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Neurocognitive Profile of Borderline Personality Disorder

PhD thesis

Mara Richman (Czégel)

Doctoral School of Mental Health Sciences

Semmelweis University

Supervisor: Zsolt Unoka, MD, PhD

Head of the Complex Examination Committee: Dániel Bereczki, DSc

Members of the Complex Examination Committee: Csaba Pléh, DSc

Róbert Bódizs, PhD

Budapest

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Abbreviations

AM: Autobiographical Memory
AMT: Autobiographical Memory Test
APA: American Psychiatric Association
BDI: Beck Depression Inventory
BPD: Borderline Personality Disorder
BPG: Borderline Personality Group
BPO: Borderline Personality Organization
CaR-FA-X: capture and rumination (CaR), functional avoidance (FA), and impaired executive control (X)
DIB: Diagnostic Interview for Borderlines
DSM: Diagnostic Statistical Manual
MD: Major Depression
MSD: Mental state decoding
PTSD: Posttraumatic stress disorder
RMET: Reading in the Mind of the Eyes Test

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1. Introduction

1.1 Borderline Personality Disorder

Borderline personality disorder (BPD) is present in approximately 2% of the general population (Coid, Yang, Tyrer, Roberts, & Ullrich, 2006) and characterized by impulsivity, non-suicidal self-harming behavior, a high level of suicidal lethality, labile affectivity, emotion dysregulation, disturbed relationships, and identity disturbances. BPD is present in approximately 2% to 5.9% of the general population (Coid et al., 2006). The ranges are much higher in clinical settings at 8% to 27% in outpatient psychiatric and 40% among inpatient psychiatric populations (Lenzenweger, 2008). The prevalence in incarcerated populations is 25% to 50% (Zimmerman, Rothschild, & Chelminski, 2005). BPD patients have the highest rate of lifetime suicide attempts compared to other mental health disorders at 75% and completed suicide (10%) among patients suffering from mental disorders (Gunderson & Ridolfi, 2001).

BPD is a part of the personality disorder classification called "cluster B" in the Diagnostic Statistical Manual (DSM; American Psychiatric Association [APA], 1952). This cluster, which also includes narcissistic personality disorder (i.e., extreme sense of self which interferes with everyday functioning), antisocial personality disorder (i.e., psychopathic and lack of empathy tendencies), and histrionic personality disorder (i.e., extreme dramatized lifestyle leading to impaired relationships), is marked by dramatic or extreme tendencies (APA, 2013). In addition, males are more likely to have co-occurring personality disorders than females (Kessler et al., 1997).

The first BPD patients were presented in medical literature as early as the first medical discoveries (Friedel, 2006); however, psychologist, Adolph Stern, created the first symptomatology checklist in 1938. Stern treated patients exhibiting two or three "borderline" mental illnesses (Stern, 1938). Over the course of treatment with these patients, Stern determined that these patients had similar psychotherapeutic treatment outcomes and deemed this group the Borderline Personality Group (BPG). Gunderson (1984) elaborated on Stern's original idea by organizing BPG into more concrete symptomatology called the Borderline Personality Organization (BPO; Gunderson, 1984) Gunderson defined it as: a) an inclination for intense, perturbed interpersonal relationships with manipulation and masked dependency, b) an unstable sense of self, with feelings of emptiness and abandonment anxiety, and c) fervent rage, apathy, and

impulsivity, usually with substance abuse and/or promiscuity (Gunderson, 1984). The current DSM-5 (APA, 2013) uses Stern and Gunderson's symptoms in diagnostic criteria. There are nine symptoms for borderline personality disorder. The DSM-IV lists them as " a) frantic efforts to avoid real or imagined abandonment. (b) identity disturbance: markedly and persistently unstable self-image or sense of self
(c) impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, Substance Abuse, reckless driving, binge eating). (d) recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior (e) affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days)
(f) chronic feelings of emptiness, (g) inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights) (h) transient, stress-related paranoid ideation or severe dissociative symptoms, and i) a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation." (APA, 2013). To date, the most prevalent symptomatology discussed throughout the literature are affect instability, impulsivity, suicidal behavior (i.e., gestures, threats, or self-mutilating behavior), disturbed interpersonal relationships, and identity disturbances.

1.2 Major Symptomatology

1.2.1 Affect instability

Affect instability in BPD is noted by quick mood swings and splitting between high and low emotions. Affective instability was defined in DSM-III-R as marked shifts from baseline mood to depression, irritability, or anxiety, usually lasting a few hours and only rarely more than a few days (APA, 1987). The DSM-IV modified the definition slightly emphasizing that affective instability should reflect reactivity to different types of moods (APA, 1994).

Affect instability in BPD may be associated with neurocognitive impairments (Peters, Upton, & Baer, 2013), epigenetics issues (Gratz & Gunderson, 2006), and traumatic stress (Preissler, Dziobek, Ritter, Heekeren, & Roepke, 2010), but there has been no conclusive evidence. Studies within BPD of this nature have not focused on the epidemiology of this symptomatology. Instead, studies have focused on the origin

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and course of emotion dysregulation in patients diagnosed with BPD (Zanarini & Frankenburg, 2001).

There are stark differences in the types of BPD affect instability that are worth noting. At one end of the spectrum is the view that interpersonal relationships and affect instability are closely and inevitably intertwined. This view is associated with attachment theory, which some proponents argue is "fundamentally about emotional experiences and their regulation" (Tidwell, Reis, & Shaver, 1996). In this view, insecure attachment styles are likely to be associated with affect instability (Levy, Meehan, Weber, Reynoso, & Clarkin, 2005). At the opposite end of the spectrum is the view that negative emotional states shape both social and non-social contexts uniformly. In this view, a predisposition to intense, negative affect and limited capacity for executive control of such affect is fundamental to affect instability. A third view proclaims that affect instability happens relative to the conflicting requests of dissimilar classes of social collaboration. It was found that affect instability was associated with increased impulsivity (Chapman, Leung, and Lynch, 2008).

1.2.2 Impulsivity

Impulsivity may refer to the inclination to act on a whim and without much consideration or reflection (Soloff, White, & Diwadkar, 2014). According to the Diagnostic Interview for Borderlines (DIB; Zanarini & Frankenberg, 2001), impulsivity in BPD may manifest as one or many of the following: reckless driving, substance abuse, compulsive buying, promiscuity, aggression, binge eating, or suicidal behavior. Substance abuse and reckless driving are the most reported types of impulsivity in BPD patients when interviewed on the DIB (Coffey, Schumacher, Baschnagel, Hawk, & Holloman, 2011). Over the past several years, increased impulsivity symptomatology was shown to be a predictor for suicide attempts in patients with BPD (Lis, Greenfield, Henry, Guilé & Dougherty, 2007).

1.2.3 Suicidal behavior.

Studies have found that behavior in patients with BPD is an impulsive response to severe emotional pain (Brodsky, Malone, Ellis, Dulit, & Mann, 1997; Linehan & Dimeff, 1997). Suicidal behavior includes death by intentional, suicidal, or self-injurious acts with or without intent to die (Linehan, 1993). BPD is one of two personality disorders that contain suicidal behavior in the diagnostic criteria; the second

is antisocial personality disorder, another cluster B personality disorder (Gratz & Gunderson, 2006). Up to 75% of individuals diagnosed with BPD engage in self-destructive behavior such as self-mutilation and suicide attempts (Posner, 2006).

1.2.3 Unstable interpersonal relationships.

Disturbed interpersonal relationships, characterized as relationships that have extreme highs and lows that often end in dismissal of a relationship, in BPD are mostly caused by infidelity and feelings of abandonment (Gratz & Gunderson, 2006). Due to the mercurial nature of their romantic relationships, BPD patients are more likely to be involved in one-night encounters or sexual promiscuity as compared to other personality disorders (Berman & Montgomery, 2014). In one study, it was found that 20 percent of BPD patients are involved with a romantic partner in the context of a dating, cohabiting, or married relationships whereas the percentages of patients with promiscuity are quite higher at rates of 50% to 60% (Clarkin, Levy, Lenzenweger, & Kernberg, 2007).

Loved ones of those with BPD often cannot bare the adultery in their relationships and end up abandoning their BPD partners (Berman & Montgomery, 2014). As a method to cope with the abandonment, BPD patients end up engaging in inappropriate acts such as calling ex-partners recurrently and getting into verbal and physical fights (Linehan, Armstrong, Suarez, Allmon, & Heard, 1991). Once they are rejected by these acts, BPD patients feel empty inside. This in turn leads to disturbances in understanding their identity.

1.2.4 Identity Disturbance.

Identity disturbance is a core criterion in BPD. When identity disturbance features are prominent in a patient diagnosed with BPD, they are more likely to have issues with other symptomatology. Specifically, BPD patients with identity disturbances are more likely to have issues with disturbed interpersonal relationships and managing affect instability (Koenigsberg et al., 2002). Moreover, identity disturbance is highly correlated with feelings of desperation and despair (Zanarini et al., 1998).

1.3. Mental State Decoding

Mental state decoding (MSD) – the ability to attribute mental states to social partners from perceivable social information such as tone of voice, body posture, or facial expression – provides an important tool for maintaining social relations and

cooperation and is an important representation in borderline personality disorder and may be affected in those with BPD. Human facial cues of the eyes provide one of the most important signals of mental states (Baron-Cohen et al., 2001).

The Reading Eyes in the Mind Test (RMET; Baron-Cohen et.al, 2001) is a test that matches semantic definitions of mental states to pictures of the eye-region with emotionally valence positive, negative, or neutral expressions. This comprehensive method measures mental state decoding abilities in different disorders. Inaccurately inferring mental states from facial cues leads to distorted interpretations of other people's intentions, which can cause problems in social functioning. Previous studies analyzing impaired mental state decoding abilities in depression and borderline personality disorder have produced contradictory results.

