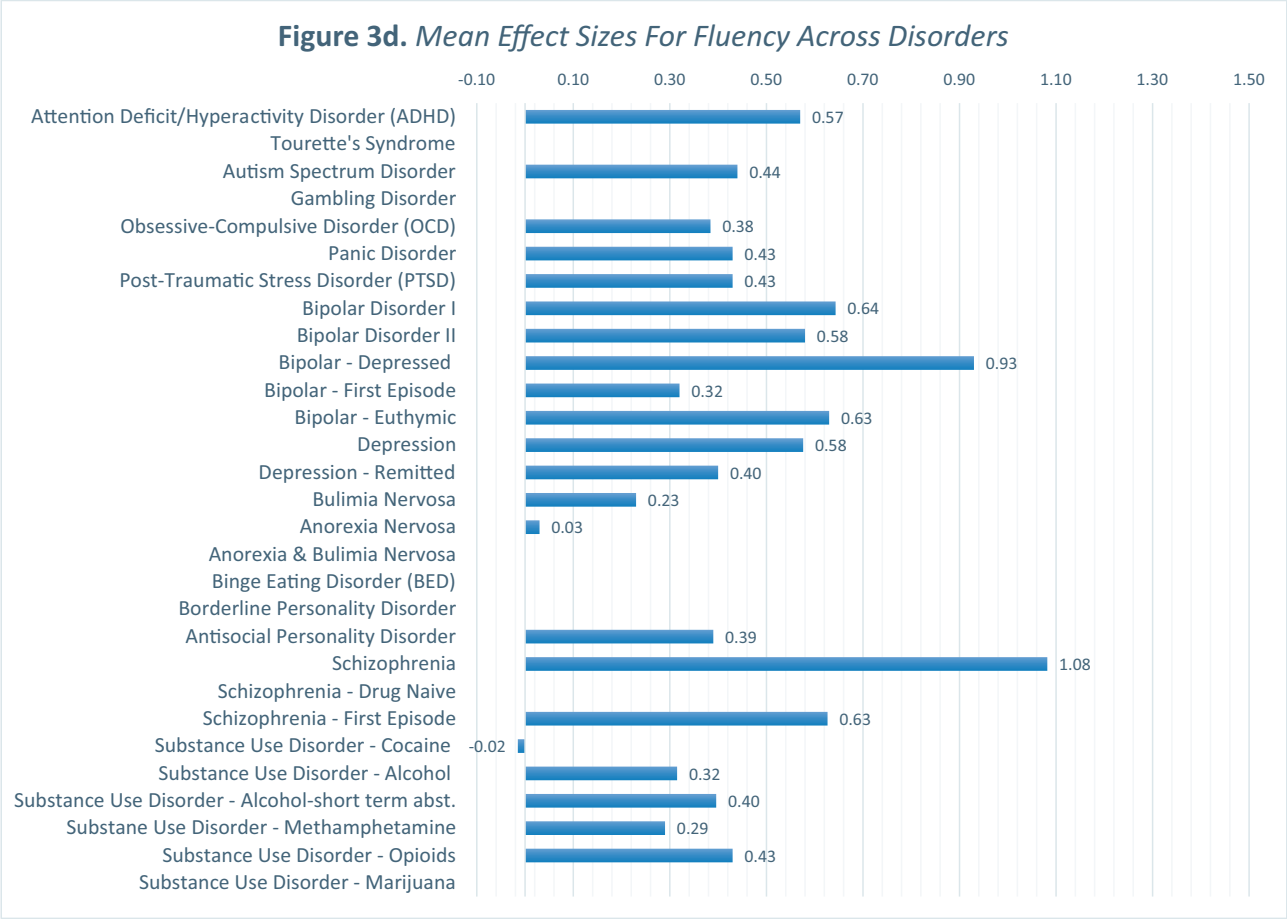

Fig. 3d. Mean effect sizes for fluency across disorders

In addition, such symptoms routinely fluctuate among individuals with psychopathology across hours or days, which poses another level of difficulty in predicting real life functions (Chaytor & Schmitter-Edgecombe, 2003). Therefore, the limited ecological validity of cognitive testing, and more so in the context of psychopathology, presents a serious challenge in distinguishing the extent to which test performance reflects brain dysfunction from the various factors which are the consequences of psychiatric disorders.

4.1.2. Cognitive impairment: Definition, specificity, and causality

A key issue in the context of cognitive testing in psychopathology relates to some interpretation biases that are all too frequent in psychiatry research (Abramovitch & Schweiger, 2015). An important and widely prevalent interpretation bias relates to the notion of cognitive impairment, or cognitive deficit. The central question pertaining to this concept is what would constitute an impairment on a cognitive function? Backed by empirical investigation, the convention in classic neuropsychology is that in the context of Mild Cognitive Impairment (MCI) or dementia, cognitive impairment can be established when a score falls between 2-3 standard deviation (SD) below the norm (equivalent to Cohen's d of 2.0-3.0; Lezak et al., 2012; Zakzanis, 2001; Zakzanis et al., 1999). A more liberal approach considers 1.0 SD below the norm as an impairment on the Wechsler intelligence and memory tests (Taylor & Heaton, 2001).

However, in the majority of neuropsychological studies in psychopathology, a statistically significant difference on a test's raw score between clinical and non-clinical control samples is interpreted as an impairment or a deficit. Yet without examining standard scores using the test's normative data, it is impossible to rule out the possibility that the two samples, while differing in performance, exhibit performance in the normal range. In addition, in cases where between-group effect sizes are presented, authors tend to interpret the magnitude of the effect size as an indicator of the magnitude of impairment (e.g., medium effect of 0.6 is interpreted as moderate impairment). Moreover, even in studies that utilize standard scores for comparison, the working definitions of what constitutes an impairment on a task vary significantly. This is due to the absence of a large-scale empirical work exploring cutoffs of cognitive impairment in psychopathology with the exception of schizophrenia, although it has been recently suggested that cognitive impairments in schizophrenia are overestimated (Steffen Moritz, Silverstein, Dietrichkeit, & Gallinat, 2020). In addition, some studies attempting to identify a 'cognitively impaired' subsample, utilized inclusion definitions that were never previously tested (e.g., a person who performed 1.5 SD below the norm or controls, on at least two tests out of the entire battery), is defined as 'cognitively impaired'. These definitions are thus arbitrary, particularly when considering that research indicates that even among non-clinical samples 50% exhibit impaired performance on one or two tests (e.g., Axelrod & Wall, 2007).

