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The Neuropsychology of Borderline Personality Disorder:

A Meta-analysis and Review

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Abstract

The neuropsychological profile of borderline personality disorder (BPD) is unclear. Past investigations have produced seemingly inconsistent results of precisely what neuropsychological deficits characterize the patient with BPD. A meta-analysis of 10 studies was conducted comparing BPD and healthy comparison groups on select neuropsychological measures comprising six domains of functioning: attention, cognitive flexibility, learning and memory, planning, speeded processing, and visuospatial abilities. BPD participants performed more poorly than controls across all neuropsychological domains, with mean effect sizes (Cohen's d) ranging from $-.29$ for cognitive flexibility to -1.43 for planning. The results suggest that persons with BPD perform more poorly than healthy comparison groups in multiple neurocognitive domains and that these deficits may be more strongly lateralized to the right hemisphere. Although neuropsychological testing appears to be sensitive to the neurocognitive deficits of BPD, the clinical utility of these results is limited. Implications of these findings for future neurocognitive investigations of BPD are discussed.

Key words: Mental Disorders, Personality Disorders, Cognition

1. Introduction

Borderline personality disorder (BPD) is a disorder characterized by affective instability, impulsivity, cognitive disruptions, and interpersonal difficulties (American Psychiatric Association, 2000) and affects approximately 2% of the population (Swartz et al., 1990). The literature of BPD abounds with reports of memory (Korfine and Hooley, 2000; Startup et al., 2001) and perceptual distortions (George and Soloff, 1986; Sundbom et al., 1989; Yee et al., 2005), symptoms suggesting a potential underlying brain pathology in this often chronic psychiatric disorder. However, a clear characterization of the neurocognitive features of BPD has proven elusive.

While initial neurobehavioral studies of BPD appeared to demonstrate a link between acquired or developmental brain dysfunction and borderline psychopathology (Andrulonis et al., 1980; van Reekum et al., 1993; van Reekum et al., 1996), these early neuropsychological investigations failed to present a consistent pattern of neurocognitive disruption. For instance, a study by Cornelius et al. (1989) was unable to detect differences between BPD patients and a healthy control group selected from historical records in the domains of memory, language, motor, and spatial functioning.

O'Leary et al. (1991) were among the first to utilize a more methodologically sound approach to examining the neurocognition of BPD, revealing distinct impairments in BPD participants relative to controls primarily in tasks assessing memory, as well as the processes of visual discrimination and filtering. These results were largely supported by studies carried out by Judd and Ruff (1993) and Swirsky-Sacchetti et al. (1993). Although the latter investigation failed to replicate a decrement in performance for the BPD group on the digit-symbol test of the Wechsler Adult Intelligence Scale–Revised

(WAIS-R; 1981), it was the only study to find a difference between groups in the interference condition of the Stroop Color and Word Test (Golden, 1978). In addition, using a short 11-item neurocognitive screening examination, Burgess (1990) demonstrated significant differences between groups on measures of memory and rhythm reproduction.

Many of the more recent neuropsychological investigations of BPD utilized more comprehensive neuropsychological batteries when compared with earlier studies and seemed to identify specific neurocognitive impairments among BPD participants. Dinn et al. (2004) compared BPD and healthy control groups in a number of cognitive domains, particularly a number of tasks assessing multiple facets of attention. BPD patients were impaired on tests of visuospatial abilities, speeded processing, and nonverbal memory skills, yet there were no striking differences observed on tests of attention, verbal memory, and alternation learning. Additional neurocognitive impairments associated with BPD were demonstrated by Bazanis et al. (2002), in which BPD participants performed more poorly on tasks assessing planning and decision-making but no differences between groups in tests of visual recognition memory, including pattern and spatial recognition. Further, Posner et al. (2002) identified a specific deficiency in an attentional network involved in conflict resolution and cognitive control, which was distinct from systems involved in emotion regulation.