Suggestions of specific links between distorted perception of the intentions of others and depression can be found in cognitive-behavioral (Beck et. al, 1979), psychodynamic (Blatt & Levy, 2003), and mentalization-based (Luyten et. al, 2006) theories. Some studies indicate that depressed patients exhibit impaired RMET performance (Lee, Harkness, Sabbagh, & Jacobson, 2005; Manstead, Dosmukhambetova, Shearn, & Clifton, 2013; Nejati, Zabihzadeh, Maleki, & Tehranchi, 2012; Wang, Wang, Chen, Zhu, & Wang, 2008, Szanto et al., 2012), while others found no conclusive evidence of this impairment (Harkness, Washburn, Theriault, Lee, & Sabbagh, 2011; Kettle, O'Brien-Simpson, & Allen, 2008). This topic is further complicated by studies that suggest depressed individuals performed better on the RMET (Harkness et al., 2005), and were more sensitive to negative social information than their healthy counterparts (Wolkenstein et al., 2011).

The mental state decoding performance in subjects suffering from BPD follows this controversy. One study reported BPD patients having impaired social interpretation relative to healthy controls according to RMET, but subjects suffering from BPD with co-occurring major depression were significantly more accurate in decoding mental states of negative stimuli (Unoka et. al, in press). The same study indicated patients with both BPD and MD performed significantly better than patients with only BPD on negative and neutral items as well as total score (Unoka et al.2015). Comorbid depression was shown increase RMET accuracy (Fertuck et al., 2009; Unoka et al.,2015), perpetuating the idea that depression in BPD intensifies vigilance toward

social information similarly to depression (Harkness et al, 2005; Wolkenstein et al, 2011).

No systematic review or meta-analysis assessing RMET performance in depression and BPD, moderating effects of co-occurring depression, or other first and second axis disorders have been published. Consequently, a meta-analysis reviewing mental state decoding ability in MD and BPD patients is of the highest clinical and conceptual relevance.

In the current study, a quantitative meta-analysis on RMET performance of adults clinically diagnosed with BPD and MD was conducted. RMET performance was analyzed in total score and on negative, neutral, and positive valences. Further, the impact of potential moderators such as demographic and clinical variables that affect the patient and healthy control groups were considered. Since co-morbidity plays a large factor in clinical outcomes, we also assessed co-morbidity with BPD as a determinant of RMET performance.

1.4. Autobiographical Memory

In addition, Autobiographical memory (AM)—the memory system that contains personal memories and knowledge of self-related past events—has been an inconsistent deficit in the cognitive profile in BPD and provides an important tool for maintaining stable self-representations. Since recollection processes of AM have basic importance to the composition of the self and a continuing sense of identity (Lorenzzoni, Silva, Poletto, Kristensen, & Gauer, 2014), the normal functioning of AM is considered a prerequisite for adaptive personality functioning (McAdams & Pals, 2006).

AM is composed of memories referring to past personal experience (Conway, 2001). According to Conway and Pleydell-Pearce (2000), "AMs are transitory dynamic mental constructions generated from an underlying knowledge base" (p. 261). During remembering, the knowledge base–contained self-relevant information enters consciousness in the form of personal memories. One important characteristic of AMs is that they contain memories of different specificity: broader conceptual themes in the life story, lifetime periods, general events, and event-specific knowledge (Conway & Pleydell-Pierce, 2000; Conway, Singer & Tagini, 2004). "Broader conceptual themes of the life story" is based on a person's understanding of how a normative life story is constructed within a given culture; it is also called the life-story schema. "Lifetime

periods" refers to representations of prolonged periods of time with distinct start and end points. "General events" represent repeated or single events in an abstract way; and "event-specific knowledge" represents concrete sensory-perceptual aspects of unique events, including visual imagery. General events represent a form of AM psychologically distinct from specific memories, and in the retrieval process they are accessed earlier. Further, specific and general AMs can be distinguished at a neural level in terms of their greater associations with different regions of a common AM retrieval network (Addis, McIntosh, Moscovitch, Crawley, & McAndrews, 2004).

Autobiographical memory has been examined in several different measures; the Autobiographical Memory Test (AMT) is the most widely used. The AMT was first used by Williams and Broadbent (1986) on suicidal patients. The test requires participants to recall a specific episodic memory, answering to emotionally positive, negative, and neutral cue words within a given time period. Thus, the AMT test measures specific memories and overgeneral memories: extended memories that lasted more than one day; categorical memories, which are thematic summaries of events; and non-memories or semantic associates. In addition, AMT assess the frequency of omissions, in which the participant was unable to furnish a response, as well as latency time, in which participants respond to a given cue word.

Generative retrieval—used by the classic AMT—is a top-down process involving the retrieval of desired specific memories. During this process, the memory cue triggers an effortful search guided by the semantic knowledge of one's own life, which eventually leads to successful recovery of a target memory. Memory for the target might be elaborated by recovering additional details that give episodic richness to the memory (Addis et al., 2004). The effortful search could fail in case of omission or could be captured at the level of semantic or more general knowledge of one's life. The reduction in episodic richness could occur early during retrieval, while one is searching for the target memory, or late during retrieval, when one elaborates on recovered information. Elaboration processes might be also sensitive to BPD because they depend on an interaction between the recovery of specific details mediated by the hippocampus and strategic control processes mediated by the prefrontal cortex (PFC). Both processes and their associated brain regions are known to be affected in BPD,

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especially with early trauma history (Nunes et al., 2009), as well as in PTSD (Gilbertson et al., 2002) and depression (Young et al., 2012).

Williams and colleagues' (2007) CaR-FA-X model postulates that overgeneral memory (OGM) results when the generative retrieval search process is aborted prematurely as a result of one or more of the three proposed mechanisms (Williams et al., 2007): capture and rumination (CaR), functional avoidance (FA), and impaired executive control (X) (Sumner, 2012). Capture and rumination refer to processes during retrieval by which conceptual self-relevant information activates rumination and hence "captures" cognitive resources and disrupts the retrieval search. Functional avoidance refers to the affect regulatory strategy by which retrieval of specific memories is avoided. Impaired executive control means that deficits in executive resources limit the ability to conduct a successful retrieval search (Sumner, 2012).

Retrieval of event-specific knowledge has been shown so far to be reduced in AM memories of patients with depression, trauma survivors, acute stress disorder, schizophrenia, posttraumatic stress disorder (PTSD), eating disorders, and suicide (Williams et al. 2007); however, it may also apply to borderline personality disorder.

In BPD, research on AM has been contradictory. In the DSM fifth edition (*DSM-5*; American Psychiatric Association, 2013), patients' diagnoses of BPD are characterized by identity disturbance: "markedly and persistently unstable self-image or sense of self" as well as "chronic feelings of emptiness," which could be related to AM malfunctioning (Bech, Elkit & Simonsen, 2015). Further, individuals with BPD are characterized with impaired front limbic connections, reduced volume of hippocampus that may underlie impaired executive functions, rumination, and capture. Also, individuals with BPD have high rates of childhood trauma (e.g., Goodman & Yehuda, 2002; Yen et al., 2002), PTSD (McGlashan et al., 2000; Yen et al., 2002; Zanarini et al., 1998), and depression (McGlashan et al., 2000; Zanarini et al., 1998), suggesting that if traumatic events, PTSD, and depression are crucial to overgenerality, then it would most likely be evident in BPD (Moore and Zoellner, 2007).

According to several earlier studies, people with BPD have a tendency for overgeneral memory (Jones et al., 1999; Korfine, 1998; Maurex et al., 2010)—thought to be a dissociative mechanism serving to avoid painful negative memories—but further studies failed to confirm this claim (Renneberg, Theobald, Nobs, & Weisbrod,

2005; Reid & Startup, 2010). Another finding contradictory to motives mentioned behind overgeneralization is that BPD patients recall more negative memories than healthy controls (Nigg, Lohr, Westen, Gold, & Silk, 1992) and view such as more relevant for today's life (Rosenbach & Renneberg, 2015).

1.5 Neuropsychological Functioning

Due to the considerable variability in symptoms, neuropsychological deficits have been identified as a robust feature of BPD and are a central manifestation of the pathophysiology of the disorder. In the first major literature analysis based on 10 studies, Ruocco (2005) found evidence of significant impairment across the full range of neuropsychological tests. The most consistent impairments were found in the domains of attention, cognitive flexibility, learning and memory, planning, processing speed, and visuospatial abilities. The nature and magnitude of such impairments, as well as their consistency, varied markedly across studies. This considerable heterogeneity of effect sizes was evident within many of the neuropsychological domains examined (Ruocco, 2005). These inconsistent findings may be explained by the confounding effects of differences in sample characteristics, co-morbidity profile and research methodologies. The relatively small number of studies included in Ruocco's meta-analysis, however, precluded any meaningful exploration of these potential moderating variables. Ten years after Ruocco's paper the available amount of studies on cognitive functioning of BPD makes it possible to explore the con- founding effects of these potential moderating variables.

Overall, while sustained attention, inhibition, executive function, decision making, language, memory, verbal IQ, visuospatial processing deficits are frequently reported, the nature and magnitude of such impairments, as well as their consistency can vary markedly across studies due to differences in sample characteristics, co-morbidity profile and research methodologies. In this context, meta-analysis is a useful tool for systematically combining all research in this area to identify cognitive deficits, showing the most robust changes in BPD. It is also a useful methodology to work out the effect of confounding factors. In this way, we may be able to better understand the pervasive cognitive disturbances that cannot be explained by the effects of medication

factors or by the effects of co-occurring mental disorders, as well as their neural underpinnings in BPD.

To our knowledge, the effects of co-occurring disorders in BPD neuropsychological functioning have not been analyzed; however, recent studies have suggested that co-morbidity with BPD is tremendously prevalent in this population. Specifically, BPD is highly co-morbid with a number of Axis I and other personality disorders). Further analysis showed that anxiety disorders were most common, followed by PTSD (Zanarini et al., 1998). Regarding personality disorders, paranoid, avoidant, and dependent personality disorders were most co-morbid both at first diagnosis and at a 6-year follow-up (Zanarini et al., 2004). Ten, twelve, and sixteen-year follow- ups showed stagnant results (Zanarini & Frankenburg, 2008). Based on this, our aim was to take into account the highly co-morbid aspects of BPD as a method to learn about neuropsychological functioning.

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2. Objectives

The three aforementioned cognitive core elements of BPD have been inconsistently represented in the literature regarding their level of impairment and high functioning. As a method to further elucidate exactly the cognitive abilities existing, three separate meta-analytic reviews, which look all studies on these topics, were undertaken. The expected outcome of the work is that a clearer understanding of the exact deficits of cognitive functioning and heightened areas will further be understood. Moreover, moderator analysis will be expected to highlight which areas are specifically related to categorical and continuous variables such as demographic and clinical variables.

