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Borderline personality disorder and neuropsychological measures of executive function: A systematic review

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ABSTRACT

The notion that neurocognitive deficits may be core to the development of borderline personality disorder (BPD) has received considerable attention in recent years, with growing evidence pointing to cognitive deficits in executive function (EF). A relationship between EF and BPD has long been suggested by evidence of high comorbidity between BPD and disorders characterized by poor EF (e.g. attention-deficit/hyperactivity disorder); however, despite a marked increase in studies of EF and BPD in recent years, the precise nature of this relationship remains unclear. We provide a systematic review of this emerging evidence base, with respect to (1) studies of participants diagnosed with BPD in which EF has been indexed in isolation from broader cognitive processes; (2) the specific domains of EF that have been most robustly associated with BPD; and (3) whether deficits in EF are uniquely associated with BPD, independent of comorbid psychopathology. Key directions for future research are discussed with respect to strategies for measuring EF and the need for research designs that control for phenotypic overlap between BPD and related forms of psychopathology. Copyright © 2015 John Wiley & Sons, Ltd

Borderline personality disorder (BPD) is characterized by turbulent, angry, anxious and depressive emotional states, emotion dysregulation, interpersonal dysfunction, identity disturbance, impulsivity, parasuicidal and suicidal tendencies (Fertuck, Lenzenweger, Clarkin, Hoermann, & Stanley, 2006; Lenzenweger & Cicchetti, 2005). Current developmental models of BPD indicate that phenotypic features of BPD are potentiated through risk processes that unfold across adolescence and implicate complex transactions between a child's genetic vulnerability and a harsh, invalidating family environment (Beauchaine, Klein, Crowell, Derbidge & Gatzke-Kopp, 2009; Crowell, Beauchaine & Linehan, 2009). It has been proposed that BPD may be best conceptualized as a disorder of emotion dysregulation, wherein emotional responses are inflexible, rigid and either over-arousing or under-arousing (Putnam & Silk, 2005). Considerable evidence also points to impulsivity as one of the earliest and most core components out of which subsequent dysfunction emerges (Berlin, Rolls, & Iversen, 2005; Crowell et al., 2009).

The notion that neurocognitive deficits play an important role in the development of BPD has received growing attention in recent years, and a

major focus has been placed on executive function (EF) (Beauchaine et al., 2009; Judd, 2005). This focus has been justified by evidence of high comorbidity between BPD and forms of psychopathology that are characterized by deficits in EF, in particular, attention-deficit/hyperactivity disorder (ADHD) (Matthies & Philipsen, 2014; Skodol et al., 2002). Moreover, a meta-analysis by Ruocco (2005) found that although individuals with BPD performed poorly compared with controls across a broad range of neurocognitive domains including attention, cognitive flexibility, learning and memory, planning, speeded processing and visuospatial abilities, the largest effect sizes were seen for planning—a key EF domain (Fincham, Carter, Van Veen, Stenger & Anderson, 2002; Lezak, 1995). Interestingly, the smallest effect size was observed for cognitive flexibility, another key EF domain. Ruocco's (2005) meta-analysis therefore not only implicates EF deficits in BPD but also suggests that these deficits may be somewhat selective with respect to specific domains of EF.

A growing number of studies have tested associations between EF and BPD using a range of research designs and methods, and results have often been mixed with regard to the magnitude of associations and respective domains of EF. Several studies have found EF deficits among BPD participants and intact performance in other neuropsychological domains. Bazanis et al. (2002) found deficits in EF (via a task of planning) but no significant deficits on tasks of visual memory. Similarly, Dinn et al. (2004) found BPD participants to be impaired on EF tasks (planning and cognitive flexibility) but not on tests of neurocognitive domains including verbal working memory or attention. In contrast, O'Leary, Browers, Gardner and Cowdry (1991) found BPD participants to be impaired on tests of memory and visual discrimination but not on any measure of EF. Other research has demonstrated minimal or no impairment for BPD participants compared with healthy controls across all neuropsychological domains indexed (Moritz et al., 2011; Sprock, Rader, Kendall & Yoder, 2000). These divergent results may in part stem from the different neuropsychological tests used to measure EF and broader cognitive domains, highlighting the need to consider measurement issues when evaluating this evidence base.