Despite the abundance of evidence in support of neurocognitive deficits in BPD, many investigations have failed to identify any remarkable differences between BPD and healthy control groups. Kunert et al. (2003) conducted extensive neuropsychological testing of BPD and healthy control participants, including assessments of intelligence,

attention, visual scanning, cognitive flexibility, working memory, planning and problem solving, and learning and memory. Although BPD participants demonstrated higher scores on self-report measures of aggressiveness and impulsiveness, the patient group only performed more poorly than controls in the reading condition of the Stroop test and made more errors in the interference condition. Similarly, Sprock et al. (2000) observed no differences between groups on any of the neuropsychological measures they employed with the exception of one non-interference condition of the Stroop test in which BPD participants took significantly longer than the healthy control group to name color-congruent words. Theunissen and Walker (2003) also found no differences between BPD and depressed controls on measures of working memory, speeded processing, cognitive flexibility, planning, and visuospatial abilities.

Evidently the relationship between neurocognition and borderline psychopathology is unclear. The purpose of the present meta-analysis is to provide a unified examination of the current literature in order to explicate the specific neuropsychological domains of functioning that may be impaired in persons with BPD. One difficulty involved in interpreting individual investigations of BPD is the varied way in which BPD has been operationally defined, whether it be self-report, semi-structured interviews, or unstructured interviews. Studies have also differed widely in the specific neuropsychological measures used to assess the functional integrity of neural systems. These two sources of variability have contributed to an unclear understanding of the potential brain pathology that may underlie BPD, and it is expected that an amalgamation of these individual findings will generate a coherent characterization of the neurocognitive features of BPD.

2. Methodology

2.1 Selection of Studies

A search for articles was conducted using PubMed and PsycINFO with a combination of key words, including *neuropsychology*, *neurocognition*, *cognitive*, *borderline personality*, and *personality disorder*. All relevant references from articles obtained through this search were also reviewed for inclusion in the analysis. Table 1 presents descriptive data for the studies identified for inclusion in the meta-analysis.

Only those studies adhering to all of the following eligibility criteria were included in the meta-analysis: (1) data required to calculate effect sizes were available (means and standard deviations of each neuropsychological measure for each group); (2) BPD participants were the population under study and not participants drawn from a non-clinical population scoring highly on BPD measures; (3) standardized, valid, and reliable neuropsychological tests were administered; (4) BPD participants met diagnostic criteria as set forth in DSM–III, DSM–III–R, DSM–IV, or ICD–10; and (5) studies were published in a peer-reviewed journal. Although a study would have been deemed eligible if additional information was obtained by contacting the authors, the author was unable to obtain such data upon request. Ten studies comprising 488 participants (BPD: $n = 225$, control: $n = 263$) satisfied eligibility requirements and were included in the meta-analysis.

2.2 Method of analysis.

Neuropsychological tests were categorized into one of six broad cognitive domains: attention, cognitive flexibility, learning and memory, planning, speeded processing, and visuospatial skills. Each test variable was coded into a single domain

judged to be best representative of the cognitive function being measured. Effect sizes for identified neuropsychological measures were calculated using the *Meta-Analysis Programs* (Schwarzer, 1994). The calculations for the formulas used in the analysis are from Hedges and Olkin (1985). Effect sizes were calculated by dividing the difference between the means of the BPD and healthy participant groups on each of the neuropsychological measures by the standard deviation (Glass, 1976). Because the effect size formula utilized in this investigation has a small-sample bias it was adjusted for using the formula for the unbiased estimator, d (see Hedges and Olkin, 1985).

The formula used in the present study to calculate effect size can result in an overestimate of the difference when there is heterogeneity of the variances (heteroscedasticity) of the two groups being compared. Heteroscedasticity was examined by computing variance ratios (VR) for each neuropsychological measure (see Grissom and Kim, 2001). The VRs were generally in an acceptable range (1.00 to 3.68) with the exception of the copy accuracy score of the Rey-Osterreith Complex Figure Test, which was excluded in the analyses for those studies (as indicated) for which heteroscedasticity would have inappropriately skewed the effect size.