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3. Methods

Three meta-analytic reviews were undertaken in each of the categories that were aforementioned as part of the cognitive profile investigated. Each looked at the moderator variable interaction as well as comorbidity.

3.1 Mental State Decoding Meta-Analysis

Relevant articles were identified through a computerized literature search using PubMed, PsychINFO, and MEDLINE Web of Science databases. Search terms included, “Reading in the Mind of the Eyes Test,” “Borderline Personality Disorder,” “Major Depression,” OR “Unipolar Depressive Disorder,” “Theory of Mind,” AND “Mental State Decoding" OR "Mentalization." The search was limited to articles that were published between 2000 and January 2014. Additionally, a manual review of each article was performed utilizing cross-references from original articles and reviews. Eligible studies compared RMET performance in patients diagnosed with BPD or MD with healthy control patients. The search yielded 30 studies that met the requirements. Studies to be included in the meta-analysis were reviewed by the supervisor and PhD student and followed these criteria: 1) focused on RMET performance in adult aged patients with BPD or adult aged patients with MD compared to healthy controls, and 2) provided data or statistical information that allowed for the calculation of an effect size.

After further review, 14 of the original 32 studies were included due to the comparative results of RMET. Reasons for exclusion included: 1) absence of control groups (N = 10), 2) control groups meeting clinical criteria for either Bipolar Disorder (N = 1) or esophageal cancer (N=1), 3) BPD groups displaying characteristics of the disorder, but no clinical diagnosis (N=1), 4) lack statistical information for calculating effect size (N = 4), and 5) clinical groups with patients having a mean age under 18 (N = 1).

Only 7 of the 13 studies included reported RMET valence scores. Authors that did not report the relevant information were contacted, but either did not have the required information or failed to respond. One study provided valence scores for patients, but not for controls. Therefore, we performed two analyses: one on accuracy (N = 13) and one on valence scores (N = 7).

3.1.2 Mental State Decoding Moderator Variables

Within the patient population, the following moderator variables were also coded: 1) mean age at the time of testing, 2) sex (i.e., percentage male), and 3) co-morbidity diagnosis. Symptom severity, additional demographic characteristics (ethnic background and education level) were considered, but ultimately were not amply reported to be included in analyses.

For co-morbidity diagnoses other than BPD and MD, there was not enough data to run meta-analyses differences on valence scores. Therefore, only analysis on RMET overall accuracy was undertaken for co-morbidity of BPD and any anxiety disorder, eating disorder, substance abuse disorder, and clusters A, B, and C personality disorders. In the studies of patients with MD, we sought to examine how co-morbidity might affect this group; however, too few of studies reported this data for us to use it in our analysis. Thus, we only assessed how co-morbidity influenced BPD groups

3.2 Autobiographical Memory Meta-Analysis

Our articles were found through a computerized literature search using the databases PubMed, PsychINFO, and MEDLINE Web of Science. Search terms included, “Autobiographical Memory Test” OR “AMT” AND “Borderline personality disorder” OR “emotionally unstable personality disorder.” The search was limited to articles that were published between January 1990 and April 2017. Additionally, a manual review of each article was per- formed utilizing cross-references from original articles and reviews. Eligible studies compared the AMT task in patients diagnosed with BPD to healthy control patients (HC). The search yielded 21 studies that met the requirements.

Inclusion criteria for studies to be included in the meta-analysis were the following: (a) focused on the outcomes of AMT in patients clinically diagnosed with BPD as compared to healthy controls; (b) was an experimentally carried-out study using data or statistical information that allowed for calculation of an effect size; and (c) was a manuscript, peer-reviewed journal article, dissertation, or book article that had statistical information such as a p value, t value, F value, or means and standard

deviation that could be used to calculate a Cohen's *d* value. Healthy control groups were chosen as an inclusion criteria factor in order to create unanimity between groups and because they are a common practice within meta-analytic studies assessing psychiatric patients. Studies were excluded if they did not include (a) a control group with a clinical diagnosis, (b) data for calculation of an effect size, (c) BPD patients not diagnosed according to clinical guidelines within the *DSM*, or (d) a control group at all. In the case that studies included all relevant inclusion criteria but did not have data reported in some capacity but could have, the student contacted the authors. Ten studies were excluded after further review. Studies were excluded because studies: (a) assessed autobiographical memory but did not use the AMT task (n = 3), (b) had an absence of control groups (n = 4), (c) had control groups of other clinical diagnoses (n = 3), and d) lacked statistical values enough for inclusion in the meta-analysis.. The final meta-analysis included 10 studies (29 effect sizes), which included all respective information of the inclusion criteria.

3.2.1 Autobiographical Memory Moderator Variables

The moderator variables were: (1) type of autobiographical memory assessed: overgeneral, omission, specific, and recall; (2) patient age at time of testing; (3) sex (i.e., percentage of sample that is male); and (4) intelligence (i.e., IQ as assessed by the Wechsler Adult Intelligence Scale (Wechsler, 1997, 2014). We sought to look at BDI scores, ethnicity, comorbidity, trauma history, medication status, and education level; however, not enough studies provided enough information for calculation of an effect size. Only three studies provided data on BDI scores, two on comorbidity, and one on medication status.

In addition, we sought to look at all the autobiographical memory types in the AMT; however, only the overgeneralizing, omission, specific, and recall types were reported in the final studies included in the meta-analysis. *Overgeneralizing* referred to the participants' response in the AMT when they could not recall personal memories of specific events; *omission* referred to the absence of responses to the AMT cue word, *specific* referred to the retrieving of a personal memory of specific events; and *recall* referred to the latency of retrieving specific memories to cue words.

3.3. Neuropsychological Functioning Meta-Analysis

Relevant articles were identified through a computerized literature search using PubMed, PsychINFO, and MEDLINE Web of Science databases. Search terms included, “Borderline personality disorder" OR "BPD” AND “neuropsychology, attention, processing speed, executive functioning, decision making, memory, intelligence, and visual perceptual" OR "spatial.” The search was limited to articles that were published between 1990 and June 2014. Additionally, we performed a manual review of each article by cross-referencing from original articles. We also took into consideration the Ruocco, 2005 paper for finding our articles.

Eligible studies compared neuropsychological functioning in borderline personality disorder (BPD) with healthy control patients. The search yielded 53 studies that met the requirements. Studies to be included in the meta-analysis were reviewed by the PhD student and supervisor and followed these criteria: 1) focused on neuropsychological performance in adult aged patients with BPD compared to healthy controls, and 2) provided data or statistical information that allowed for effect size calculation.

After further review, 25 of the original 53 studies were included after discussion between authors. Reasons for exclusion included: 1) absence of control groups (N = 12) 2) Attention Deficit Hypertension Disorder (ADHD) comparison group (N= 2) 3) clinical group mixed with BPD and antisocial personality disorder (N= 1) 3) BPD groups displaying characteristics of the disorder, but no clinical diagnosis (N=1), 4) little statistical information for calculating effect size (N = 7), and 5) clinical groups with patients having a mean age under 18 (N = 3).

3.3.1 Neuropsychological Functioning Moderator Variables

We sought to define neuropsychological functioning by looking at effect size across ten domains including: 1) overall intellectual ability (i.e., full-scale IQ), 2) verbal intelligence (VIQ), 3) nonverbal intelligence (PIQ), 4) executive functioning, 5) attention, 6) decision making, 7) processing speed, 8) memory 9) visuospatial abilities and 10) language. Assignment of neuropsychological tests to selected domains was guided by the classifications made in source articles and consensus of the authors. In

the absence of assignment in source articles, tests were assigned to domain based on discussions between supervisor and PhD student.

Within the patient population, the following moderator variables were also coded: 1) mean age at the time of testing, 2) sex (i.e., percentage male), 3) race (i.e., percentage Caucasian) 4) mean education of patient, 5) mean education of the patients' parents (i.e., mean in years), 6) anti-depressant prescription (i.e. percentage on an anti-depressant prescription) and 7) co-morbidity diagnosis (i.e. percentage of sample with either cluster A, B, C personality disorders, major depression (MD), any eating disorder, any substance abuse history, any anxiety disorder, or post-traumatic stress disorder (PTSD). Bipolar disorder was considered as an additional co-morbidity to include, but not ample enough studies reported such disorder. We decided to assess current co-morbidity as not enough studies reported lifetime co-morbidity. We also sought to analyze differences between patients on inpatient vs. outpatient status, handedness, and other medications (e.g. anti-psychotics, benzodiazepines, and phase prophylactics); however, there was not enough data reported in these categories.

Statistical Analyses

The meta-analyses was conducted with Comprehensive Meta-Analysis Version 3.0 software (Borenstein, 2005). Scores were standardized by calculating Cohen's *d* of studies comparing scores between patients and healthy controls. Effect sizes were calculated based on the difference of two raw means divided by the pooled standard deviation (SD) and were classified as small ($d = 0.2$), medium ($d = 0.5$), or large ($d \geq 0.8$) (Cohen, 1988). Cohen's *d* was calculated from reported means and standard deviations (SD), univariate F-tests, t-statistics, or p-values. Confidence intervals (CI) and Z-values of the effect sizes were used to assess statistical significance. The Cochran *Q*-statistic was used to examine homogeneity of the effect sizes across studies as well as between clinical diagnoses (Hedges, 1985). When analysis of the *Q*-statistic revealed significant within-group heterogeneity, a random-effects model was used for the significance level. In addition to a visual funnel plot, methods for the evaluation of potential publication bias included those recommended by Begg and Mazumdar (1994) and Egger, Smith, Schneider, and Minder (1997). When categorical domains exhibited significant heterogeneity, potential moderators were considered with the *Q*-statistic.

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The effects of demographic moderator variables such as age (e.g. mean age) and sex (e.g. male percentage) were analyzed with meta-regression.

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4. Results

Results from all three meta-analyses are elaborated on below and divided by subtype.

4.1 Mental State Decoding

Overall accuracy meta-analysis results Analysis of effect sizes across differences in performance on patients with BPD and patients with MD revealed a moderate overall effect size (N = 14, d = -0.334 p= 0.02) that was significantly heterogeneous (Q_B = 58.63, p < .001). Given that the variability in effect sizes between patient and healthy comparison groups differed more from sampling error alone, analysis of the moderator variables was assumed. See figure 5.