A second longstanding problem related to interpretations of neuropsychological test performance in research setting is the tendency to interpret a difference between clinical and control samples on tests that measure a specific function as indicative of a specific neuropsychological deficit (Loren J Chapman & Chapman, 1973; Loren J. Chapman & Chapman, 1978; Keef, 1995; MacDonald 3rd & Carter, 2002). Neuropsychological tests, such as ones that assess memory, visuospatial function, motor skills, and particularly higher order executive function, overlap substantially, are intercorrelated, and include redundant outcome indices (Keef, 1995). This problem is particularly evident in studies that utilize only a handful, or even a single neuropsychological test. For example, in a study comparing clinical and control samples on a

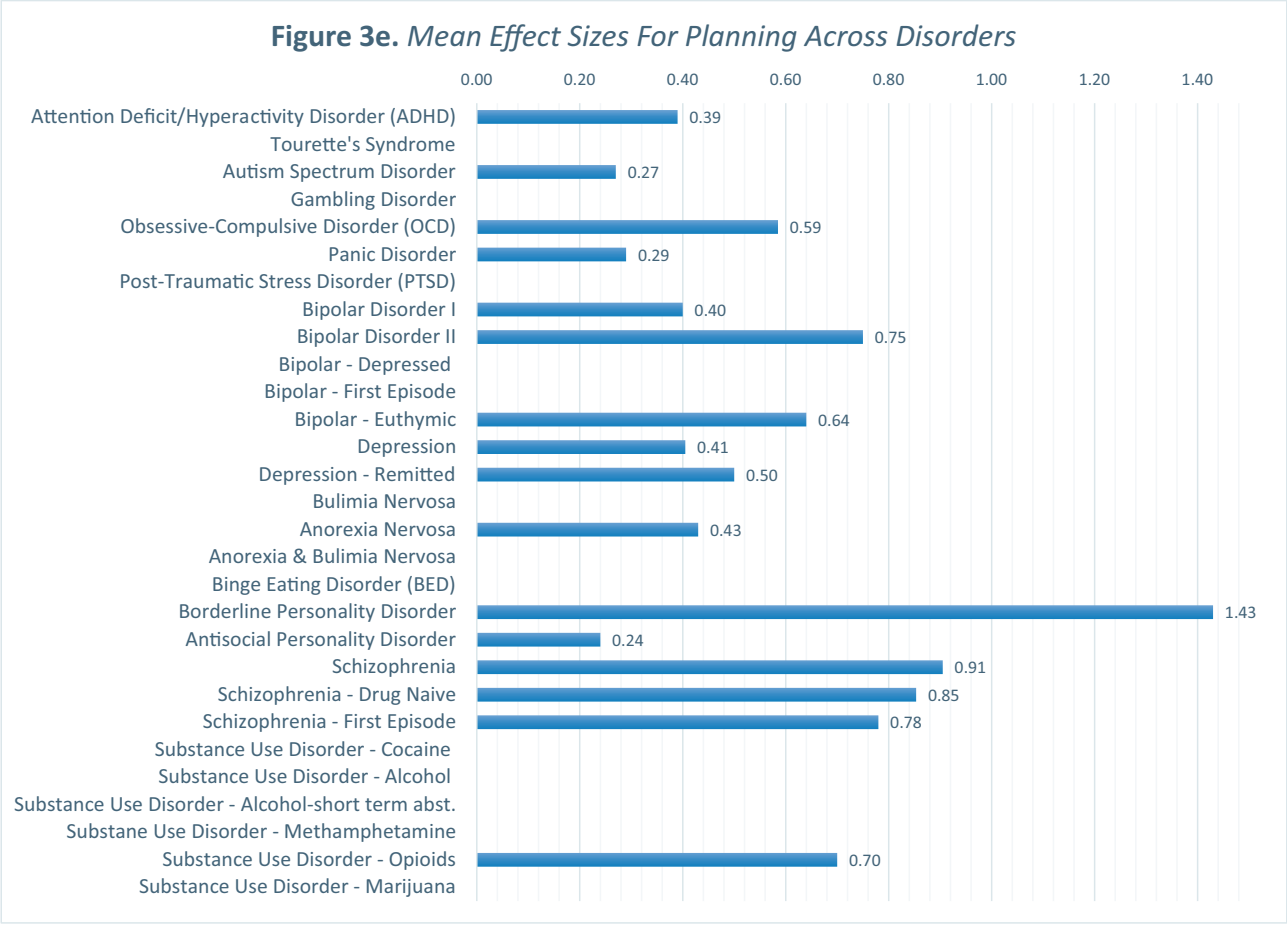

Fig. 3e. Mean effect sizes for planning across disorders

single response inhibition test, researchers may erroneously interpret a significant performance difference as a dysfunction in inhibitory control, without ascertaining whether or not there is an underlying issue with working memory and general attention, which subserves inhibitory control.

This difficulty becomes more pronounced in complex executive function tests which suffer from significant issues with task impurity and collinearity (Miyake & Friedman, 2012). This issue warrants a scrupulous design and interpretation of regression and multivariate models in neuropsychological research (Morrison, 2003). Moreover, in the context of psychopathology, in addition to the conflation of different cognitive domains, specific symptoms may further obfuscate inferences, as symptoms may be associated with global or specific effects. For example, in the case of MDD, where broad general global performance deficit is frequently found, *"This general deficit may be attributable to any number of problems, including, among others, lack of motivation, lethargy, distraction secondary to intrusive thoughts, and general motor retardation."* (Keef, 1995, p. 8).

In the context of neuropsychological test performance in research settings, specificity pertains both to the notion of a specific cognitive deficit (e.g., verbal memory deficit), but also to the notion of a deficit being 'disorder-specific' (e.g., verbal memory deficit as a specific marker of ADHD). This is a common interpretation error, and many researchers tend to conflate these two biases and interpret their results regarding deficits on *specific* cognitive function as *specific* to the disorder examined. Indeed, the notion of disorder specific cognitive etiological markers has been prevalent in the literature on cognitive functions in psychiatric disorders (Abramovitch & Schweiger, 2015). However, the results of the present review greatly weaken such argument.

Finally, many researchers examining cognitive function in psychopathology hold an all too prevalent, a priori misconception regarding causality of neuropsychological functions: when a neuropsychological underperformance is found in a disorder, the former is assumed to play a causal role in the latter. This directionality is heavily influenced by the biomedical/disease model of psychopathology, where the underlying assumption is that faulty brain structures and functions result in hard wired cognitive deficits (Abramovitch & Schweiger, 2015; Deacon, 2013). Moreover, some have suggested that such cognitive 'impairments' are causal factors leading to specific disorders. For example, in the context of bipolar disorder, some have suggested an etiological mechanism where neural abnormalities produce faulty executive functions associated with emotional regulation (Green, Cahill, & Malhi, 2007). In the context of SCZ, it has been suggested that deficient cognitive flexibility is a major heritable causal etiological factor (Owens et al., 2011; Rethelyi et al., 2012). Another example for such a causal misinterpretation is the notion that deficient response inhibition is a causal etiological factor in OCD (Harsányi et al., 2014; Menzies et al., 2007; Menzies et al., 2008). However, the scientific basis of these disorder-specific causal interpretations is lacking (Abramovitch & Abramowitz, 2014; Abramovitch & Schweiger, 2015). In fact, such causal interpretation has led to a number of studies attempting to target these neuropsychological 'causal' factors in treatment, by means of cognitive remediation, in the hope that treating the cognitive deficits would in turn improve psychopathological symptoms. Studies using this 'reverse causality' approach have produced disappointing results. Indeed, a recent review of this body of literature lead the authors to conclude "…*that cognitive deficits in most psychopathologies represent the effects, rather than the causes of psychopathology*." (Kim et al., 2018, p.