Table 2 lists effect sizes and corresponding cognitive domains for each neuropsychological variable included in the meta-analysis. Positive effect sizes represent data wherein the healthy group performed more poorly on the neuropsychological tests relative to the BPD group; negative effect sizes refer to those tests for which the BPD group performed more poorly relative to the healthy group. Because many studies employed multiple measures of the same neuropsychological domain, effect sizes were calculated within each study for each of the six identified domains. The unit of analysis

was the mean standardized difference for each neuropsychological domain across studies. The effect for each study across all neuropsychological domains was weighed according to its respective sample size (Hedges and Olkin, 1985). Tests of homogeneity of variance, reported using the Q statistic, were conducted for each cognitive domain to determine whether the effect sizes pooled across studies were derived from a single population. In cases where the value of Q exceeded the critical value of alpha ($p = 0.05$), the samples were not examined for potential moderator variables because the limited number of studies did not allow for a meaningful investigation of these variables.

3. Results

Cohen (1988) provided guidelines by which to interpret effect sizes. An effect size (d) of 0.2 is considered small, 0.5 medium, and 0.8 or higher large. Effect sizes were calculated between BPD and healthy control groups across studies for each neuropsychological domain.

3.1 Attention

Seven studies reported data incorporating measures of attention for a total of 327 participants (BPD: $n = 146$, control: $n = 181$). The mean effect size based on weighted effect sizes was -0.59 (SE = 0.22) with a range of -1.74 to -0.01. The effect size is significantly different from zero ($z = -4.47, p < 0.001$) and the sample of effect sizes is heterogeneous ($Q = 26.560, p < 0.001$).

3.2 Cognitive Flexibility

Data from five studies employing tasks requiring cognitive flexibility were examined with a total sample size of 258 participants (BPD: $n = 107$, control: $n = 151$). The weighted mean effect size was -0.29 (SE = 0.13) and effect sizes ranged from -1.06

to +0.11. The effect size is significantly different from zero ($z = -2.20, p = 0.01$) and the sample of effect sizes is homogeneous ($Q = 5.13, p = 0.40$).

3.3 Speeded processing

Data were reported for six studies assessing processing speed in a total of 213 participants (BPD: $n = 105$, control: $n = 108$). The mean weighted effect size was -0.68 (SE = 0.14) and ranged from -1.62 to -0.15. The effect size is different from zero ($z = -4.77, p < 0.001$) and the sample of effect sizes is homogeneous ($Q = 8.83, p = 0.12$).

3.4 Learning and Memory

Six investigations utilizing neuropsychological measures of memory reported data for a total of 196 participants (BPD: $n = 98$, control: $n = 98$). The mean weighted effect size was -0.66 (SE = 0.15) with effect sizes ranging from -1.60 to -0.16. The weighted mean effect size is significantly different from zero ($z = -4.42, p < 0.001$) and the sample of effect sizes is homogeneous ($Q = 10.86, p = 0.05$). To explore the nature of the memory deficits in BPD, further analyses were carried out to examine verbal and nonverbal measures of memory. A mean weighted effect size of -0.45 (SE = 0.16) was obtained for the verbal memory domain based on a sample size of 166 participants (BPD: $n = 83$, control: $n = 83$), which was significantly different from zero ($z = -2.88, p = 0.002$) and homogeneous ($Q = 3.21, p = 0.52$). For the domain of nonverbal memory, the mean weighted effect size was -1.59 (SE = .23) in a sample of 100 participants (BPD: $n = 50$, control: $n = 50$). This sample of effect sizes was significantly different from zero ($z = -6.82, p < 0.001$) and homogeneous ($Q = 4.33, p = 0.23$).

3.5 Planning

Two studies reported data for a total of 130 participants (BPD: $n = 65$, control: $n = 65$) in which neuropsychological tests of planning were administered. The mean weighted effect size was -1.43 ($SE = 0.20$) with effect sizes ranging from -3.93 to $+0.06$. The weighted mean effect size is significantly different from zero ($z = -6.16$, $p < 0.001$) and the sample of effect sizes is heterogeneous ($Q = 70.32$, $p < 0.001$).