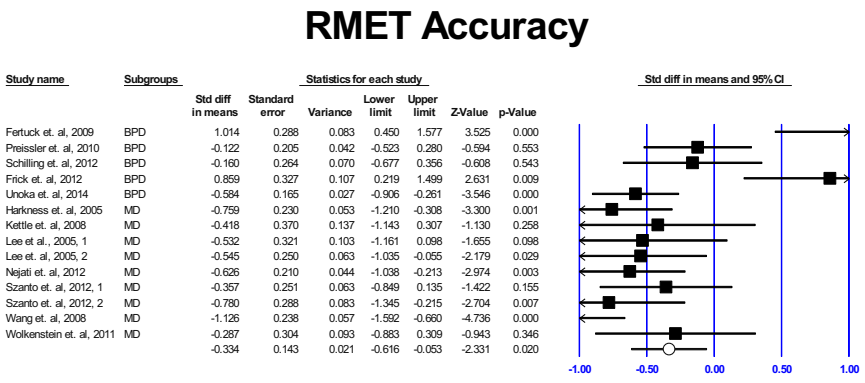

Figure 5. Overall meta-analysis accuracy scores for the RMET

4.1.1 Publication bias

There was no evidence of any publication bias possibility as indicated by non-significant Begg & Mazumdar rank correlation (P = 0.55) and Egger (P = 0.612) tests. Nevertheless, calculation of a fail-safe N revealed that a total of 87 'null' studies would be needed to reduce the observed effect to 0.20. This shows that the analysis contains enough studies for completion of the meta-analysis.

4.1.2 Overall RMET valence scores

Analysis of effect sizes across differences in valence performance in patients with MD and patients with BPD revealed a moderate overall effect size (N = 21, d = -0.34) that was significantly heterogeneous (Q_B = 35.49, p < .001). Given that the variability in effect sizes between patient and healthy comparison groups differed more from sampling error alone, analysis of the moderator variables was assumed.

4.1.3 Publication bias in valence scores.

Analysis for possible response bias revealed an asymmetric funnel plot and significant Begg (p = .0001, 1-tailed) and Egger (p = .001, 1-tailed) tests, suggesting a potential publication bias in this literature. To address this, we calculated a fail-safe N, which exposed that 812 "null" studies would need to be found and incorporated in the analysis to negate the presented effect. As such, the current data are felt to accurately represent the extant literature on RMET valence scores.

4.1.4 Moderator Analysis

4.1.5 BPD vs. MD on overall accuracy.

Moderator analysis comparing patients with BPD to patients with MD was significantly heterogeneous (Q_B [12] = 48.337, p < .001). Large effect sizes were seen in performance among patients with MD (d = -0.751). BPD patient results were not significant (p= 0.48). See figure 6.

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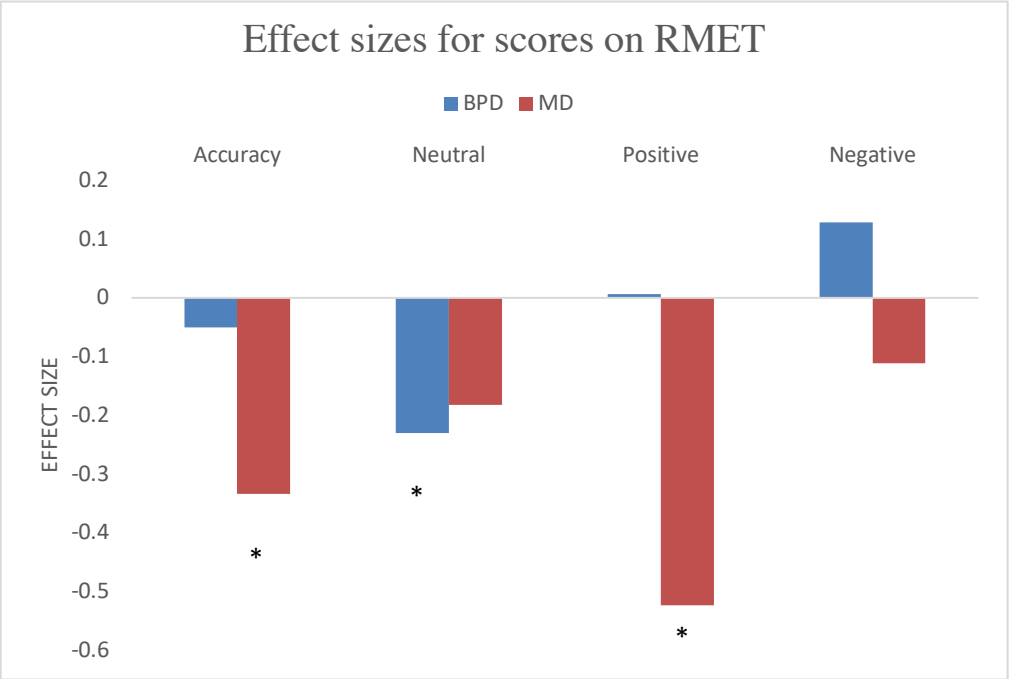

Figure 6. Effect sizes for significant scores on the RMET. *= Statistically significant

4.1.6 BPD v. MD on valence scores.

For overall valence, MD (N =21, *d* = -0.272) was significantly worse than BPD and healthy controls. Both MD (Q_B [10] = 73.50, p < .001) and BPD (Q_B [6] = 70.76, p < .001) showed significant heterogeneity among valence type. When comparing among valence type, patients with MD were significantly impaired on positive valence (*d* = -0.523). Conversely, patients with BPD were significantly impaired on neutral valence (*d* = -0.230). See figures 7 and 8.

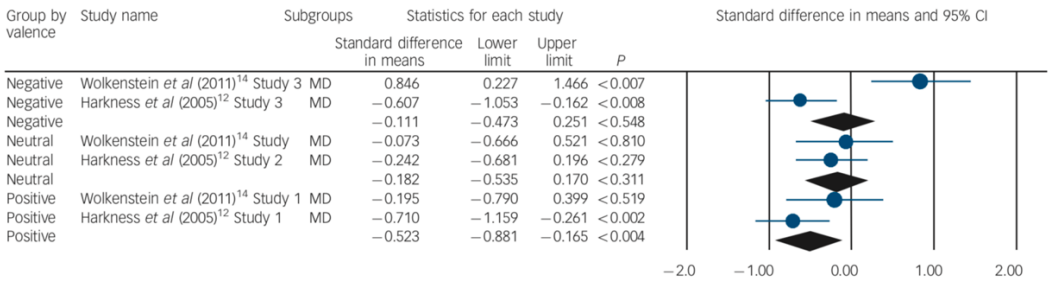

Figure 7. RMET meta-analysis valence scores in patients with MD

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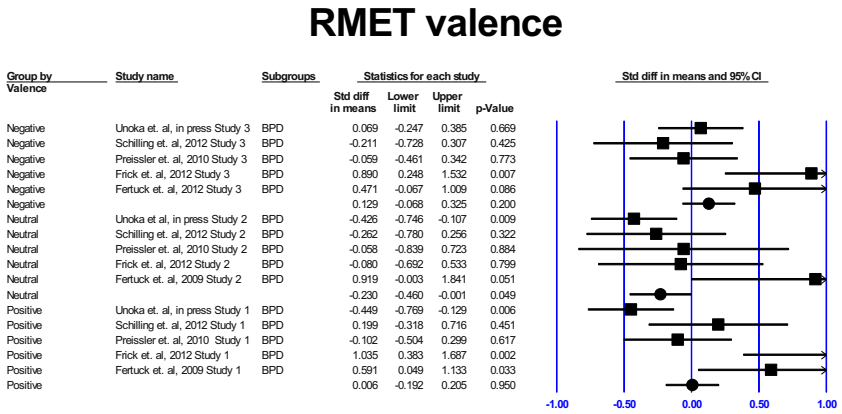

Figure 8. RMET meta-analysis valence scores in patients with BPD

4.1.7. Borderline personality disorder with major depression

4.1.7.1 Overall accuracy.

Comorbidity of BPD and MD significantly affected overall accuracy on RMET (N = 5, Z = 3.45, p <.0005). Patients with BPD and MD performed better than BPD alone.

4.1.7.2 Positive valence scores.

Those with BPD and MD performed better on positive valence tasks of the RMET than regular BPD patients. (Z= 2.79 p < .005). For both, see figure 8.

4.1.8 Borderline personality disorder and other disorders

4.1.8.1 Any anxiety disorders.

The relationship between comorbidity of BPD and any anxiety disorder on overall RMET performance observed was not significant (N = 5, Z = .336, p = .75). The relationship between co-morbidity of BPD and any anxiety disorder among RMET valence was not significant (N= 15; Z= .56; p= .86).

4.1.8.2 Any Eating Disorders

The relationship between comorbidity of BPD and any eating disorder in relation to performance on the RMET test was observed to be significant (N= 5 effects; Z= 1.13; $p < .005$). Patients with a dual diagnosis of BPD and any eating disorder performed better on the test than having a disorder of BPD without it. Valence showed no differences between co-morbid BPD and eating disorder (N= 15; Z= .18, $p = .93$).

4.1.8.3 Any substance abuse disorder history.

The relationship between comorbidity of BPD and any substance abuse disorder history was not observed to be significant. (N= 5; Z= .486; p= .67). Valence results were not significant (N= 15; Z= .57; p= .83).

4.1.8.4 Any cluster A personality disorder.

The relationship between co-morbidity of BPD and any cluster A personality disorder performance on the RMET was observed to be not significant. (N= 4, Z= -2.28; p= .63). Valence scores were also not significant (N= 12; Z= -3.38; p= .83).

4.1.8.5 Any cluster B personality disorder.

The relationship between co-morbidity of BPD and any cluster B personality disorder performance on the RMET was observed to be significant. (N= 4; Z= -3.17 $p < .001$). Patients with a dual diagnosis of BPD and any cluster B personality disorder performed worse on the test than having a disorder of BPD without it. This patient group also performed worse than healthy controls. Valence scores were not significant (N= 12; Z= -.68; p= .32).