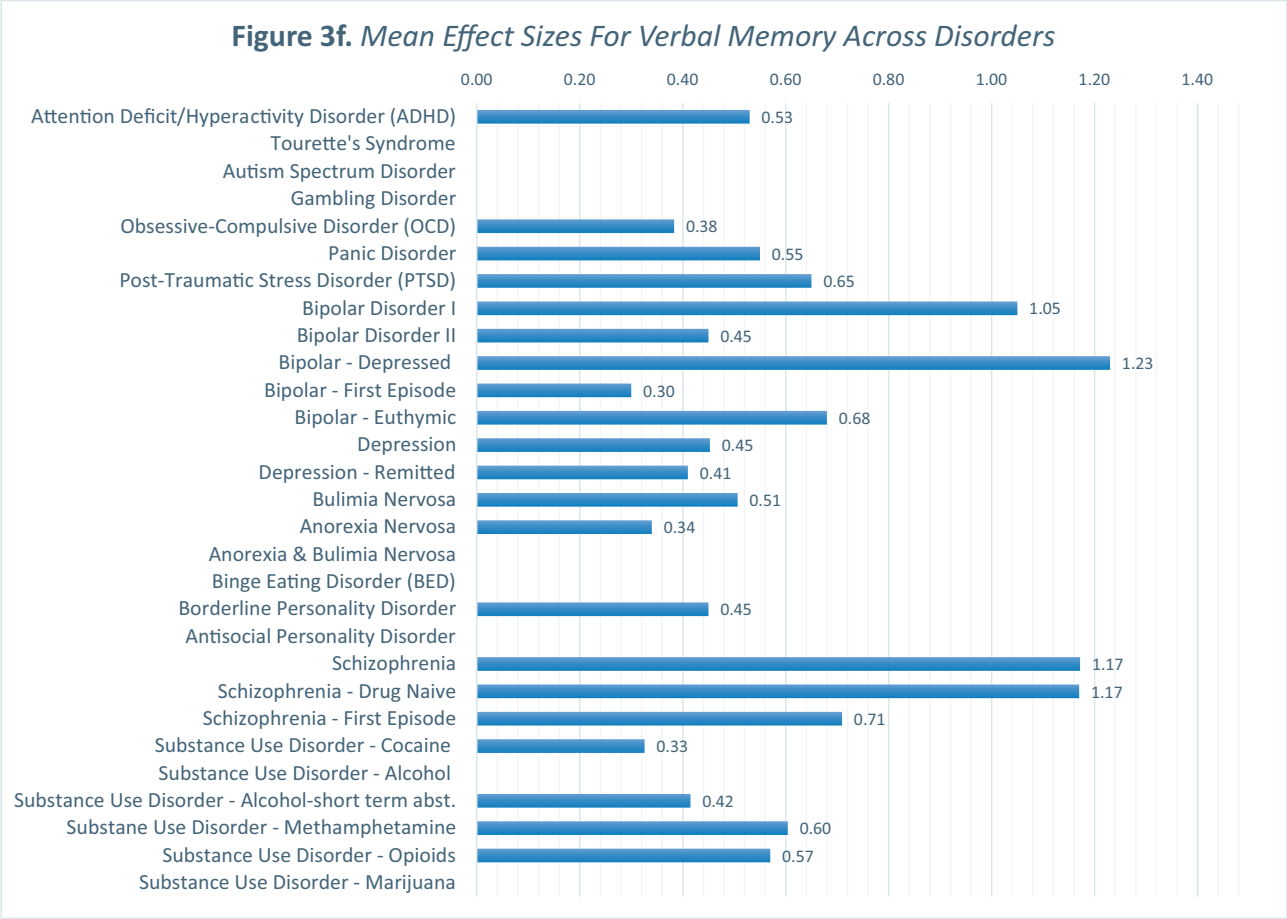

Fig. 3f. Mean effect sizes for verbal memory across disorders

14).

In sum, there are inherent limitations to the direct interpretation of neuropsychological test data in general, and particularly in the context of psychopathology. These limitations include specificity, multicollinearity, ecological validity, construct validity, and related misinterpretations. In addition, no systematic attempt to examine predictors of everyday functions in psychopathology was made, nor any empiric examination performed of what constitutes a neuropsychological impairment. These limitations preclude the conclusion that the results of the present study are indicative of cognitive *impairments* across all disorders and domains. Rather, these results suggest that there is a transdiagnostic phenomenon where underperformance on neuropsychological testing is found across cognitive domains. We turn now to consider potential factors which could account for the present findings.

4.2. Transdiagnostic constituents associated with the C factor

4.2.1. Biological explanation – The disease model of psychopathology

Perhaps a natural explanation for the ubiquitous lower scores on most neuropsychological tests across all forms of psychopathology is that psychopathology, in its multiform phenotypes, is the expression of brain pathology, either as structural and/or functional abnormalities. Indeed, in their review Caspi and Moffitt (2018) outline genetic research, as well as functional and structural brain imaging research, which lend support for a unitary p factor, rather than to more disorder-specific correlates. In the biological model of psychopathology, the primary etiological factor is heritability of categorical disorders, or a predisposition to develop such conditions. That is, abnormalities resulting in psychiatric disorders are presumed to reflect genetic variations, which may emerge at any developmental stage. The resulting genetic abnormalities may give rise to structural and functional brain abnormalities, which produce various phenotypic disorders and, presumably, also cognitive difficulties. As a matter of an overarching model, this explanation is problematic, since all psychic life, all behavior and emotions, involve the functioning of the brain, so to state that abnormality of the psyche is the result of abnormality in the brain is analogous to stating that the cause of wind is air motion. As aptly put by Lilienfeld (2014):

"*Yet investigators…must be careful not to confuse biological mediation with biological etiology. This tempting semantic slippage is subtle but substantial in its implications. For example, in principle, a psychological condition could be triggered largely by psychosocial factors, such as childhood sexual or physical abuse. Although this condition would of course be mediated by brain circuitry, its etiology would be primarily environmental.*" (Lilienfeld, 2014, p. 132).

In order to avoid this circularity, and make the causal explanation a coherent one, the biological explanation requires the individuation of syndromes and symptoms to particular abnormalities of processes, neuroanatomical structures (localization), neurotransmitters, genes, etc. In other words, in the framework of the biological model, any form of psychopathology, or 'mental illness', will ultimately be reduced to, and causally explained by, biological abnormalities. However, attempts to link discrete pathological mental functions to cerebral abnormalities have not been successful, and even the most sophisticated imaging technologies has yet to definitively link any neuroanatomical region with a specific disorder. Nor has the search for genetic, neurotransmitter, or other functional bases of brain tissue resulted in better understanding of psychiatric disorders or their treatment to date (Deacon, 2013). Indeed, this state of affairs has been articulated by the American