3.6 Visuospatial abilities

Eight investigations reported data for 373 participants (BPD: $n = 163$, control: $n = 210$) for neuropsychological tests of visuospatial abilities. The data for the Rey-Osterreith Complex Figure test were excluded for one study (Judd and Ruff, 1993) because of a ceiling effect in which the authors reported that 92% of the normal group compared with 20% of the BPD group achieved perfect or near perfect scores. The skewed distribution of scores within this study on this test measure appeared to misrepresent the effect size due to significant heteroscedasticity (see Grissom and Kim, 2001). The mean weighted effect size was -0.59 ($SE = 0.11$) with effect sizes ranging from -1.87 to $+0.08$. The effect size differs significantly from zero ($z = -5.16$, $p < 0.001$) and the sample of effect sizes is heterogeneous ($Q = 52.09$, $p < 0.001$).

4. Discussion

The results of this meta-analysis revealed a significant difference between BPD and healthy comparison groups across multiple neuropsychological domains. BPD patients generally performed more poorly than healthy comparison groups on global dimensions of attention, cognitive flexibility, learning and memory, planning, speeded processing, and visuospatial abilities. The effect sizes were primarily in the medium to

large range, with the smallest effect size observed for the cognitive flexibility domain and the largest for planning.

Despite heterogeneity within the data, the significant effect sizes suggest that BPD patients demonstrate deficits across a wide range of neurocognitive domains. Significant effect sizes in the domains of attention, cognitive flexibility, and speeded processing suggest potential frontal lobe dysfunction in persons with BPD (Mitchell et al., 2004; Monchi et al., 2001; Stuss et al., 2001). This is consistent with data demonstrating significant correlations between neuropsychological measures of frontal lobe function and BPD symptomatology in a sample of normal young adults (Ruocco and Trobst, 2003) and a head-injured sample (Ruocco and Swirsky-Sacchetti, 2005). A large effect size for the planning domain and medium-to-large effect size for the visuospatial domain also suggests frontal and possible parietal lobe pathology (Fincham et al., 2002; Jacobs and Anderson, 2002; Aleman et al., 2002; Zago and Tzourio-Mazoyer, 2002; Newman et al., 2003), while deficits in learning and memory implicate potential dysfunction of frontotemporal regions (Kelley et al., 1998; Johnson et al., 2001).

These findings of global deficits in neuropsychological functioning in BPD provide support for the Jacksonian biopsychosocial model of BPD (Mearns et al., 1999). The Jacksonian model asserts that many of the symptomatic features of BPD, including dysregulated affect, identity disturbance, somatization, and dissociation, are caused by disrupted connections between the prefrontal cortex and other brain regions subserving higher cognitive functions. The model predicts a global neurocognitive impairment rather than discrete localized deficits, postulating that a cascade of neuropsychological impairments is present in BPD, perhaps the result of experience-mediated disruption of

prefrontal circuitry. The results of the meta-analysis are on the whole consistent with the Jacksonian model, as BPD patients appear to demonstrate widespread neuropsychological deficits linked largely to functioning of the frontal lobes.

Drawing conclusions about specific loci of brain pathology based on averaged scores on multiple neuropsychological measures across large groups of participants is difficult given the broad range of neuropsychological deficits revealed in this analysis. However, some merit might be given to the localizing value of certain test measures. For instance, while further exploration of the domain of learning and memory revealed significant decrements in both verbal and nonverbal memory performance for BPD participants relative to healthy comparison groups, a much larger difference was found for nonverbal rather than verbal domains of memory. These findings suggest a frontotemporal dysfunction that is more strongly lateralized to the right hemisphere, which is consistent with data implicating a specific dysfunction of the right hemisphere in BPD (Dinn et al., 2004; Niederhofer, 2004). A more comprehensive understanding of the spatial specificity of brain pathology in BPD would be gained from investigations incorporating both neuropsychological and neuroimaging methods of assessment.