4.1.8.6 Any cluster C personality disorder.

he relationship between a co-morbidity of BPD and any cluster C personality disorder performance on the RMET was observed to be significant. (N= 4; Z= -3.01 $p < .001$). Patients with a dual diagnosis of BPD and any cluster C personality disorder performed worse on the test than having a disorder of BPD without it. This patient group also performed worse than healthy controls. Valence scores were not significant (N= 12; Z= -.89; p= .12).

4.1.9 Demographic characteristics

4.1.9.1 Age.

Analysis of age composition among the samples reporting mean age revealed no significant differences for BPD and MD among accuracy (N=11; Z= -0.26; p=.53).

4.1.9.2 Sex.

Analysis of sex composition of the samples reporting gender percent revealed that no significant differences among BPD and MD among accuracy ($N = 9$; $Z = -1.81$, $p = .6$).

4.2 Autobiographical Memory Meta-Analysis Results

4.2.1 Overall Meta-Analysis Results

The total sample size included 1,979 participants. Table 1 shows descriptive statistics of the studies. Analysis of effect sizes across differences in performance of patients with BPD as compared to HCs revealed a moderate overall effect size, $d = –0.564$, that was significantly heterogeneous, QB (114.34), $p < .0001$. Given that the variability in effect sizes between patient and healthy comparison groups differed more than from sampling error alone, analysis of the moderator variables was assumed. A forest plot of all studies can be seen in Figure 3. Descriptive statistics are in table 1.

4.2.2 Publication Bias.

Existence of possible response bias revealed an asymmetric funnel plot and significant Begg ($p = .001$, 1-tailed) and Egger ($p = .003$, 1-tailed) tests, suggesting a potential publication bias. To address this, calculation of a fail-safe N exposed that 900 “null” studies would need to be found and incorporated in the analysis to negate the presented effect. Thus, the data accurately represented the autobiographical literature of AMT in patients diagnosed with BPD as compared to healthy controls.

4.2.3 Type of Autobiographical Memory.

The significant impairments in BPD were the following: overgeneralizing (d = –1.09; $p < .0001$), omission (d = –0.64; $p < .001$), and specific (d = –0.39; $p < .01$). No significance existed in recall (d = -0.32; $p = 0.33$). Results are depicted in Figure 4.

4.2.4 Age.

In BPD, meta-regression revealed that as age increases, samples with an older mean age had fewer impairments in autobiographical memory ($Z = 2.81$; $p < .0001$). Such results prompted further analyses in which we looked at autobiographical memory type and age. It was found that there was a significant association with age and specific memory. As age increased, there were fewer autobiographical specific memory impairments ($Z = –1.3$; $p= .004$).

4.2.5 Sex.

When looking at sex as a moderator variable in BPD patients, results revealed no significant differences. ($Z = –1.62$; $p = 0.04$).

4.2.6 Intelligence.

There was no significant moderation by IQ ($Z = 0.81$, $p = 0.56$).

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Study Name	% male	Number of effect sizes	Stimulus types
Jones 1999	21.8	5	Eighteen words, six positive, six negative and six neutral, were used as cues in each version
Kremers 2004 Subgroup 1	8.4	1	Five negative words and five positive words were read aloud on each card.
Kremers 2004 Subgroup 2	6.4	1	Five negative words and five positive words were read aloud on each card.
Kremers 2006	0	6	Respondents were asked to mention a specific moment when he or she had displayed traits that were presented one by one and asked to recall a specific event
Maurex 2010	0	5	36 cue words with positive, negative, and neutral words alternating.
Reid 2010, Subgroup 1	22.8	2	lists of six positive and six negative cue words. Neutral cue words, were not administered
Reid 2010, Subgroup 2	22.2	2	lists of six positive and six negative cue words. Neutral cue words, were not administered
Rennenberg 2005	0	6	15 emotional cue words: 5 positive (happy, successful, safe, carefree, interested), 5 negative (sorry, lonely, hurt, clumsy, angry) and 5 neutral adjectives (modern, personal, oval, cultural, historic).
Rosenbach 2015	6.7	2	five rejection-related cue words (rejected, neglected, ignored, repelled, unwanted) and five positive cue words (safe, carefree, happy, successful, interested)

Table 1. Study Descrpitives for Autobiographical Memory Included Studies

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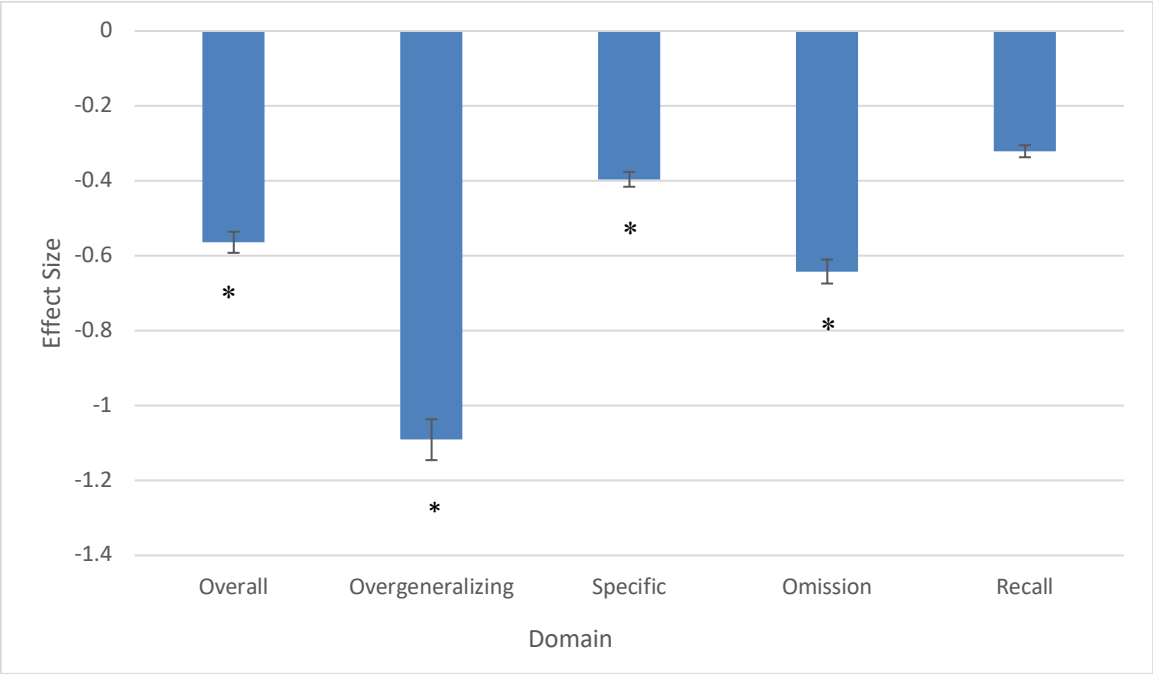

Figure 4. Autobiographical memory by disorder and memory type. Error bars represented at a 95% confidence interval; * denotes significance

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4.3 Neuropsychological Functioning Meta-Analysis results

4.3.1 Overall Meta-Analysis Results.

Analysis of 202 effect sizes across neuropsychological domains for the entire sample revealed a small to medium overall effect size (N =8957, d = -0.31, p <. 01) that was significantly heterogeneous (Q_B [201] = 71.5, p < .001). Given that the variability in effect sizes between patient and healthy comparison groups differed more than would be expected from a sample error, analysis of moderator variables was commenced.

4.3.2 Publication Bias.

Analysis for the presence of possible response bias revealed an asymmetric funnel plot and significant Begg (p = .001, 1-tailed) and Egger (p = .001, 1-tailed) tests, suggesting a potential "file drawer" problem and/or publication bias in this literature. To address the second, calculation of a fail-safe N revealed that 7055 "null" studies would need to be found and incorporated in the analysis to negate the presented effect. As such, the current data accurately represent the existing literature regarding neuropsychological functioning in patients with BPD.

4.3.3 Moderator Analysis

4.3.3.1 Neuropsychological domain.

Moderator analysis across the 10 specific domains of neuropsychological function revealed significant heterogeneity among effects (Q_B[9] =71.5 p < .01). As can be seen in Figure 1, significant effect sizes were seen in decision making (d = 0.617) memory (d = -0.57) and, executive functioning (d = -0.48); smaller significant effect sizes were seen in processing speed (d= -0.22), verbal intelligence (VIQ) (d= -0.38), visuospatial abilities (d= -0.41), and attention (d= -0.18). The values were: (full scale IQ; d= -.012, p= .56; language; d= -.041, p= .334; PIQ *d*= -.0883, p= 0.083). See figure 1.

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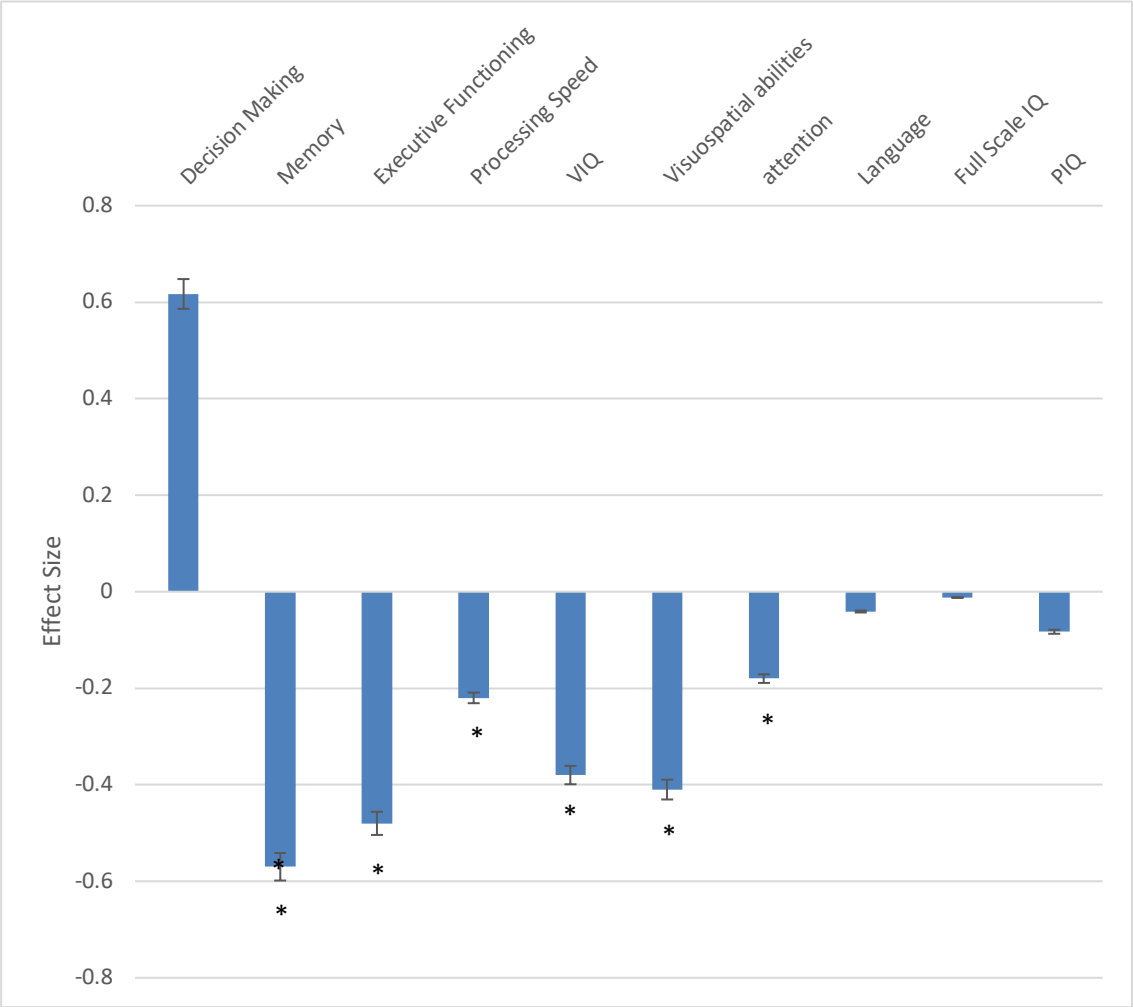