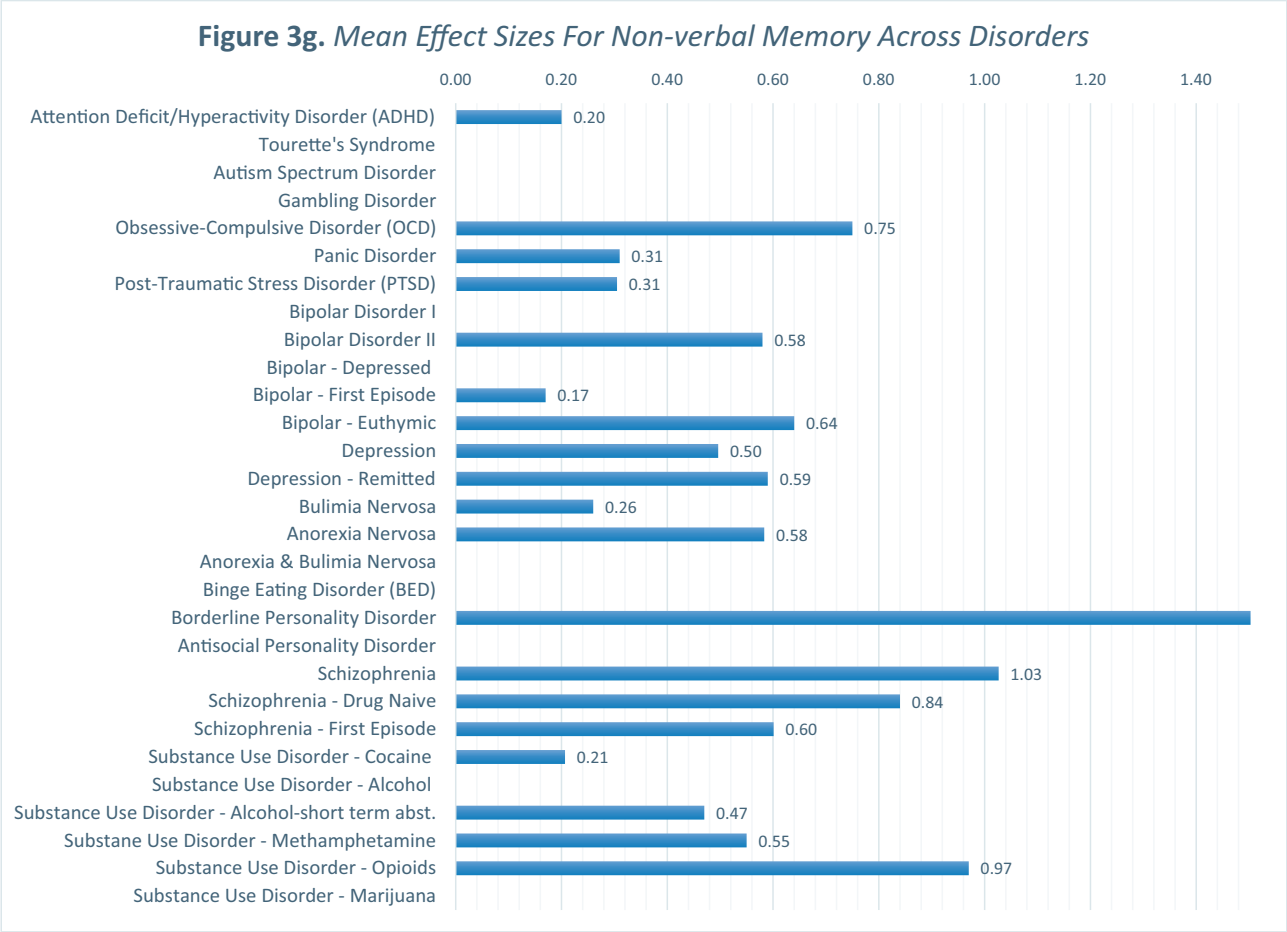

Fig. 3g. Mean effect sizes for non-verbal memory across disorders

Psychiatric Association: *"…brain science has not advanced to the point where scientists or clinicians can point to readily discernible pathologic lesions or genetic abnormalities that in and of themselves serve as reliable or predictive biomarkers of a given mental disorder or mental disorders as a group"* (APA, 2003).

Nevertheless, the search for discrete patterns of neuropsychological test performance as correlates for one disorder or another is not without merit. For example, the present study reveals that certain diagnostic categories that represent more severe p are associated with larger c (e.g., the largest effects for neuropsychological deficits in the present review were found in SCZ). Such results seem to encourage the search for neurocognitive endophenotypes via neuropsychological profiling. But the presence of diminished neuropsychological test performance may not reflect an underlying endophenotype, but rather, reflect the **consequence** of psychological changes related to multiple factors and their interactions, potentially including familial and genetic risks. Thus, a more viable approach is to examine interactions among psychological and biological aspects without preconception about causality (e.g., Caspi et al., 2002).

4.2.1.1. Medications. Among clinical populations, the majority of individuals seeking treatment for mental health conditions are prescribed at least one psychotropic drug. Thus, it stands to reason that most participants covered in the present study were likely to be medicated at the time of neuropsychological testing (although a plurality of studies disallowed intake of stimulants or benzodiazepines at the day of testing). Ample research indicates that three classes of psychotropic medications may have significant deleterious impact on performance of cognitive tasks, namely, neuroleptics (i.e., antipsychotics), antiepileptic (i.e., 'mood stabilizers'), and benzodiazepines (Barker, Greenwood, Jackson, & Crowe, 2004; Nevado-Holgado, Kim, Winchester, Gallacher, & Lovestone, 2016; Prado, Watt, & Crowe, 2018; Stein & Strickland, 1998). Moreover, long term use of these three drug classes is associated with further reduced cognitive performance and a dose-related effect on cognition is frequently reported (Barker et al., 2004; Helmstaedter & Witt, 2020; Husa et al., 2017).

It should be emphasized that polypharmacy, either within, or more commonly between, drug classes, is exceedingly prevalent (Guthrie, Makubate, Hernandez-Santiago, & Dreischulte, 2015). For example, a large study examining polypharmacy in office-based psychiatric practice, indicated a rise in polypharmacy where in 2006 60% of the sample were receiving $\geq$2 medications, and 33% were receiving $\geq$3 medications (Mojtabai & Olfson, 2010), and even among depressed patients seen in psychiatrists' private practice, 58% receive polypharmacy (Glezer, Byatt, Cook Jr., & Rothschild, 2009). Polypharmacy in hospital/in-patients settings ranges between 80-90% of patients, with some studies reporting a mean concomitant medications in the past two weeks that is $>$ 5 (Carmona-Huerta et al., 2019; Chakos et al., 2006; Kukreja, Kalra, Shah, & Shrivastava, 2013). Importantly, ample evidence indicates that the degree of cognitive deficiencies from polypharmacy exceed those due to monotherapy (Elie et al., 2010; Rawle, Cooper, Kuh, & Richards, 2018). As such, psychiatric medications may partially explain cognitive deficiencies in psychopathology.

Results of the present study may reflect these findings, since disorders associated with psychotic symptoms, increased neuroleptics use and higher prevalence of polypharmacy involving neuroleptics, were found to be associated with larger effect sizes, reflecting more severe cognitive deficiency. Notably, most studies covered in this review