A significant problem in investigating the association between BPD and EF is that BPD is a highly heterogeneous disorder (Widiger, 1992). Likewise, high rates of comorbid disorders occur among those with BPD (Grant et al., 2008). The issue of comorbidity serves as a significant hindrance when investigating the unique contributions of EF to BPD. Given the forms of psychopathology with which BPD is often comorbid, it is reasonable to speculate that various EF deficits may represent 'liability constructs' that confer risk for both BPD and disorders such as ADHD (Levy, Hawes, & Johns, in press; Travers & King, 2005). However, important questions remain as to whether EF deficits merely amplify impairment among individuals with BPD by conferring risk for various comorbid disorders such as this or whether they contribute to BPD independently. Such an issue has important implications for the design of targeted interventions for individuals with BPD, which have begun to incorporate cognitive remediation in recent years (e.g. Reeder, Stevens, Liddement, & Huddy, 2014).

Conceptualizations of executive function

Executive function is commonly conceptualized in terms of cognitive processes that allow for future, goal-directed behaviours, consisting processes of planning, organization, inhibitory control, cognitive flexibility and problem solving (Anderson, 2011; Carlson, Zelazo, & Faja, 2013; Diamond, 2013). In recent decades, there has been increasing interest in investigating the specific components or domains that comprise EF and the methods by which these domains may be indexed in isolation from one another (Royall et al., 2002; Unterrainer et al., 2004). One of the most influential models of EF is that of Lezak (1995), which

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identifies four broad domains of EF: volition (the ability to generate goals and form intentions), planning (the ability to generate a sequence of steps to achieve goals), purposive action (the ability to carry out plan and modify as needed, e.g. cognitive flexibility) and effective performance (the ability to monitor and self-correct as necessary, e.g. cognitive inhibition) (Anderson, 2011). Lezak's framework has informed key advances in models of EF and psychopathology, providing researchers with a basis for examining EF as distinct from broader cognitive processes. For example, in order to investigate the unique associations between EF and antisocial behaviour, Morgan and Lilienfeld (2000) conducted a meta-analysis focused only on studies in which EF was measured using tests that incorporate at least one of the domains identified by Lezak (1995) and that have been shown to index frontal lobe function.

Aims of the current review

Ruocco's (2005) meta-analysis highlighted that deficits in planning—a key domain of EF—appeared to be more strongly associated with risk for BPD than deficits in any other neurocognitive variable. In line with this, the major aim of this paper was to perform a systematic review of available evidence regarding associations between BPD and EF, as distinct from the broader neurocognitive profile of BPD. To the authors' best knowledge, this association has not previously been subject to a systematic examination of this kind. Furthermore, we aimed to investigate the association between EF and BPD with respect to three specific questions. First, as research into the neurocognitive correlates of BPD has examined a broad range of processes related to EF, it is necessary to differentiate studies that index core domains of EF from those concerned with broader overlapping processes. For this purpose, we examined the extent to which BPD is associated with individual differences in EF as operationalized according to the neuropsychological measurement criteria employed by Morgan and Lilienfeld (2000) (as described in the Inclusion Criteria Section), in order to investigate EF as measured by the most valid neuropsychological tests available. Second, in line with current conceptualizations of EF that emphasize overlapping yet dissociable domains, we aimed to identify which, if any, domain(s) of EF is most strongly implicated in BPD. Finally, we examined the availability of evidence pertaining to the assumption that BPD is uniquely associated with deficits in EF, independent of comorbid disorders that may also be associated with EF deficits.

Method

Systematic search strategy

A comprehensive literature search was conducted among published articles indexed in the following databases: PsycInfo, Medline and Web of Science. Key words included the following: ('Borderline Personality Disorder' OR 'Borderline' OR 'Borderline States' OR 'Borderline Features') AND ('Executive Function' OR 'Neurocognition' OR 'Neuropsychology' OR 'Planning' OR 'Cognitive' OR 'Cognitive Control' OR 'Cognitive Processes' OR 'Cognitive Flexibility' OR 'Set Shifting' OR 'Inhibition' OR 'Problem Solving' OR 'Decision Making'). Results were limited to English language, peer-reviewed articles. Reference lists of included studies were further examined for any potentially relevant studies.