Emerging functional neuroimaging studies employing both cognitive and emotional paradigms generally coincide with the present findings, implicating potential dysfunction of frontal and temporal brain regions in BPD (Tebartz van Elst et al., 2003; Driessen et al., 2004; Schmahl et al., 2004; Vollm et al., 2004), as well as other neuroanatomical sites not readily evaluated by traditional neuropsychological testing, such as the amygdala (Donegan et al., 2003). While structural brain imaging investigations provide more evidence for left hemisphere abnormalities in BPD,

functional neuroimaging studies reveal a more widespread pattern of differences between BPD participants and healthy controls (for a review, see Ruocco, in press). The finding of greater nonverbal compared with verbal memory deficits in the present study might be further examined through functional neuroimaging studies of verbal and nonverbal memory encoding and retrieval processes in BPD in order to identify those specific mechanisms that might be impaired in BPD participants across both memory domains.

Although further analyses within the domain of learning and memory were useful for examining the lateralization of memory deficits in BPD, considerable heterogeneity of effect sizes was evident within many of the neuropsychological domains examined, with the only homogeneous samples of effect sizes observed for the speeded processing and learning and memory domains. A number of factors might contribute to the heterogeneity of effect sizes, with the most prominent source being the wide array of tests used across studies to assess particular domains of function. Within the attention domain, for instance, a large number of diverse cognitive measures were utilized across studies to assess the construct of attention, with many different measures used to assess several facets of this construct even within a single study (e.g., Posner et al., 2002). Further heterogeneity is introduced by the variety of diagnostic systems utilized across studies to arrive at the BPD diagnosis. Similarly, the methods and instruments utilized to assess BPD psychopathology (e.g., self-report inventories, structured and unstructured interviews) differed from one study to another, likely contributing heterogeneity to the sample of effect sizes and resulting in an incorporation of diverse conceptualizations of BPD within the meta-analysis (e.g., DSM–III, DSM–IV, and Millon’s theory). Whether patient groups were medicated or participating in other forms of treatment at the time of

study further escalates the heterogeneity of patient samples. The relatively small number of studies included in the meta-analysis, however, precluded any meaningful exploration of these potential moderating variables. Thus, the results of the present study must be tempered with some consideration of these limitations.

A number of factors also limit the external validity of the current findings, particularly with regard to the characteristics of the samples included in the meta-analysis. For instance, although there is some variability in gender compositions across the studies examined, the amalgamated sample is predominantly female. Therefore, caution must be exercised in extending these results to males with BPD. Additionally, the inclusion of samples of BPD participants reported as being diagnosed with other co-occurring psychiatric conditions not only promotes heterogeneity of effect sizes but also complicates the degree to which the results of the meta-analysis can be extended to BPD populations with other concurrent Axis I and Axis II disorders. Recent research indeed suggests differential neurophysiological findings with respect to histories of post-traumatic stress disorder and abuse in individuals with BPD (e.g., Driessen et al., 2004; Schmahl, et al., 2004). To confront this challenge, future investigations ought to utilize appropriate Axis I and Axis II patient comparison groups to achieve greater specificity of the observed deficits in BPD and examine systematically the differential impact of co-occurring psychiatric conditions on neuropsychological functioning in BPD.

Additionally, the findings of the present study should be interpreted with caution given statistical limitations inherent in meta-analytic strategies involving Cohen's d statistic. In particular, heteroscedasticity of neuropsychological data for BPD and healthy comparison groups may overestimate Cohen's d as well as the Q statistic used to

calculate heterogeneity of effect sizes (Grissom and Kim, 2001). The distribution of variance ratios between BPD and healthy groups was examined to determine the extent to which heteroscedasticity might have impacted the present findings. It was revealed that the VRs of the data included in the meta-analysis generally fell within acceptable ranges, with extreme cases of heteroscedasticity removed from the analyses.