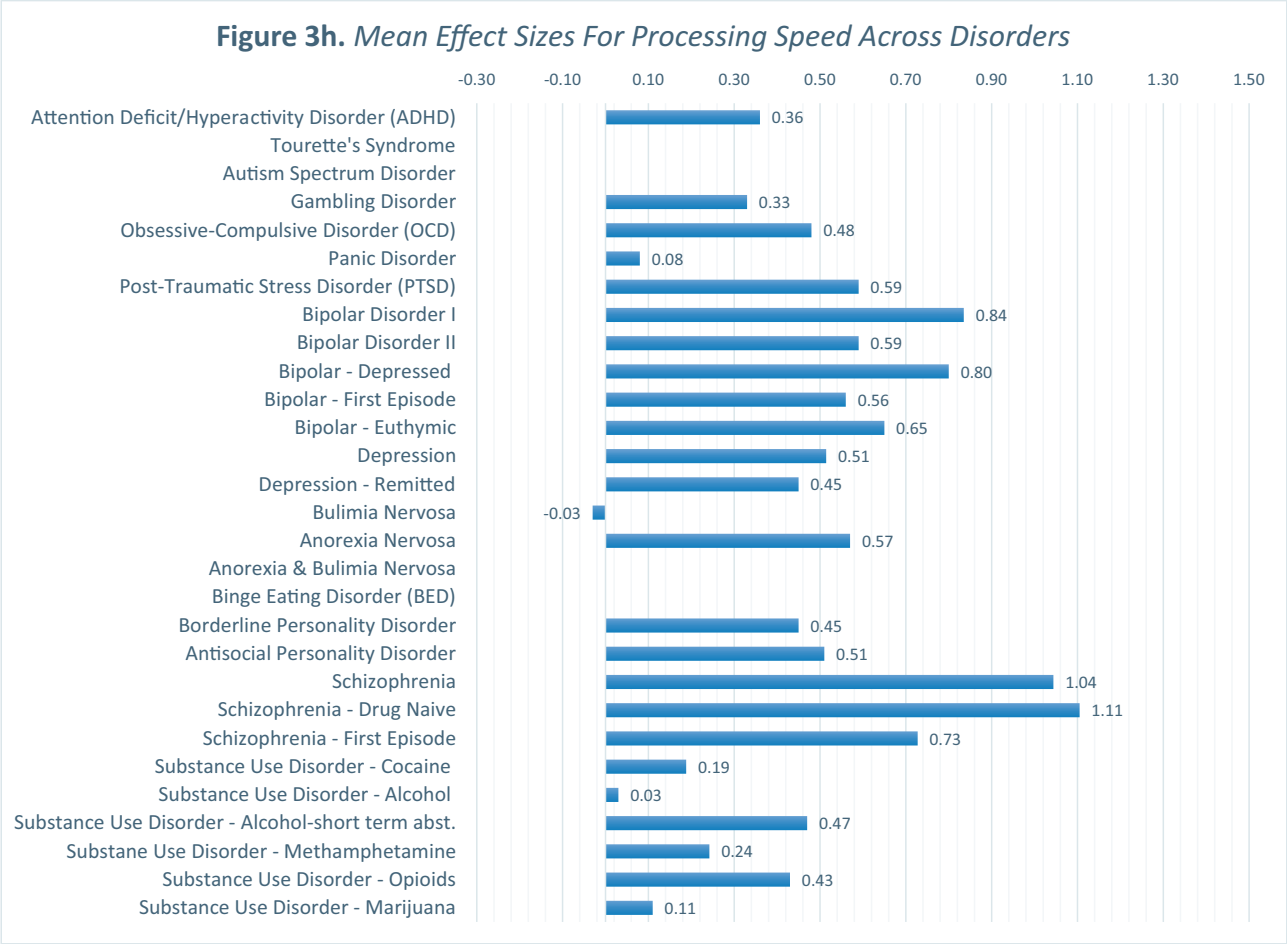

Fig. 3h. Mean effect sizes for processing speed across disorders

include treatment-seeking individuals, the plurality of whom receive medications, and approximately half of those patients receive polypharmacy. However, the vast majority of studies report the distribution of participants taking specific medications, but rarely provide information about the prevalence of participants receiving polypharmacy.

In sum, the results of the present review do not support any neuropsychological profile, domain, subdomain, or tests, which are correlated with any one particular disorder. More importantly, the contemporary biological perspective of psychopathology does not offer a coherent biological explanation for the ubiquity of cognitive deficiencies that accompanies psychopathology. In contrast, some evidence suggests that most forms of psychotropic treatments, while resulting in symptom reductions, may actually lead to cognitive deficiency as well, particularly in cases of polypharmacy. Although in the context of the biomedical model of psychopathology some have argued that the holy grail of biomarker research in psychiatry lies in future sophisticated interactive models incorporating multiple physiological markers (e.g., Scarr et al., 2015), it is crucial to examine psychological factors found across disorders that may be causally, or reciprocally related to cognitive deficiencies.

4.3. Motivation: Effort, reward, and anhedonia

4.3.1. Effort

Diagnosis of DSM disorders relies heavily on subjective self-report, and thus symptom production and exaggeration impede the accurate assessment of DSM disorders. Similarly, neuropsychological testing is subject to response biases that can range from deliberate feigning in order to obtain primary or secondary gain, to poor motivation due to apathy or lack of interest (Lezak et al., 2012). In fact, the overall prevalence of probable malingering and symptom magnification in neuropsychological assessment ranges from 8-35%, depending on the context in which the assessment is conducted (Mittenberg, Patton, Canyock, & Condit, 2002). Multiple tests were developed to identify response bias. These tests are known colloquially as 'effort tests' and formally as Performance Validity Tests (PVT; Vickery, Berry, Inman, Harris, & Orey, 2001). In addition, multiple built-in algorithms have been developed to facilitate identification of response bias using classic neuropsychological tests (Millis & Volinsky, 2001). The use of these tests in any neuropsychological assessment is of critical importance, as reflected in a consensus statement by the American Academy of Clinical Neuropsychology (AACN) on effort, response bias and malingering (Heilbronner et al., 2009). These guidelines clearly state that if following proper utilization of PVT it has been determined that the examinee's performance is non-credible, the results become invalid. Most studies that assess effort involve brain injured individuals in forensic settings associated with potential for primary and secondary gains. Such gains are typically not present in research settings, which may be the underlying reason why it is extremely rare to find a study examining cognitive function in psychopathology that utilizes PVT. However, the presence of psychopathology, particularly psychotic disorders (Goldberg, Back-Madruga, & Boone, 2007), but also depressive, anxiety and dissociative disorders, is associated with reduced effort on neuropsychological tests, even in the absence of litigation and compensation.

Although relatively under-researched (Goldberg et al., 2007), data are available regarding non-severe psychiatric disorders and feigned cognitive impairments. Several studies documented failure on effort tests ranging between 14-75% across non-severe disorders that was