Inclusion criteria

The following criteria were applied with respect to the identification of relevant studies. First, studies were required to involve participants that met diagnostic criteria for BPD as set forth in DSM-IV or DSM-5; participants drawn from a non-clinical population scoring highly on BPD measures were not included. Second, it was required that studies employ neuropsychological measures that have been established as indexing EF, according to the criteria applied by Morgan and Lilienfeld (2000), that is, the test (1) assesses one of the following

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cognitive domains: volition, planning, purposive action (e.g. cognitive flexibility) and effective performance (e.g. cognitive inhibition); (2) has been found in several studies to differentiate patients with frontal lobe lesions from patients with brain damage or lesions in other areas; and/or (3) has been shown in brain imaging research to preferentially activate the frontal cortex. Tests meeting these criteria include the Porteus Maze Test (Q score) (planning), the Stroop Interference Test (cognitive inhibition), the Trail Making Test (Part B) (cognitive flexibility), the Wisconsin Card Sorting Test (WCST; perseverative error score) (cognitive flexibility), the Verbal Fluency Tests (cognitive inhibition) and the Towers Tests—Tower of London and Tower of Hanoi (planning). Third, it was required that studies report data on separate measures of EF; any study that combined different measures into a composite score was excluded.

Results

Study characteristics

An initial search identified 52 articles in which data on the relationship between EF and BPD were reported, 12 of which met the inclusion criteria pertaining to our research questions (see Figure 1 for flow diagram). The full text of approximately one-third of potentially relevant articles was coded by a second rater based on the specified inclusion criteria. Cohen's kappa indicated 100% interrater agreement (K = 1). Key methodological characteristics of these studies are presented in Table 1. The majority of these studies comprised

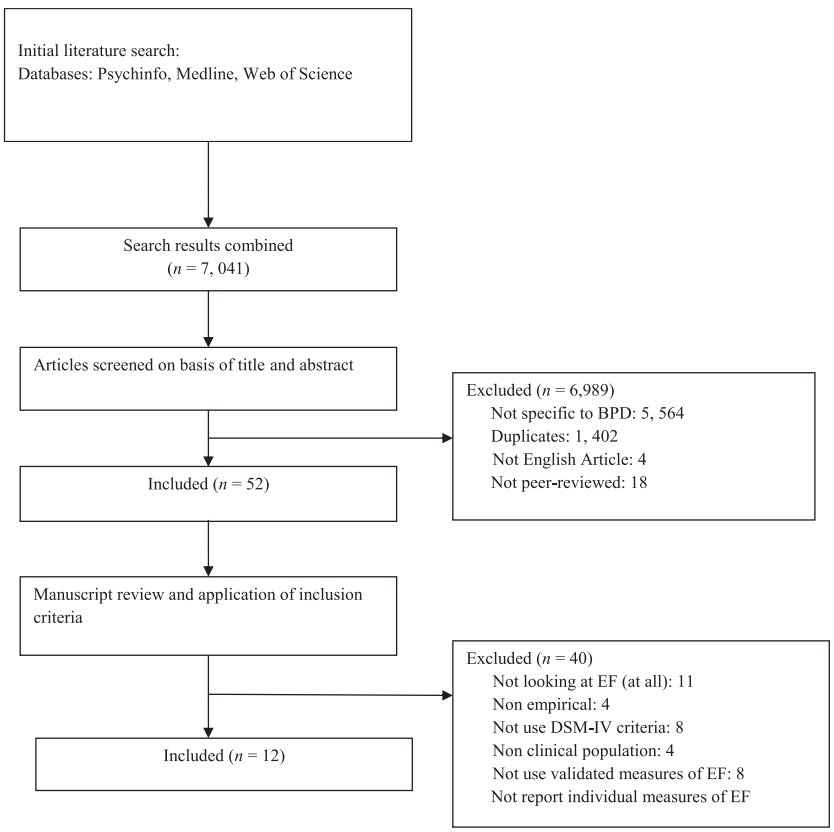

Figure 1: Flow diagram of review selection

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Table 1: Characteristics of identified studies