While the present study found neuropsychological testing to be sensitive to BPD deficits in multiple domains of cognitive functioning, the neuropsychological findings generally appear to possess limited clinical utility. As suggested by Zakzanis (2001), effect sizes greater than 3.0 – equating to approximately 5% test measure overlap between patient and control samples – may be a useful criterion for evaluating the sensitivity of neuropsychological tasks and for determining specific test markers of neurocognitive disorders. The effect sizes observed in this analysis across all six neuropsychological domains ranged from -0.29 to -1.43, with the largest effect size still demonstrating 32% overlap in test measures between BPD and healthy comparison groups. Thus, according to this criterion of clinical utility, neuropsychological testing appears to be inadequate in specifying discrete neurocognitive test markers characteristic of BPD.

This is not to say that these findings have no direct clinical implications, however preliminary. Indeed, clinicians should be cognizant of the possibility that clients with BPD may be at a disadvantage in terms of attention, learning, and memory, cognitive skills that might certainly impact their ability to communicate effectively and engage successfully in treatment. Clinicians might also consider whether giving these patients medications with cognitive side effects is warranted, and whether other side effects might

be less harmful to the client. While the impact of medications, as well as other treatment modalities, on the findings of the present meta-analysis is unclear, contemplation of this potentially consequential issue is justified. As well, clinicians should be attentive to the BPD client's poor judgment and the extent to which neuropsychological symptoms might play a role in placing a client at greater risk for suicide.

In light of the results of the present meta-analysis, the seeming inconsistencies observed across past neuropsychological investigations of BPD appear to be artificial. Based on the effect sizes obtained across the six neurocognitive domains, it is apparent that most prior investigations lacked sufficient statistical power to detect potential differences between BPD and healthy comparison groups on common neuropsychological tasks. For instance, although autobiographical memory distortions and recollections of trauma and abuse are common among patients with BPD (Jones et al., 1999), individual investigations examining performance on neuropsychological measures of memory were largely inconsistent in detecting differences between BPD participants and healthy control groups. Based on the results of the present meta-analysis, however, it is apparent that the total sample size required to detect this difference between groups on general tests of learning and memory with a power of 0.80, if the effect does indeed exist, is approximately 90 participants (45 BPD and 45 healthy control). Interestingly, the mean sample size of past investigations is less than half of that which would be necessary to detect the effect with sufficient statistical power. An alternative procedure that might generate more consistent data within and across neuropsychological investigations of BPD would involve aggregating multiple measures of particular neuropsychological constructs and performing between-group analyses

based on these amalgamated measures (Haase and McCaffrey, 2004). This method proved useful in a recent neuropsychological investigation of BPD conducted by Monarch et al. (2004), which found significant deficits for BPD patients in seven of nine cognitive domains tested.

In this regard, a multimode-multimethod approach (Campbell and Fiske, 1959) to examining the neuropsychological features of BPD seems essential. Such an approach would advocate a meaningful integration of multiple measures of neuropsychological and personality assessment, as well as novel functional neuroimaging paradigms, providing for more intricate testing of neurobehavioral hypotheses of BPD. For instance, while multivariate (e.g., Trull et al., 2003) and neurocognitive studies of BPD have yielded fruitful findings in their respective modes of inquiry, the two approaches have rarely been integrated despite preliminary evidence that normal personality dimensions and frontal lobe function might contribute differentially and interactively to various forms of personality disorder symptomatology (Ruocco and Trobst, 2003, 2004). As well, ecologically-valid functional neuroimaging methods that allow for the investigation of brain-behavior relationships in interpersonal circumstances hold particular promise for the study of personality disorders. Specifically, functional near infrared spectroscopy (fNIRS) would be particularly useful for the examination of interpersonal behavior in BPD (see Irani et al., in press), a domain of functioning particularly relevant to personality disorders (Wiggins and Trobst, 1999). Undoubtedly, emerging functional neuroimaging technologies will play a critical role in capturing those neurobehavioral features of BPD and other personality disorders which might not be adequately assessed using other such technologies. The novel application of neuropsychological and

ecologically-valid functional neuroimaging methods rooted in personality theory will prove invaluable for elucidating the neural underpinnings of this certainly complex and multi-faceted neurobehavioral disorder.

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Table 1.

Descriptive data for participants across studies included in the meta-analysis.