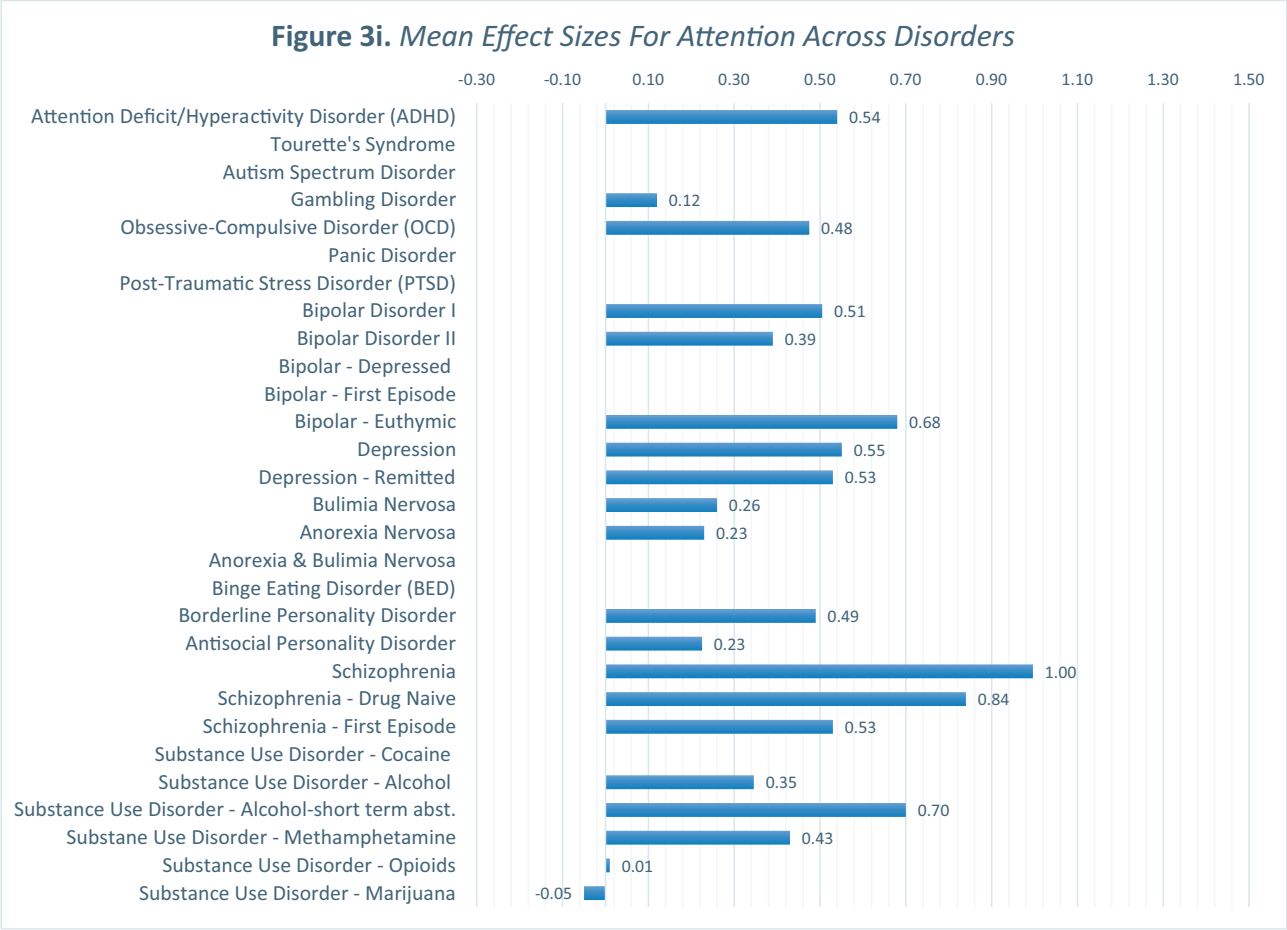

Fig. 3i. Mean effect sizes for attention across disorders

associated with under performance on neuropsychological tests (Dandachi-FitzGerald, Ponds, Peters, & Merckelbach, 2011; Gorissen, Sanz, & Schmand, 2005; McWhirter, Ritchie, Stone, & Carson, 2020). Notably, multiple studies examined effort in PTSD, with high rates of failure on PVT's (Demakis, Gervais, & Rohling, 2008). The most consistent findings for PVT failures in psychopathology, however, relates to schizophrenia, psychotic disorders, and depression with psychotic symptoms, particularly in the context of negative symptoms. Indeed, high rates of failing PVTs, ranging between 22% (Schroeder & Marshall, 2011) to 72% (Gorissen et al., 2005) have been consistently found among these conditions, both within and outside the context of litigation, or any obvious external primary or secondary gain (Marcopulos et al., 2014). These very high rates, particularly in inpatient settings, may in part affect account for some of the C factor's variance. It is important to note that reduced effort may stem from a multitude of possible factors, including lack of interest in neuropsychological assessment, general indifference, irritability, or assigning low importance to neuropsychological assessment (Heilbronner et al., 2009; Marcopulos et al., 2014). In accordance with the foregoing, studies show that monetary incentives result in a significant improvement in test performance, including memory, attention and cognitive control (Madan & Spetch, 2012; Robinson et al., 2012).

In sum, there is ample evidence that effort on neuropsychological tasks may substantially alter performance, including in the context of psychopathology. However, the scarcity of research pertaining to motivation in the context of neuropsychological testing across most DSM disorders hinders a cogent inference regarding the transdiagnostic impact of effort. It is clear, then, that further research is required, and researchers examining cognitive function in clinical populations are encouraged to assess effort using valid PVT's.

4.3.2. Reward and anhedonia

Additional consideration of motivation-related factors that may be associated with the C factor in psychopathology are neurobiological alterations in reward processing and reactivity, and a related psychological construct: anhedonia. Research into cognitive functions in the context of psychopathology usually entails completion of multiple questionnaires, a semi-structured diagnostic interview, and extensive neuropsychological batteries. The latter requires exertion of cognitive effort for prolonged periods of time. Studies indicate that performance on cognitive tests, such as tests of cognitive control, working memory, memory and attention, is positively associated with reward processes and incentives (Ferdinand & Czernochowski, 2018; Salamone, Yohn, López-Cruz, San Miguel, & Correa, 2016; Williams, Biel, Dyson, & Spaniol, 2017; Wittmann et al., 2005).

Extensive literature has documented aberrant reward reactivity in a variety of mental disorders when compared to controls. These include MDD (Bogdan, Nikolova, & Pizzagalli, 2013), PTSD (Nawijn et al., 2015), OCD (Figee et al., 2016), SCZ (Strauss, Waltz, & Gold, 2014), social anxiety (Richey et al., 2014) BD (Alloy, Olino, Freed, & Nusslock, 2016), personality disorders, (Murray, Waller, & Hyde, 2018; Völlm et al., 2007), ADHD (Volkow et al., 2009), and substance abuse (Cooper, Robison, & Mazei-Robison, 2017). In fact, it has been suggested that abnormal reward reactivity (i.e., reduced activity in reward pathways/regions compared to controls) is a transdiagnostic risk factor across disorders (Baskin-Sommers & Foti, 2015; A. Sharma et al., 2017). In turn, ample research shows that abnormal reward reactivity and the presence and value of incentives directly impact performance on