Study	*n*	Sample	Mean age BPD	Mean age control	Gender % (female)	Measure of BPD
Beblo et al. (2006)	42	BPD (22) Control (22)	32 (7.9)	32 (7.9)	100	SCID-II
Black et al. (2009)	45	BPD (25) Control (20)	34.6 (11.0)	38.0 (12.3)	80	SCID-II
Bustamante et al. (2009)	85	BPD (51) Control (34)	31.3	31.3	71.8	IPDE
Dinn et al. (2004)	18	BPD (9) Control (9)	30.1 (8.7)	27.2 (8.7)	100	Clinician diagnosis
Fertuck et al. (2006)	55	MDD (33) MDD–BPD (22)	33.8 (10.0)	50.0 (14.2)	71	SCID-II
Gvirts et al (2012)	98	BPD (27) Control (29) BPD parent (20)	29.6 (9.49)	31.3 (8.90)	82	SCID-II
Kunert et al (2003)	43	BPD (23) Control (23)	29.9 (8.7)	38.3 (12.9)	76	IPDE
Lampe et al. (2007)	83	BPD (21) ADHD (22) BPD + ADHD (20) Control (20)	24.6 (6.2)	ADHD 29.95 (8.2) ADHD + BPD 26.50 (7.0) Control 28.7 (6.9)	71	IPDE
LeGris et al. (2012)	83	BPD (42) Control (41)	32.2 (10.5)	31.2 (9.0)	100	MSI-BPD
Lenzenweger et al. (2004)	92	BPD (24) Control (68)	31.92 (9.15)	29.24 (6.67)	100	IPDE
Moritz et al. (2011)	40	BPD (20) Control (20)	29.70 (6.24)	33.35 (12.82)	85	SCID-II
Travers and King (2005)	80	BPD organic injury or disorder (30) BPD no organic factors (50)	Organic 30.8 (8.0) Non-organic 30.0 (8.1)	N/A	30	SCID-II

Note: BPD, borderline personality disorder; SCID-II, Structured Clinical Interviews for DSM-IV; IPDE, International Personality Disorder Examination; MDD, major depressive disorder; MDD–BPD, major depressive disorder–borderline personality disorder; ADHD, attention-deficit/hyperactivity disorder; MSI-BPD, McLean Screening Instrument-borderline personality disorder; N/A, not applicable.

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mixed-gender samples; however, all but one comprised a majority (>70%) of female participants. Four studies employed women only samples. Sample size across these studies ranged from $n = 18$ to $n = 98$, with an overall mean sample size of $n = 64$. In 11 of these studies, the clinical status of participants was determined using one or more standardized diagnostic measures, whereas one study relied on clinicians specializing in the treatment of BPD to confirm diagnosis. Of the 12 studies identified, 10 included a control group of healthy participants, who were generally matched for age and gender to the clinical participants. The two studies that did not employ healthy controls used other clinical comparison groups (major depressive disorder (MDD) and organic factors—BPD). Studies varied significantly with respect to exclusion criteria regarding comorbid psychopathology.

Outcomes

The key findings of the 12 identified studies, with regard to associations between EF and BPD, are presented in Table 2. Five of the identified studies employed either the Tower of London or Tower of Hanoi and the Porteus Maze Test, which are understood to predominately measure the domain of planning (Kendall & Hollon, 1981; Unterrainer et al., 2004). Seven of the studies employed the Trails (Part B) or WCST, which is considered to predominately measure cognitive flexibility/set shifting (Bechtold Kortte, Horner & Windham, 2002; Crone, Ridderinkhof, Worm, Somsen & Van Der Molen, 2004). Seven studies utilized either the Stroop or verbal fluency tasks, which are understood to predominantly index cognitive inhibition/executive control (Goldstein et al., 2011; Westerhausen, Kompus & Hugdahl, 2011; Shao, Janse, Visser & Meyer, 2014). No identified tasks were found to unambiguously meet the criteria for the domain of volition, which is particularly a difficult construct to measure via neuropsychological tests. For studies that identified significant associations, effect sizes are reported in terms of Cohen's *d*. When not reported in the original studies, Cohen's *d* was calculated from other reported values. Negative effect sizes represent results for which BPD groups performed more poorly on the neuropsychological tests of EF relative to control groups.