Study	<i>N</i> BPD	<i>N</i> Control	Mean Age (years) BPD	Mean Age (years) Control	Gender (% BPD female)	Measures	Type of BPD Diagnosis
O'Leary et al. (1991)	16	16	29.4	27.5	81	SIDP	DSM-III
Judd and Ruff (1993)	25	25	33.0	33.0	80	DIB	DSM-III
Swirsky-Sacchetti et al. (1993)	10	10	30.3	29.0	100	DIB; SCID-II; MCMI-III	DSM-III-R
Bazanis et al. (2002)	42	42	30.6	33.3	59	SCID-II	DSM-III-R
Harris et al. (2002)	15	15	31.1	29.4	67	unknown	DSM-IV
Posner et al. (2002)	39	30	30.0	22.0	97	IPDE	DSM-IV
Kunert et al. (2003)	23	23	29.9	38.3	87	IPDE	ICD-10
Dinn et al. (2004)	9	9	30.1	27.2	100	unknown	DSM-IV
Stevens et al. (2004)	22	25	31.9	30.5	100	DIB	DSM-IV
Lezenweger et al. (2004)	24	68	31.9	29.24	100	IPDE	DSM-IV

DIB=Diagnostic Interview for Borderlines (Gunderson et al., 1981); SCID-II=Structured Clinical Interview for DSM-III-R Personality Disorder (Spitzer et al., 1990); SIDP= Structured Interview for the DSM-III Personality Disorders (Stangl et al., 1985); IPDE=International Personality Disorder Examination (Loranger, 1996, 1999); MCMI-III=Millon Multiaxial Clinical Inventory-III (Millon, 1997); DIB-R=Diagnostic Interview for Borderlines-Revised (Zanarini et al., 1989)

Table 2. Individual effect sizes included in the meta-analytic review.

Study	Test	Domain	Effect size (d)
O'Leary et al. (1991)	WAIS-R - Digit Span	attention	-.2306
	WAIS-R - Arithmetic	attention	-.2566
	WAIS-R - Block Design	visuospatial	-.0289
	WAIS-R - Object Assembly	visuospatial	-.2566
	WAIS-R - Digit Symbol	speeded processing	-1.2363
	WCST - Perseverative Errors	cognitive flexibility	-.3309
	WCST - Nonperseverative Errors	cognitive flexibility	-.2837
	WMS - Logical Memory	learning/memory	-.7460
	WMS - Delayed Logical Memory	learning/memory	-.7813
	WMS - Visual Reproductions	learning/memory	-.5886
	WMS - Verbal Paired-Associate Learning	learning/memory	-.2282
	WMS - Delayed Associate Learning	learning/memory	.0481
	WMS - Digits Forward	attention	-.4233
	WMS - Digits Backward	attention	-.5682
	Rey-Osterrieth - Copy Organization	visuospatial	-.5581
	Rey-Osterrieth - Recall Accuracy	learning/memory	-1.0907
	Rey-Osterrieth - Delayed Recall	learning/memory	-.9539
	Corsi Blocks - Forward	attention	-.8448
	Corsi Blocks - Backward	attention	-.6338
	Verbal Incidental Learning Test - Free Recall	learning/memory	-.0704
Judd and Ruff (1993)	Ruff Figural Fluency - Mean Designs	speeded processing	-1.1687
	Selective Reminding Test - Total Recall	learning/memory	-.6367
	Selective Reminding Test - Sum CLTR	learning/memory	-.5489
	Selective Reminding Test - Retrieval: Storage	learning/memory	-.5440
	Selective Reminding Test - 60 min. Recall	learning/memory	-.1342
	WAIS-R - Block Design	visuospatial	.0820
	WAIS-R - Digit Symbol	speeded processing	-.9125
	WAIS-R - Digit Span	attention	.1390
	Controlled Oral Word Association	cognitive flexibility	-.1260
	Stroop - Time	speeded processing	-.1573
Stroop - Error	attention	-.3105	
Swirsky-Sacchetti et al. (1993)	WAIS-R - Arithmetic	attention	-.7696
	WAIS-R - Digit Span	attention	-.6062
	WAIS-R - Block Design	visuospatial	-.7993
	WAIS-R - Digit Symbol	speeded processing	-.3525
	Symbol-Digit - Written	speeded processing	-.1843
	Symbol-Digit - Oral	speeded processing	-.0494
	TMT A	speeded processing	-.6006
	TMT B	cognitive flexibility	-.3564
	WMS - Logical Memory	learning/memory	-1.9244
	WMS - Delayed Logical Memory	learning/memory	-.0995
	WMS - Figural Memory	learning/memory	-1.0883
	WMS - Figural Memory Delayed Recall	learning/memory	-.8549
	Rey-Osterrieth - Recall Accuracy	learning/memory	-.6499
	Controlled Oral Word Association	cognitive flexibility	-.3101
	Rey-Osterrieth - Copy Organization	visuospatial	-.7917
	Raven's Matrices	visuospatial	-.5159
	Hooper Visual Organization Test	visuospatial	-.8233
	WCST - No. Errors	cognitive flexibility	-.5031
	Stroop - Word	speeded processing	-.4982
	Stroop - Color	speeded processing	-.5207
Stroop - Interference	attention	-.6768	