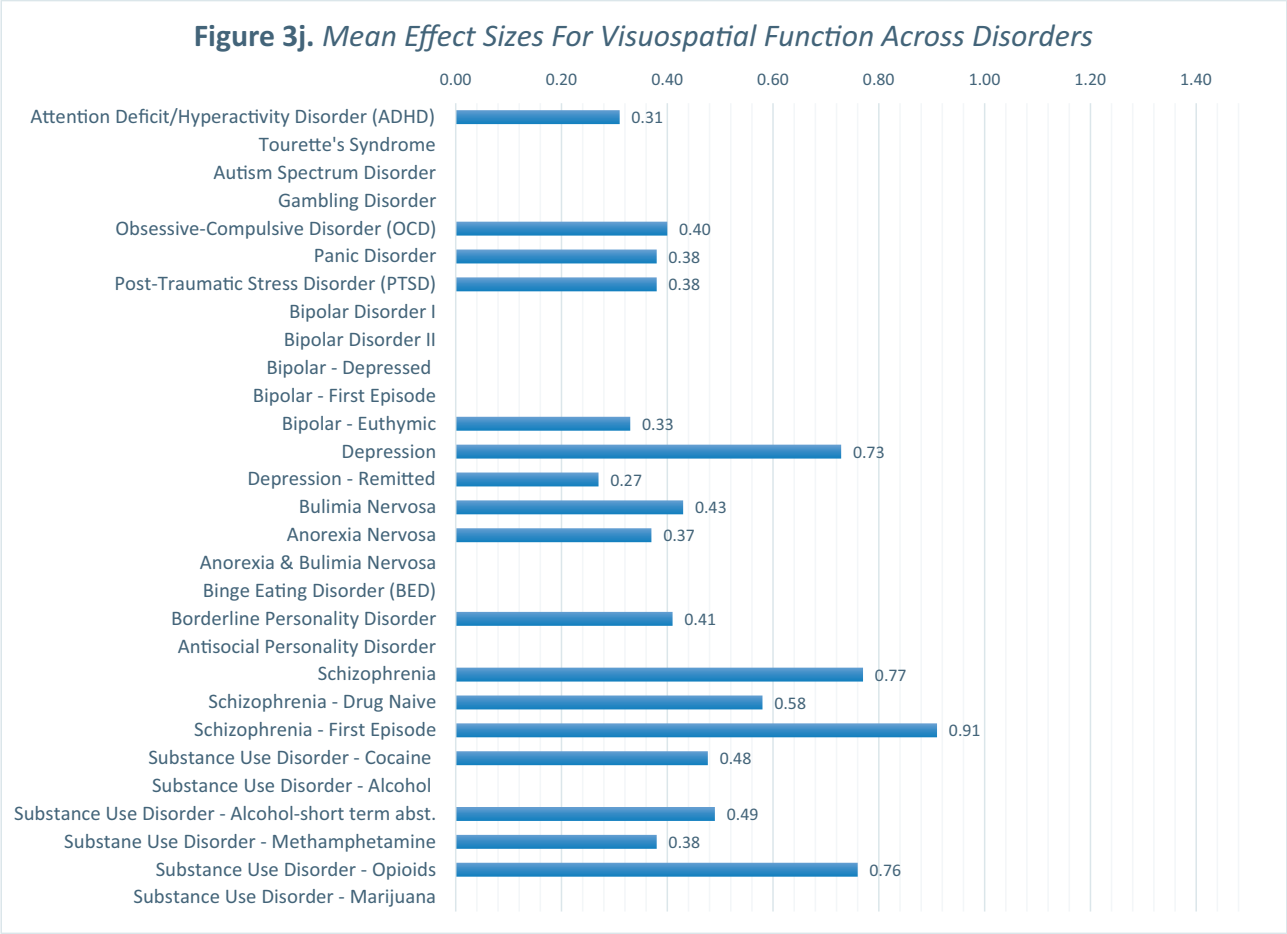

Fig. 3j. Mean effect sizes for visuospatial function across disorders

cognitive tests. Similar findings have been found in DSM disorders. For example, individuals diagnosed with MDD and SCZ tend to bias their decision making toward reduced effort (Salamone et al., 2016). Others have documented that reduced motivation and effort were associated with underperformance across neuropsychological domains in SCZ accounting for 15% of the C factor variance (Foussias et al., 2015), Accordingly, associations between reward dysfunction and underperformance on cognitive tests were reported in multiple disorders including MDD (Subramaniapillai et al., 2019), ADHD (Marx, Höpcke, Berger, Wandschneider, & Herpertz, 2013), PTSD (Hayes, VanElzakker, & Shin, 2012), GD (Potenza, 2014), SUD (Motzkin, Baskin-Sommers, Newman, Kiehl, & Koenigs, 2014), and binge eating disorder (Balodis et al., 2013).

Anhedonia, which refers to the inability, or reduced ability to experience pleasure, is a robust psychological construct closely associated with lowered reward reactivity, particularly in the ventral striatum/nucleus accumbens (Wacker, Dillon, & Pizzagalli, 2009). Anhedonia has been identified in multiple DSM disorders including depression (Elhai et al., 2012), OCD (Abramovitch, Pizzagalli, Reuman, & Wilhelm, 2014), hoarding disorder (Pushkarskaya et al., 2019), PTSD (Kashdan, Elhai, & Frueh, 2006), substance use disorders (Hatzigiakoumis, Martinotti, Di Giannantonio, & Janiri, 2011), illness (health) anxiety (Skjernov et al., 2020), and BD (Rizvi, Lambert, & Kennedy, 2018). In fact, it has recently been proposed that anhedonia should be considered as a transdiagnostic construct in psychiatry (Husain & Roiser, 2018). Given the close association between anhedonia and deficient reward responsivity, it is unsurprising that anhedonia is linked to various alterations in cognitive functions, including memory, attention, cognitive control and decision making (Dillon & Pizzagalli, 2018; Dubal, Pierson, & Jouvent, 2000; Franken, Van Strien, & Nijs, 2006; Huys, Pizzagalli, Bogdan, & Dayan, 2013; McIntyre et al., 2016)

In sum, reward system dysfunction and anhedonia are likely to be transdiagnostic across DSM disorders. In turn, these constructs are related to poorer cognitive function across neuropsychological domains, with evidence for this association in multiple DSM disorders. Thus, it is plausible that anhedonia contributes, at least in part, to the ubiquity of cognitive deficits across disorders.

4.4. Meta-cognition in psychopathology: Confidence, self-efficacy, and self-stigma

Meta-cognition is the capacity to assess, reflect, control, and evaluate one's cognitions (Rouault, McWilliams, Allen, & Fleming, 2018). Alterations in various aspects of meta-cognition have been observed across disorders (Sun, Zhu, & So, 2017). The more prominent alterations in meta-cognition that may impact cognitive function include self-efficacy (Bandura, Caprara, Barbaranelli, Gerbino, & Pastorelli, 2003), self-stigma (Corrigan, Larson, & Rusch, 2009), and more recently, attitudes toward neuropsychological testing (S. Moritz et al., 2017).

4.4.1. Self-efficacy

Albert Bandura originally defined Self-efficacy as *"...people's judgments of their capabilities to organize and execute courses of action required to attain designated types of performances. It is concerned not with the skills one has but with the judgments of what one can do with whatever skills one possesses*" (Bandura, 1986, p. 391). Importantly, self-efficacy is closely related to both motivation and performance, including performance related to cognitive function (Ouweneel, Schaufeli, & Le Blanc, 2013;

Schunk, 1995). Self-efficacy has been found to play an important role in psychopathology across age groups and geographic regions, most notably in the context of symptoms of anxiety, stress, and depression (e.g., Luszczynska, Gutiérrez-Doña, & Schwarzer, 2005). Indeed, longitudinal studies found that high self-efficacy is associated with fewer depressive symptoms, and that an increase in self efficacy leads to reduction in symptoms of depression (W. D. Scott & Dearing, 2012). Other studies reported a 2-3 fold higher risk for psychopathology among people with low self-efficacy (Andersson, Moore, Hensing, Krantz, & Staland-Nyman, 2014).

As noted by Bandura and Cervone (1983), individuals with lower self-esteem may be more strongly discouraged from making an error, which may further reduce test performance, while people with higher self-efficacy may be motivated to perform better (Bandura & Cervone, 1983). Interestingly, this has been empirically tested in a sample of MDD versus controls (Elliott et al., 1996). The authors found that committing errors resulted in a subsequent increase in errors in the MDD sample, but not in the control sample. More generally, self-efficacy has been found to be associated with academic performance (Bandura, Barbaranelli, Caprara, & Pastorelli, 1996), but also with verbal, spatial, and numerical neuropsychological test performance (Paunonen & Hong, 2010), and with performance on IQ tests (Jurecska Diomaris, Lee Chloe, Chang Kelly, & Sequeira, 2011). Other experimental studies showed that threat to self-esteem impairs selective attention via elevation of state anxiety and leads to diversion of attentional resources to task-irrelevant stimuli (Hedva Braunstein-Bercovitz, 2003; H. Braunstein-Bercovitz, Dimentman-Ashkenazi, & Lubow, 2001). Finally, threats to self-esteem were also found to impair cognitive control (Keinan, Friedland, Kahneman, & Roth, 1999).

In sum, a decline in self-efficacy and self-esteem is associated with psychopathology and appears to be transdiagnostic. These constructs affect general motivation and have been found to play a causal role in deficient performance on an array of cognitive tasks.

4.4.2. Self-stigma

Stigma about psychological disorders is a pervasive and persistent global issue, affecting both the lay public as well as mental health professionals across disciplines (Corrigan, 2000). A related construct, self-stigma, is an omnipresent construct that has been extensively studied in psychopathology. Research on self-stigma in psychopathology is important in elucidating how societal stereotypes and prejudice regarding psychopathology negatively impact people who suffer from psychopathology, and ultimately affect performance via reduction in self-efficacy and self-esteem. More importantly, an associated 'Why Try' model elucidates the importance of enhanced avoidance, an important factor associated with low self-efficacy (e.g., "*why should I even try to get a job?*"; "*Why should I even bother to make an effort…*?"; Corrigan et al., 2009). Although no research has been done to examine the direct impact of self-stigma on the C factor, its indirect effect on the C factor via self-efficacy suggests that this is a worthwhile venue for further research.