Figure 1. The outcome of the neuropsychological meta-analysis with domains. *: denotes statistical significance

4.3.3.2 Age.

Analysis of age showed no significant differences (N= 99; Z= 4.42, p= .67). The average age was 31.09.

4.3.3.3 Gender.

Analysis of gender composition of the samples revealed no significant differences (N = 72, Z = -1.48, p= .21).

4.3.3.4 Race.

Analysis of race as a moderator variable exposed no significant differences (N= 6, Z= -1.44, p= .25).

4.3.3.5 Patient education.

Analysis of mean education year of patients showed a smaller magnitude of deficit was seen in those with more education in a global cognition (N = 87, Z = -2.66, p= .007).

4.3.3.6 Parent of patient education.

Analysis of the parental mean education showed that those patients with parents having more education level had better neuropsychological functioning as far as a global cognition (N= 4, Z= 4.90, p < .0001).

4.3.3.7 Any anti-depressants.

Analysis of samples including percentage of patients prescribed to anti-depressants revealed no significant differences (N= 9, Z= 0.84, p= .97).

4.3.3.8 Co-morbid BPD with any cluster A personality disorder.

Analysis of the percentage of samples with co morbidity of cluster A personality disorders from each group exposed that as samples increased with percentage of comorbidity of cluster A personality disorders, patients performed worse (N= 22, Z= -4.19, p < .0001).

4.3.3.9 Co-morbid BPD with any cluster A personality disorders on neuropsychological domains.

When assessing the differences among each neuropsychological domain, results exposed samples containing co-morbidity cluster A personality disorders had no significant differences between specific neuropsychological domains (p = .93).

4.3.3.10 Co-morbid BPD with any cluster A personality disorders on gender.

When assessing the differences between genders and neuropsychological

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functioning, it was found that samples including woman with a higher percentage co-morbid BPD and cluster A personality disorders performed worse than men (N= 21 effects, Z= -6.39, $p < .0001$) explaining the heterogeneity. Figure 2 has these items.

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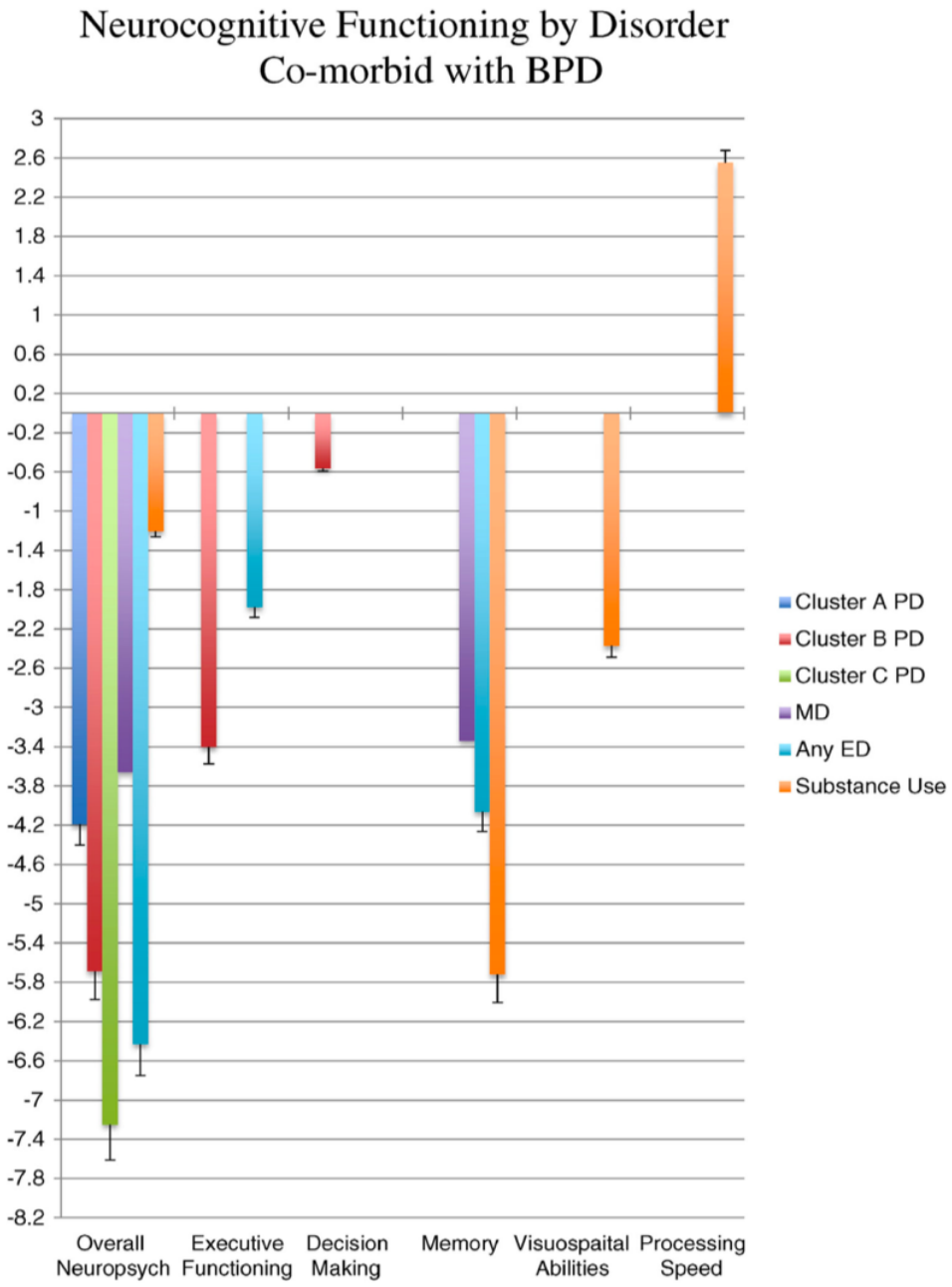

Figure 2. Significant domains in co-occurring disorders with BPD depicted in bar graph. The colored legend on the right of the figure highlights which co-morbid disorder with BPD. The Z scores are visually depicted. Note: this only refers to the studies that have included values within these variables. For example, if a study did not include any co-morbid disorders reported, then it was not included in this analysis.

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4.4 Co-morbidity variables

4.4.1 Co-morbid BPD with any cluster B personality disorder.

BPD patients performed worse in neuropsychological functioning when a higher percentage of co-morbid cluster B personality disorder was evident (N= 41 effects, Z= -5.69, $p < .0001$).

4.4.2 Co-morbid BPD with any cluster B personality disorders on neuropsychological domains.

Results showed that BPD samples with a higher percentage co- occurring with any cluster B personality disorder were more impaired in decision making (Z= -.56, $p < .0001$) and executive functioning (Z= -3.40, $p < .0001$). See figure 2.

4.4.3 Co-morbid BPD with any cluster C personality disorder.

Meta-analysis revealed those samples with a higher percentage of co-occurring cluster C personality disorder did worse performed samples with a lower percentage of co-occurring cluster C personality disorders (N= 22 effects, Z= -7.25, $p < .0001$). See figure 2.

4.4.4.Co-morbid BPD with any cluster C personality disorder on neuropsychological domains.

When assessing the differences among each neuropsychological domain, results exposed the samples with any BPD co-occurring with any cluster C personality disorder had no significant differences between any specific neuropsychological domains ($p = .81$). See figure 2.

4.4.5 Co-morbid BPD with any cluster C personality disorders on gender.

When assessing the differences between genders and neuropsychological functioning, it was found that samples with a higher percentage of men with co-morbid BPD and cluster A personality disorders performed worse than woman with a higher percentage of co-morbid BPD and Cluster A personality disorders

(N= 21, Z= -7.48, p < .0001) explaining the heterogeneity. See figure 2.

4.4.6 Co-morbid BPD with MD.

Analysis of co-occurring BPD with MD showed those samples a higher percentage of patients with BPD/ MD co-occurring did worse than patients with a lower percentage BPD and MD co-occurring (N= 82, Z= - 3.66, p < .0001). See figure 2.

4.4.7 Co-morbid BPD with MD on neuropsychological domains.

When assessing the differences among each neuropsychological domain, results exposed the samples with a higher percentage BPD/ MD co-occurring were the most impaired in memory (Z= -3.34, p < .0001). See figure 2.

4.4.8. Co-morbid BPD with any eating disorder

Analysis showed that the samples which had a higher percentage of any co-occurring eating disorder with BPD performed worse than with a lower percentage (N = 94 effects, Z = −6.43, p b .0001).