Bazanis et al. (2002)	Tower of London - Mean moves	planning	-4.7265
	Pattern Recognition	visuospatial	-2.0745
	Spatial Recognition	visuospatial	-1.6125
Harris et al. (2002)	TOL - Mean Time 1st Move	planning	-3.1333
	Rey-Osterrieth - Copy Accuracy	visuospatial	-1.6182
	Rey-Osterrieth - 1-min Delay	learning/memory	-1.1816
Posner et al. (2002)	Rey-Osterrieth - 30-min Delay	learning/memory	-2.0116
	Attentional Network Test - Alerting	attention	-.9366
	Attentional Network Test - Orienting	attention	-.4758
Kunert et al. (2003)	Attentional Network Test - Conflict	attention	-3.8196
	Tower of Hanoi - Trials	planning	.0614
	Stroop - Word	speeded processing	-.8131
Dinn et al. (2004)	Stroop - Color	speeded processing	-.6197
	Stroop - Interference	attention	-.6651
	Alertness - Mean Reaction Time	attention	-.2400
	Go/no-go - Mean Reaction Time	attention	.2397
	Divided Attentioness - Mean Reaction Time	attention	.0023
	Visual Scanning - Mean Reaction Time	attention	.0427
	Flexibility - Correct Reactions	cognitive flexibility	.1125
	Working Memory - Number Correct	working memory	-.4281
	Selective Reminding Test - Total Recall	learning/memory	-.1635
	Object Alternation Test	cognitive flexibility	-.6098
	Word Fluency Test	cognitive flexibility	-1.1835
	TMT A	speeded processing	-1.6187
	TMT B	cognitive flexibility	-1.3171
Stevens et al. (2004)	Rey-Osterrieth - Copy Accuracy	visuospatial	-1.5351
	Rey-Osterrieth - Recall Accuracy	learning/memory	-2.3514
	WMS - Logical Memory	learning/memory	-1.4029
	Digit Span - Forward	attention	-1.2242
	Digit Span - Backward	attention	-.8003
	WMS - Verbal Paired-Associate Learning	learning/memory	-.5755
	Porteus Maze Task	visuospatial	-2.2029
	Divergent Thinking Task	cognitive flexibility	-1.1414
	Digit Symbol	speeded processing	-.1545
	Mental Rotation (2-D)	visuospatial	-.2360
Lezenweger et al. (2004)	Mental Rotation (3-D)	visuospatial	-.2459
	Spatial Delayed Response Task	visuospatial	.1594
	Continuous Performance Test	attention	-.0122
	WCST - Errors (%)	cognitive flexibility	-.4650

Note: WAIS-R=Wechsler Adult Intelligence Scale-Revised; WMS=Wechsler Memory Scale; WCST=Wisconsin Card Sorting Test; TMT=Trail Making Test