4.4.3. Attitude toward cognitive testing

Recent research examined the association between individuals' attitudes toward neuropsychological testing and actual test performance in several disorders. Results from a series of studies in participants with OCD, MDD, and SCZ, as well as control samples, showed that, compared with controls, underperformance among clinical samples was to a large extent mediated by attitudes towards testing, as reported prospectively and retrospectively (S. Moritz, Hauschildt, Saathoff, & Jelinek, 2017; S. Moritz et al., 2017). Although more research is needed, there is a good reason to suspect that some individuals may indeed experience difficulties in the context of being tested on a battery of tests assessing multiple cognitive function (e.g., individuals with elevated perfectionism). Nevertheless, the question regarding the type and extent of the impact of attitudes toward testing on test performance requires further study.

Taken together, ample evidence exists to support the notion that meta-cognitive factors, attribution and perceptions associated with psychopathology affect the C factor in psychopathology. However, more direct experimental and longitudinal studies are warranted in clinical and analogue samples to clarify the extent of meta-cognition in impairing neuropsychological performance.

4.5. Psychopathology and cognitive function – Transdiagnostic resource models

The transdiagnostic factors reviewed above may contribute to our understanding of the central finding of this study, namely, that the C factor is ubiquitous in psychopathology and is associated with the broad spectrum of DSM disorders, as well as with dimensional traits and symptoms. Other than pharmacotherapy, the transdiagnostic factors reviewed above could be conceptualized as different motivational aspects associated with multidimensional effects of psychopathology. Taken together, these putative contributing factors can also be viewed conceptually as consequences of the overall burden of psychopathology. Such a conceptualization may be aligned with the p factor model, particularly given two central premises of the model (Caspi et al., 2014). Since its inception, the p factor model percieved cognitive dysfunction as an integral part of p, and the notion of a-priori limited resources has been discussed in this context (Caspi et al., 2014; Caspi & Moffitt, 2018). Several resource models have been proposed offering important insight as to the potential mechanisms linking core transdiagnostic symptoms (i.e., negative affect, anxiety and stress) and cognitive dysfunction.

Resource theory is a global framework suggesting that a general reservoir of mental resources exist, from which organisms draw to complete task demands (Kahneman, 1973). This model posits that effort is associated with attention control, and that the task demand entails the amount of effort that is required, and that failure to meet the required effort level would results in deficient task performance. Mental effort may be regulated based on individual appraisal of the task demand, the person's current level of performance, and one's subjective evaluation (self-appraisal) of stress and comfort. Pertaining to the latter point, research indicates that stress alters the total *quantity* of available resources and that overall changes in resources availability are a function of emotion-focused coping (Staal, 2004). Consequently, performance on neuropsychological tests may be suboptimal when resources are depleted, perceived to be limited, or reduced by affective burden such as depression, anxiety, and stress – the hallmark symptoms or sequelae of most psychological disorders.

Models examining the impact of stressors on performance define stress as the interaction between the perceived demand, the *perceived ability* to cope, and the perception of the *importance* of one's ability to cope with the demand. Such framework, therefore, is consistent with research findings that threat to self-esteem leads to reduced performance on tasks assessing attention control and selective attention (Hedva Braunstein-Bercovitz, 2003; H. Braunstein-Bercovitz et al., 2001). Along these lines, Carver, Johnson, and Timpano (2017) recently offered substantive account of a construct that may underlie the p factor, namely a transdiagnostic 'impulsive responsivity to emotions'. Other models attempted to address the impact of specific aspects of general stress upon cognitive functions. For example Eysenck and colleagues developed a comprehensive resource depletion model, namely the Attentional Control Theory (Eysenck, Derakshan, Santos, & Calvo, 2007). This theory asserts that anxiety depletes attentional control resources which will result in reduced processing efficiency, primarily affecting response inhibition, set shifting and working memory, and ample research is available to support this prediction (e.g., Berggren, Richards, Taylor, & Derakshan, 2013; Najmi, Amir, Frosio, & Ayers, 2015).

Given that that these models' operationalization of stress include anxiety, negative affect, and general distress, all of which are ubiquitous in psychopathology, and given that psychopathology is characterized by

disturbance in motivation and self-perceptions, it is plausible that reduced availability of resources may hold explanatory power for the transdiagnostic phenomena of deficient cognitive function in psychopathology - the C factor.

4.6. Strengths and limitations of the present study

The strengths of the present study include the systematic nature of this review that, together with its scope, entails a comprehensive account of all available data on cognitive function and psychopathology to date. This literature review was augmented by a review of research into cognitive function across dimensional studies of psychopathology, as well as across major disorders for which meta analyses were not available. However, the present study is not free of limitations. First, the discussion of potential account for the findings of the present study was not intended to be an exhaustive review of every potential etiological factor that may contribute to the C factor. Rather, we discussed primary factors that should be considered, to encourage a necessary discourse which will hopefully lead to further interdisciplinary research of the transdiagnostic mechanisms underlying our findings. In addition, as noted in the methods section, we were unable to obtain information regarding which specific studies were included in most of the meta-analyses reviewed herein. Thus, there is inherent overlap in studies that may appear in more than one meta-analysis. Given that many meta-analyses did not include a list of included studies, and that our attempt to contact authors to obtain such lists was only partially successful, we opted to present all effect sizes, as well as unweighted means, thus enabling readers to evaluate the literature independently. Notably, our results indicate that different meta-analyses that examined similar populations/disorders, and employed similar exclusion and inclusion criteria, yield similar effect sizes for neuropsychological domains. Consequently, such overlap does not pose a threat for the integrity of our results.

5. Conclusion

The results of the present study can be condensed into three main conclusions. First, psychopathology (or p), categorically or dimensionally defined, is associated with broad underperformance on cognitive tasks across cognitive domains, hence the C factor should be viewed as a transdiagnostic phenomenon related to the presence of psychopathology, and not to any specific disorder. Second, results support the notion that elevated p is associated with elevated c, where greater deficiency was found in SCZ and bipolar disorder. Across other disorders, effect sizes indicative of suboptimal performance were largely small to medium in magnitude. Third, a review of potential transdiagnostic features that may explain this phenomenon supports a conclusion that motivational and emotional factors across psychopathology have substantial impact on neuropsychological test performance. However, this conclusion may be viewed as preliminary, given the lack of research directly examining this association. In addition, polypharmacy is another factor which must be considered as contributing to the C factor, and should be addressed in research on cognitive functions in psychopathology.

This review underscores the need to address transdiagnostic factors such as effort, motivation, self-efficacy, self-stigma, anhedonia, polypharmacy, attitudes toward testing, as well as the limitations of neuropsychological research in psychopathology, in the context of assessment of cognitive functions and its interpretations. We hope that this review would be utilized as a roadmap for further research. In addition, decades of research resulted in an abundance of data comparing clinical and non-clinical samples on classic neuropsychological batteries. Given the plurality of data suggesting that cognitive deficiencies are transdiagnostic, we join others in recommending that there is no need for additional simple descriptive investigations of this type across most disorders (Abramovitch & Cooperman, 2015; Snyder, Miyake et al., 2015). Future studies should address motivational aspects impacting cognitive functions in different clinical and non-clinical populations and examine longitudinal changes. In terms of neuropsychological assessment in psychopathology per se, future studies should incorporate effort testing, report and control for polypharmacy, examine changes in test performance as a function of response to treatment, and transition their focus to incorporate more tests utilizing the verisimilitude approach, thereby increasing ecological validity, utility, and facilitating generalizability.

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AA conceptualized the study and conducted data analyses, literature search, and wrote the first draft of the study. AS contributed to the conceptualization of the study and co-wrote the first draft of the study. TS conducted literature search, data entry, and data analysis. All authors approved the final version of the manuscript.

Declaration of Competing Interest

The authors declare no conflict of interest pertaining to the present work

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