4.4.9 Co-morbid BPD with any eating disorder on neuropsychological domains.

Analysis between each neuropsychological domain showed that those with a higher percentage of any co-occurring eating disorder with BPD had higher levels of impairments in executive functioning (Z = −1.98, p = .005) and memory (Z = −4.06, p b .0001).

4.4.10 Co-morbid BPD with any substance abuse disorder/history Analysis showed that the samples which had a higher percentage of any co-occurring substance abuse with BPD performed worse than with a lower percentage (N = 68 effects, Z = −1.2, p b .0001).

4.4.11 Co-morbid BPD with any substance abuse disorder/history on neuropsychological domains.

Analysis between each neuropsychological do- main showed that those with a

higher percentage of any co-occurring substance abuse disorder/history with BPD have higher levels of impairments in memory (Z = –5.72, p b .0001), visuospatial abilities (Z = –2.94, p = .04), and VIQ (Z = –2.37, p = .012); however, analysis showed that the samples which had a higher percentage of any co- occurring substance abuse/history disorder with BPD are better on processing speed than with a lower percentage (Z = 2.55, p b .0001).

4.4.12 Co-morbid BPD with any anxiety disorder.

No significant differences were seen in this group (p= .1627). Co-morbid BPD with PTSD. No significant differences were seen in this group (p= .69). See figure 2.

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5. Discussion

The current work extends what we know on the overall cognitive functioning in borderline personality disorder. The discussion following below will outline each withstanding area with further elaboration.

5.1 Mental State Decoding

The meta-analysis conducted extends the current literature on the RMET performance in patients with BPD and patients with MD. The meta-analysis was performed relative to healthy comparison subjects and reviewed potential moderator variables that may influence RMET performance. To our knowledge this is the first meta-analysis to assess RMET performance differences among these patient populations. In addition, studies assessing either patients with BPD or MD have been limited by a small sample. Our study prolongs prior findings to a large sample in accuracy (N=935) and even larger in valence (N=1525), thus generalizing and providing new findings for the literature.

Our meta-analysis results from thirteen studies revealed a large overall effect size for global RMET accuracy performance. These results revealed patients with MD and BPD performed significantly worse than healthy controls. In addition, the overall effect was heterogeneous with differences in performance between patients with BPD and MD. This moderator analysis revealed patients with MD performed worse than BPD patients. Literature has emphasized patients with BPD have vast impairments in mentalization (Bateman & Fonagy, 2008). However, these meta-analyses may elaborate on decreased mentalization abilities in patients with major depression (Harkeness, 2005; Wolkenstein, 2003).

In addition to accuracy, we assessed valence outcomes among patient groups. Of the studies, we were limited, but we were able to extract valence scores and examine within patient and valence differences. Overall valence scores showed significant differences, which allowed this calculation of within group valence analysis.

The positive valence deficit in MD indicates impairment in the processing of positive facial emotional cues. These findings are in line with previous studies of MD and the processing of facial expressions with positive valence. Patients with major depression relative to healthy controls show reduced accuracy of happy facial expressions, reduced attention towards positive facial expressions (Suslow et al., 2001), selective attention away from happy faces (Surguladze et al., 2005), and tendency to evaluate neutral and ambiguous expressions less happy (Bourke et al., 2010). Our results provide partial support for the hypothesis of a mood congruent impairment of mental state decoding in MD, specifically that low positive affectivity and anhedonia is related to impaired ability to decode rewarding positive facial expressions. The neural basis of this social reward processing deficit may be related to the decreased activity in bilateral fusiform gyri and ventral striatum (right putamen) in response to happy faces, which was found by Surguladze et al. (2005). Further, these positive valence-decoding impairments could affect the interpersonal perception and could contribute to low self-esteem, social isolation, and impairment. Contrary to some previous findings (Harkness et al, 2005; Wolkenstein et al, 2011; Fertuck et al., 2009; Unoka et al., in press) and negativity bias theory of depression in our meta-analysis we did not find more accurate decoding ability of facial expressions with negative valence. These results show that MD patients are not homogenous regarding selective attention to negative emotional stimuli (negativity bias), and related higher accuracy in decoding negative facial expression. Further RMET studies on MD should report symptom profile and severity indexes to make possible the identification of sub-groups with more and sub-groups with less accurate negative mental state decoding abilities.

We found that BPD patients were overall significantly impaired in decoding mental states with neutral valence (e.g., reflective). Our findings are consistent with the findings of a meta-analysis on facial emotion recognition in BPD (Daros et al., 2013); patients suffering of BPD misattribute emotions to faces depicting neutral expressions. This emotionalizing tendency may relate the largely consistent findings of amygdala hyperactivity during facial emotion processing

(Donegan, 2003, Frick, 2012 Mier 2012, Mizenberg 2007). Further, this emotionalizing tendency of affectively neutral expressions in BPD can contribute to misunderstandings in social interactions, i.e. patients with BPD may interpret the neutral expressions as a threat (Domes et. al, 2004).

For BPD and MD co-morbidity, our results showed that patients with BPD and MD comorbidity performed better on RMET accuracy and positive valence task than BPD or MD alone. As such, our findings represent a new informative clinical profile of comorbid BPD and MD. BPD with comorbid MD regarding mental state decoding ability is different from both BPD and MD alone. The RMET positive valence findings are in line with Daros et.al. (2004) meta-analysis of studies investigated facial emotion recognition ability of BPD patients from emotional stimuli at 100% intensity. In that meta-analysis, it was found that BPD with MD have more intact positive emotional facial expression recognition ability than MD alone. One implication of our finding is that more accurate mental state decoding of RMET total accuracy and positive valence scores may represent an important feature of BPD with MD that might be useful for distinguishing patients with BPD with MD patients from those with BPD or MD alone. It is important to note that comorbid MD may reflect the depressive symptoms of BPD at a more severe stage of their clinical trajectory and may not describe major depression as a distinct comorbid diagnostic entity. These results may point to the inability of structured diagnostic interviews based on the DSM-IV system to differentiate between major depressive episode and depressive symptoms of BPD.

BPD patients with co-occurring eating disorder performed better on the test than having a disorder of BPD without it. We collapsed eating disorders in one group because most of the analyzed studies reported them that way. Most of the previous study found intact RMET performance in bulimia nervosa (BN) and RMET impairment in anorexia nervosa (AN) (Russell et al., 2009; Harrison et al., 2009) with the exception of other studies that found that RMET performance of AN was similar to healthy controls (Kenyon et al., 2012; Adenzato et al., 2012), and the one where all subtypes of ED were studied and it was found deficits only in BN and Eating Disorders Not Otherwise Specified (EDNOS; Medina-Pradas et

al., 2012). Although in previous studies there were contrasting findings on RMET performance in different subgroups of eating disorders, our results revealed a subgroup of BPD patients with comorbid eating disorders with a relatively good RMET performance.

Our other important finding is that BPD patients who met DSM-IV criteria for any cluster B or any cluster C personality disorder performed worse on the RMET than having a disorder of BPD without them. Comorbidity of these disorders with BPD is considerable (McGlashan et al, 2000; Grant et al., 2008). There are relatively few studies about RMET performance in personality disorders from these two clusters (Richell et al., 2003; Dolan and Fullam, 2004). Our results point to potential mental state decoding impairment in these personality disorders, but we cannot exclude that the co-occurring symptoms of other personality disorder may reflect the severity of BPD or dysfunction of personality in general and not the presence of distinct diagnostic entities. Future research should carefully consider the contributions of comorbid cluster B and C personality disorders to RMET performance in BPD.

The current study is characterized by some limitations. First, our analysis contained studies that only comprised a healthy comparison group. Further, studies were excluded if patients suffered from personality disorders/mental health disorders not relevant to our study. Studies were also excluded if patients exhibited BPD characteristics but were not diagnosed with respect the DSM-IV (Diagnostic Statistical Manual, 2002). Meta analyses were carefully conducted in order to prevent complications with the calculation of effect sizes on contrasting populations of controls, patients, and different clinical diagnoses. Moderator variable examination was limited as few studies reported education level and ethnicity. Next, the analysis was limited to the inclusion of cross-sectional studies only. Our analysis of the moderator effects of comorbidity may reflect symptom severity rather than multiple morbidity. Finally, our results are limited by a small amount of studies accuracy with co-morbidity results only reported in some of the studies.

5.2 Autobiographical Memory

The present meta-analysis extends the current literature in autobiographical memory functioning in patients with BPD by quantifying the magnitude of autobiographical memory impairment relative to HC participants. We also specified potential moderator variables that may influence autobiographical memory performance in BPD. We found two systematic reviews on BPD (Bech et al., 2015; Van den Broeck, Claes, Pieters, Hermans, & Raes, 2015), but according to our present knowledge, there is no meta-analysis on autobiographical memory functioning in this patient population. Our meta- analysis extends these narrative reviews by quantifying the contradictory results of the previous studies, and by adding further studies into our analysis comprising of 2,024 participants (BPD: n = 1,017, HC: n = 1,007).

We found a medium overall effect size for autobiographical memory impairments in the BPD group. Because this meta-analysis was well powered and not undermined by a significant publication bias, we concluded that autobiographical memory impairment as measured by the AMT distinguishes BPD patients from healthy comparison participants; however, the high heterogeneity across studies pointed to the importance of taking into account the moderating effects of the patients' age, gender, and intelligence.

In the BPD group, the effect size was very large for overgenerality, omission, and specificity, while it was not significant for recall latency. These results regarding the specificity of memories of BPD patients suggest that they responded more with repeated events and with memories that lasted longer than one day to AMT cue words, as well as came up with more overgeneral memories and fewer specific memories in response to cue words in AMT than the HC. Also, the BPD group omitted more answers in AMT than the HC. However, they did not differ from the HC in recall latency when responding to cue words.

Our results indicate that the diagnosis of BPD is associated with significant autobiographical memory impairments. Gender and intelligence did not influence

autobiographical memory performance of the BPD patients. However, age as a moderating variable did influence BPD patients' AM performance. Older BPD patients report more specific autobiographical memories, as memory specificity increases with age in BPD, but not in the HC group. Although these results are in accordance with some previous research, they do however contradict others. Older participants were also found to have better AM performance in a study by Kennedy, Mather, and Carstensen (2004) in healthy females. This may be related to the symptom reduction with age in BPD patients (Gunderson et al., 2011). In other studies, it was found that females consistently recalled more AMs than males, and they were generally faster in recalling (Fujita, Diener, & Sandvick, 1991). However, other studies found no gender differences in AM performance (Kihlstrom & Harackiewicz, 1982; Strongman & Kemp, 1991), except for negative memories, of which women recalled more than men (Ros & Latorre, 2010).