Evidence of significant differences between participants with BPD and comparison groups was found in eight studies, the clear majority. Across these studies, the association between EF and BPD was generally found to be of a moderate-to-large effect size. In terms of specific domains of EF, the largest effect sizes were seen for that of planning ($d = -0.64$ to -2.31) followed by the domains of cognitive flexibility/set shifting ($d = -0.42$ to -1.70) and cognitive inhibition/executive control ($d = -0.53$ to -1.56). However, as discussed in more detail in the succeeding text, it was apparent that no single domain of EF demonstrated entirely consistent associations with BPD, with considerable variation seen among the findings for these respective domains.

Discussion

Is BPD characterized by executive function deficits?

Research into the neurocognitive underpinnings of BPD has grown considerably in recent decades and has associated the disorder with a range of deficits (Ruocco, 2005). Although this research has provided much evidence that self-regulatory dysfunction and impulsivity are core to BPD, questions regarding the specific cognitive domains in which self-regulatory deficits are located remain largely unresolved. Only a relatively small number of studies in the current literature meet with the neuropsychological criteria that have previously been used to draw conclusions regarding specific associations between EF and other forms of psychopathology (e.g. Morgan & Lilienfeld, 2000). We identified 12 studies in which measures that are understood to index EF domains independent of broader overlapping processes have been used to compare individuals with BPD with comparison groups. Among these studies, the majority (66%)

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Table 2: Results of identified studies

Study	Measure(s)	EF domain	Effect size (Cohen's *d*)
Beblo et al. (2006)	Trail making B (s)	Cognitive flexibility	−0.7*
	TOH four plates (moves)	Planning	ns
	TOH four plates (s)	Planning	−1.2*
	FAS	Cognitive inhibition	ns
	Animals	Cognitive inhibition	ns
Black et al. (2009)	Stroop	Cognitive inhibition	
	Colour naming		ns
	Word reading		ns
	Interference		−1.56*
	Trails B	Cognitive flexibility	ns
	Trails A		−0.72*
	WCST	Cognitive flexibility	
	Preservative error		ns
	Categories complete		−0.79*
	COWA		ns
	BDAE	Cognitive inhibition	−0.81*
		Cognitive inhibition	
Bustamante et al. (2009)	TOL	Planning	
	Total moves		ns
	Latency time		ns
	Execution time		ns
	Problem solving time		ns
Dinn et al. (2004)	FAS	Cognitive inhibition	ns
	Porteus maze	Planning	−2.31***
	Trail A (s)	Cognitive flexibility	−1.70*
	Trail B (s)		−1.38*
Fertuck et al. (2006)	Trail A	Cognitive flexibility	ns
	Trail B		ns
	Stroop	Cognitive inhibition	ns
	WCST	Cognitive flexibility	
	Categories		ns
	Errors		ns
	Perseverative errors		ns
	Non-perseverative errors		ns
	Failure to maintain set		ns
Gvirts et al (2012)	TOL	Planning	
	Two moves		−0.79***
	Three moves		−0.90**
	Four moves		−0.79**
	Five moves		−1.01***
	Problems solved in Minimum moves		−0.64*
Kunert et al (2003)	TOH (moves)	Planning	ns
	Stroop	Cognitive inhibition	
	Reading		−0.85*

(Continues)

Table 2: (continued)

Study	Measure(s)	EF domain	Effect size (Cohen's *d*)
	Colour naming		ns
	Interference		ns
	Errors		0.78*
Lampe et al. (2007)	Stroop	Cognitive inhibition	
	RT congruent		ns
	RT incongruent		ns
	RT congruent/incongruent		ns
Lenzenweger et al. (2004)	WCST	Cognitive flexibility	
	Category		ns
	Perseverative Response (%)		−0.48**
	Perseverative Errors (%)		−.44*
	Errors (%)		−.42*
LeGris et al. (2012)	Stroop	Cognitive inhibition	
	Colour RT		−0.53*
	Word RT		−0.56*
	Incongruent colour		−0.63**
	Word Interference		−0.54*
Moritz et al. (2011)	Trails	Cognitive flexibility	ns
	A		ns
	B		
Travers and King (2005)	Trails	Cognitive flexibility	
	A		—
	B		0.50*
	COWA	Cognitive inhibition	0.90***

Note: EF, executive function; TOH, Tower of Hanoi; FAS, Verbal Fluency Test; WCST, Wisconsin Card Sorting Test; COWA, Controlled Oral Word Association Test; BDAE, Boston Diagnostic Aphasia Examination Test; TOL, Tower of London; RT, reaction time; NS, not significant.
*<0.05;
**<0.01;
***<0.00.

reported findings that individuals with BPD exhibit distinct dysfunction in domains of EF relative to other populations.

Such evidence should be considered alongside findings from research that has utilized measures that were not included in the specific criteria applied here yet have nonetheless been conceptualized as measures of EF elsewhere. For example, the Iowa Gambling Task is a measure of problem solving and decision-making processes that are often conceptualized in terms of affective aspects of EF. Like the measures of EF specified in our criteria, impaired performance on this task has been demonstrated by individuals with BPD in a number of studies to date (Black et al., 2009; Haaland & Landro, 2007; LeGris, Links, van Reekum, Tannock & Toplak, 2012;Schuermann, Kathmann, Stiglmayr, Renneberg & Endrass, 2011). Although research in this area remains limited compared with that focused on the

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neurocognitive underpinnings of other personality-based forms of psychopathology (e.g. psychopathy), the preliminary evidence that is now available provides support for the notion that BPD is characterized by dysfunction in EF domains. Our review has also highlighted that questions concerning EF and comorbid conditions, such as ADHD, have been neglected in studies of BPD to date. Important questions remain regarding the specific domains of EF that most account for the dysfunction associated with BPD, and the extent to which this dysfunction is mediated by the contributions of EF to comorbid psychopathology.

Are specific domains of executive function key markers for BPD?

Despite growing evidence that EF deficits play a role in BPD, the specific domains of EF (e.g. planning, cognitive flexibility and cognitive inhibition) that may account for these contributions are far less apparent. Existing research indicates that EF deficits associated with BPD are not limited to any single domain of EF. Moreover, no single domain of EF has conclusively emerged as a more distinct marker for the disorder than another. The inconsistent results that have been reported for associations between BPD and specific domains of EF to date are all the more striking given the restricted range of neuropsychological measures examined here, suggesting that these findings are not simply an artefact of different studies reporting on different indices of EF.

Findings from the past decade have nonetheless provided growing evidence that deficits in the domain of planning may be particularly prominent among individuals with BPD. Three studies of BPD participants have found deficits in this domain to be associated with large effect sizes relative to other domains (Beblo et al., 2006; Dinn et al., 2004; Gvirts et al., 2012). This domain is understood to reflect higher-order processes of abstract thinking, temporal sequencing and reasoning (Kramer et al., 2014); and deficits in planning have also been implicated in ADHD (Willcutt, Doyle, Nigg, Faraone & Pennington, 2005), post-traumatic stress disorder and depression (Olff, Polak, Witteveen & Denys, 2014). At the same time, however, other research has reported no differences in planning performance between such participants with BPD and healthy controls (Kunert, Druecke, Sass & Herpertz, 2003). Furthermore, current data suggest that association between BPD and planning deficits may be more modest in magnitude than the large effects that were indicated at the time of Ruocco's (2005) review.

Findings regarding the domains of cognitive flexibility and cognitive inhibition have been similarly discrepant. Studies have found moderate effects for impairment in cognitive flexibility (Beblo et al., 2006, Lenzenweger, Clarkin, Fertuck & Kernberg, 2004), whereas others have reported no difference in this domain between participants with BPD and controls (Moritz et al., 2011). Effects associated with the domain of cognitive inhibition have likewise been mixed (Dinn et al., 2004; Black et al., 2009) and, in some research, have been found to all but disappear when controlling for level of education (LeGris et al., 2012). It should be noted that similar discrepancies can also be seen across studies of other cognitive processes that are often conceptualized in terms of EF, such as working memory (Diamond, 2013). Evidence that deficits in working memory represent potential markers for BPD has been reported in some studies (Baez et al., 2014; Hagenhoff et al., 2013), whereas others have found performance in this domain among individuals with BPD to be comparable with that of normative samples (Kunert et al., 2003; Le Gris et al., 2012).

Is BPD associated with poor EF independent of comorbid psychopathology?

Questions and controversies concerning comorbidity have driven major shifts in the conceptualization and classification of psychopathology in recent years (Cuthbert & Kozak, 2013; Insel

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et al., 2010), and much attention has been drawn to the high rates of comorbid psychopathology that often feature in presentations of BPD (Gremaud-Heitz et al., 2014; Tomko, Trull, Wood & Sher, 2014). Despite this, the extent to which neurocognitive deficits and other forms of dysfunction associated with BPD are explained by comorbid psychopathology has rarely been tested explicitly in studies of these deficits. Indeed, issues related to comorbid psychopathology have often been emphasized in researchers' discussions of study design limitations (Beblo et al., 2006; Bustamante et al., 2009; Kunert et al., 2003). Only a small number of studies (Black et al., 2009; Fertuck et al., 2006; Lenzenweger et al., 2004; Travers & King. 2005) have explicitly controlled for depression or anxiety when testing for EF correlates of BPD. Interestingly, Black et al. (2009) found significant differences in cognitive flexibility (WCST) to disappear when controlling for comorbid alcohol abuse and depression. It seems likely that divergent approaches to sampling participants with BPD and attending to overlapping symptom domains in terms of measurement and covariate analysis may account in part for the mixed findings in this field.

Among studies of BPD and EF to date, we identified four in which such deficits have been investigated using designs based on the grouping of participants in terms of comorbid conditions. Two studies (Lampe et al., 2007; Travers & King, 2005) found that individuals with BPD and a comorbid disorder (e.g. ADHD and organic brain injury) performed significantly worse on tasks of EF relative to individuals with BPD alone. Furthermore, individuals with pure BPD did not demonstrate impaired EF performance relative to healthy controls (Lampe et al., 2007; Travers & King, 2005). In addition to these findings, Fertuck et al. (2006) found no difference between individuals with MDD and individuals with comorbid BPD-MDD in terms of cognitive flexibility and inhibition, suggesting that these deficits in EF were explained largely by comorbid mood psychopathology in this sample. Interestingly, Bustamante et al. (2009) found that individuals with BPD were not significantly impaired in the domain of planning compared with controls, whereas such impairments were found to characterize a subgroup of participants with BPD, and the high degree of comorbid pathology within their sample may play a role in discordant findings between these two sub-groups. Such findings would appear to support the notion that deficits in EF may represent 'liability constructs' that confer risk for both BPD and the disorders with which it is commonly comorbid (Levy, Hawes, & Johns, in press; Travers & King, 2005). Conversely, evidence that these deficits are uniquely associated with BPD independent of overlapping psychopathology remains limited.

Limitations of the review

There are several limitations to the current review that are worth noting. Unlike Ruocco (2005), we conducted a systematic review rather than a meta-analysis to investigate the association between BPD and EF. A systematic review is a useful strategy in identifying and collating studies with conflicting findings in order to synthesize results when only a small number of studies are available in an emerging area (Akonberg, 2005). At the same time, however, a meta-analytic strategy provides a rigorous quantitative review of the existing literature and a more precise estimate of effect (Haidich, 2010). As such, it would be beneficial to apply the meta-analytic method to this relationship in the future, once a sufficient number of appropriate studies become available.

Next, we investigated the role of EF solely via performance on neuropsychological measures of EF. Neuroimaging research into the neurocognitive structure of BPD has indicated that individuals with BPD may demonstrate reduced volume in the amygdala and hippocampus (Driessen, Herrmann, Stahl et al., 2000; Nunes et al., 2009). In addition, a recent review by Krause-Utz et al. (2014) found evidence for structural and functional abnormalities in the anterior cingulated cortex, orbitofrontal cortex and dorsolateral prefrontal cortex. This review,

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and an earlier review of the neuroimaging literature, also highlighted that research in this area is likewise hindered by challenges related to disentangling comorbid psychopathology, in particular post-traumatic stress disorder and depression (Krauz-Utz et al., 2014; Lis, Greenfield, Henry, Guile & Dougherty, 2007). This further demonstrates the control of overlapping psychopathology in BPD research as a priority.

Also, given that we limited our review to English language papers, it is possible that this criterion resulted in the exclusion of some relevant research on this topic. Finally, we note that the definition of EF we employed was narrow in scope. This definition was employed, as in previous research reviews, in order to limit our review to findings from the most established neuropsychological tests of EF. However, we note that recent research has also indicated that 'hot' or emotionally affective cognitive tests may be more strongly implicated in BPD than 'cold' or emotionally neutral tasks (Sebastian et al., 2013; Domes et al., 2006), and we recognize that definition of EF does not fully encompass the capacities indexed in such hot tasks. We also acknowledge that while EF relies largely on the frontal cortex, EF is understood to be mediated by multiple brain regions, thereby accounting for the varied clinical presentation of executive dysfunction across a range of disorders (see Elliott, 2003, for review). Finally, we note that our review focused on the cognitive domain of EF exclusively, and we did not review the relationship between BPD and other cognitive domains.

Priorities for future research

At least three key directions for future research are indicated by the evidence that is presently available. First, current data point to the domain of planning as a promising focus for ongoing studies and a priority for multi-method research incorporating performance-based and neuroimaging methods. However, in order to better understand the role that deficits in this domain play in the risk processes that contribute to BPD, there is value to examining the contributions of these deficits relative to other EF domains, and alongside the broader cognitive processes that have likewise been associated with BPD. Such processes include deficits in visual memory and visuospatial abilities (Beblo et al., 2006) and non-verbal working memory (Dinn et al., 2004; Gvirts et al., 2012).

Second, there is a need for evidence that can clarify the extent to which deficits in EF are associated with BPD independent of the various forms of psychopathology with which it commonly co-occurs. It is understandable that researchers have often chosen not to sample only 'pure' presentations of the BPD, given the high rates of comorbidity that characterize the disorder at the population level. Because of the heterogeneity of BPD and comorbid disorders, one approach may be a research that investigates EF in relation to disorders on the externalizing spectrum. However, it is also apparent that studies of BPD and EF have rarely examined phenotypic overlap between BPD and mood or externalizing symptom domains in covariate analyses. We therefore recommend that future research designs should not exclude participants with comorbid psychological disorders but rather statistically control for these symptoms and the severity of these symptoms (particularly ADHD and depression), in order to clarify the extent to which EF deficits are uniquely associated with BPD.

Third, despite the emphasis on domains related to EF in current developmental models of BPD, little is known about the specific EF deficits that characterize children and adolescents at risk for developing BPD. There is considerable evidence to indicate that phenotypic features of BPD may often emerge during childhood and adolescence and are preceded by more common childhood disorders characterized by self-regulatory deficits (Chanen, Jovev, McCutcheon, Jackson & McGorry, 2008; Hawes, 2014). For example, in a recent longitudinal study by Stepp, Burke, Hipwell and Loeber (2012), increased symptoms of ADHD and ODD at age 8 years were found to

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predict BPD symptoms at age 14 years, as did rates of growth in these symptoms. There is at least indirect support for the presence of EF deficits in children with elevated features of BPD (Coolidge, Segal, Stewart & Ellett, 2000; Paris, Zelkowitz, Guzder, Joseph & Feldman, 1999); however, we identified no studies in which such features have been investigated in relation to specific domains of EF prior to adulthood. There is a clear need for research in which such domains are investigated as precursors to, and correlates of, features of BPD in childhood and adolescence. Such research stands to inform developmental models of BPD; and in turn, emerging strategies for early detection, treatment and prevention focused on attentional processes (Racer & Dishion, 2012).

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