Main findings on impaired AM functioning in BPD patients point to a specific retrieval style characterized by high level of overgenerality, low level of specificity, and more categorical answers to AMT cue words, as well as retrieving more extended and semantic memories to AMT cue words. These results can be conceptualized within the context of a basic model of autobiographical memory: the self-memory system model of Conway and colleagues (Conway & Pleydell-Pearce, 2000).

In the self-memory system model, four broad levels of specificity have been identified: more broad, conceptual themes in the life story, lifetime periods, general events, and event-specific knowledge. Over-general, low-specific memories fit the non-event-specific memory category. In the AMT task, how- ever, the goal is to retrieve event-specific AMs. As opposed to non-event-specific memories, event-specific AMs can be retrieved via two processes: generative and direct retrieval. Generative retrieval—used by the classic AMT—is a top-down process involving the retrieval of desired specific memories. In contrast, direct retrieval results in the retrieval of a specific memory when event-specific knowledge is activated by cues in the environment (rather than retrieved intentionally by the individual) (Sumner, 2012). The Williams and colleagues' (2007) CaR-FA-X model postulates that

overgeneral memory (OGM) results when the generative retrieval search process is aborted prematurely as a result of one or more of the three proposed mechanisms (Williams et al., 2007): capture and rumination (CaR), functional avoidance (FA), and impaired executive control (X) (Sumner, 2012).

5.2.1 Capture.

Our findings on high overgenerality and low specificity among BPD may be explained by the CaR-FA-X model. BPD patients are characterized by a high level of early maladaptive schemas (EMS) (Kellogg, & Young, 2006; Unoka & Richman, 2016) that may capture the memory retrieval at more general levels of memory representation and block the retrieval of specific autobiographical memories.

In Spinhoven, Bockting, Kremers, Schene, and Williams's study (2007), patients with BPD completed the Young Schema Questionnaire (YSQ) and the AMT. In the AMT, BPD patients retrieved fewer specific AMs in response to cue words that thematically matched highly endorsed schemas. These results suggest that an impaired retrieval of specific memories may be the result of certain cues activating generic, higher-order mental representations. Thus, we suggest that early maladaptive schema captured retrieval processes may lead to non-specific and malevolent interpersonal representations in BPD.

5.2.2 Rumination.

It was also found that sadness-related and anger-related ruminations are both characteristic to BPD patients (Selby, Anestis, Bender, & Joiner, 2009). We found only one study (Van den Broeck et al., 2015) that investigated the association between the level of rumination and OGM among BPD patients, and it found that the more severe the BPD patients' depressive symptoms were and the more they ruminated, the less capable they were of retrieving specific AMs. However, when depression severity scores measured by the Beck Depression Inventory-II (BDI; Beck Steer, & Brown, 1996) were patriated out, sadness-related ruminations and memory specificity were no longer significantly related. In addition, the

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association between memory specificity and depressive severity were independent of the state or diagnosis of depression (Van den Broeck et al., 2015). It is a further question whether the association between low level of specificity and more general rumination or anger-related rumination—also specific to BPD patients—could be also be affected by the severity of depression. To further understand the associations between rumination and AM specificity in BPD, we need further research.

5.2.3 Functional Avoidance.

Core BPD symptoms like self-harming behavior, drug and alcohol abuse, different forms of dissociations, and unstable identity have a function of avoiding emotionally painful experiences. These kinds of avoidant behaviors have a short-term advantage of distress reduction, but in the long run they may cause further problems by preventing patients from processing important information and experiences, as well as refraining from rigid attempts to suppress negative cognitions/emotions, which may have paradoxical effects of enhancing the accessibility of these thoughts and/ or the intensity of these emotions (Berking Neacsiu, Comtois, & Linehan, 2009).

Overgenerality and a low level of specificity answers to AMT cue words may play a similar role in avoiding emotionally painful memories. We can speculate that BPD patients who use the aforementioned avoidant strategies may also use more overgeneral memory as an avoidant strategy, and there should therefore be an association between them. Some findings indicate an association between dissociation and more general memories (Jones et al., 1999), while others do not (Kremers et al., 2004; Renneberg et al., 2005). It has been shown that BPD patients who showed the greatest overgeneral retrieval reported the fewest parasuicidal acts (Startup et al., 2001). According to the authors' interpretation of this result, overgeneral autobiographical recall may help protect individuals with BPD from parasuicidal acts by helping them avoid distressing memories

5.2.4 Impaired Executive Control.

During the recalling of a specific AM by the effortful generative retrieval process, generic descriptions are progressively inhibited to reach to a specific event (Conway & Pleydell-Pearce, 2000). In case of insufficient executive control, the process of episodic memory retrieval is prematurely interrupted, leading to the recollection of a general memory (Haque & Conway, 2001; Heeren, Van Broeck, & Philippot, 2009; Williams et al., 2007). It was found that cognitive flexibility and executive control play an important role in specific AM retrieval (Dalgleish et al., 2007; Heeren et al., 2009; Williams & Dritschel, 1992). In addition, cognitive flexibility may partially mediate the impact of mindfulness training on overgeneral (categoric) memories (Heeren at al., 2009). Also, one meta analytic study on BPD found a significant impairment of executive functioning (Unoka & Richman, 2016), while other studies found that better visual (Maurex et al., 2010) and verbal (Reid & Startup, 2010) working memory was associated with higher memory specificity (Spinhoven et al., 2007). Our finding of a high level of omission in the BPD group may also be related to impaired executive functioning. Again, further research is needed to clarify the specific role of executive impairments in overgeneral memory among patients suffering from BPD.

To sum up, there are reasons both for and against thinking that high overgenerality and low specificity among BPD patients may be explained by the CaR-FA-X model. To fully answer this question, further investigation is needed. Studies that investigated the association between overgeneral memory and some elements of the CaR-FA-X model are correlational, and it is a further problem that the elements of the model measured by the questionnaires or rating scales included items not specifically related to autobiographical memory. It is not possible to make a conclusive evaluation of the model from research using such designs. It would be important to conduct studies that include all the elements of the CaR-FA-X model and experimentally manipulate them in an autobiographical memory retrieval context.

One important limitation of the AMT cueing task is that it indexes access to valanced memories without directly indexing memories that are personally important to the individual (Jansari & Parkin, 1996; Rybash & Monaghan, 1999).

To address this limitation, Singer and Moffitt (1991, 1992) developed an approach that indexed "self-defining memories." Self-defining memories are defined as memories that reflect one's identity and are affectively intense, repetitive, and vivid, and comprise enduring concerns about oneself (Singer & Salovey, 1993).

In addition to the use of the CaR-FA-X model, there are other interpretations of our results that point to limitations with our study. The use of medication, early trauma experiences, general symptom severity, severity of depressive symptoms, and PTSD and depression comorbidity could play a role in the level of memory specificity in our BPD sample. We could not test their potential effects because there were not enough studies providing information for calculation of an effect size.

This meta-analysis is limited by several factors. The number of studies included is small, with only some autobiographical domains. Another issue is that although we uncovered an association between autobiographical memory impairments and BPD, the studies included were cross-sectional. To solve the issues around the CaR-Fa-X model and the state or trait nature of BPD and autobiographical memory impairments, a longitudinal follow- through cohort study with repeated measures in both symptoms and cognitive domains and AM is required. Future research should focus on a study design that includes participant groups with a range of severity levels along first and second axis symptom dimensions, detailed reporting of comorbidity, and common measures of autobiographical memory, trauma type, and severity, which would provide a better comparison across groups, as well as a clearer indication of the way in which severity of BPD is related to cognitive impairment.

5.3 Neuropsychological Functioning

The present meta-analysis extends the current literature on neuropsychological functioning in patients with BPD by quantifying the magnitude of cognitive impairment relative to healthy comparison subjects. We also specified potential moderator variables that may influence neuropsychological performance

in BPD. The previous meta-analysis on neuropsychological functioning (Ruocco, 2005) consisted of 10 studies comprising 488 participants (BPD: N = 225, control: N = 263); our meta-analysis extends this by adding additional studies and participants.

We found a large overall effect size for global cognition in BPD group. Because this meta-analysis was well powered, not undermined by a significant publication bias, we concluded that cognitive impairment distinguishes BPD patients from healthy comparison participants; however, the high heterogeneity across studies pointed to the importance of taking into account the moderating effects of the patients, their parents' education, and the presence of co-morbid disorders. The magnitude of the observed cognitive impairments in the subjects was large for decision making, memory and executive functioning; medium for global cognition; and small for visuospatial abilities, attention, and verbal intelligence and processing speed. Our results indicate that the diagnosis of BPD is associated with significant cognitive impairment and that co-morbid disorders lead to further deterioration of the cognitive functioning.

This overall effect size was heterogeneous and was significantly influenced by some moderating variables including neuropsychological domain, education and co-morbid diagnosis. Compared with controls, BPD subjects showed deficits in the decision making, memory, executive functioning, processing speed, verbal intelligence, visuospatial abilities, and attention, while no differences were observed in the overall intellectual ability (i.e., full-scale IQ), non-verbal intelligence, and language domains. Age, gender, sex, race and anti-depressant treatment did not influence cognitive performance of the BPD subjects, while BPD patients with more education and with parents of a higher educational level had better neuropsychological functioning. In addition, samples with a higher percentage of BPD with co-morbid personality disorders, major depression, eating disorders, any substance abuse disorders, in order of deficit, performed worse than with a lower percentage of such disorders, however anxiety disorders and PTSD co- morbidity did not affect the cognitive performance of the BPD group.

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This impaired cognitive functioning in BPD patients is in line with underlying anomalies in the brain structure, function, and neurochemistry. The large magnitude of effect sizes for executive functioning and decision making, as well as the small deficit for attention, is consistent with the previously found structural abnormalities (Irle, Lange, & Sachsse, 2005; Van Elst et al., 2003), resting state and task related dysfunctioning and altered connectivity among brain regions (Minzenberg, Fan, New, Tang, & Siever, 2007) and hypometabolism (De La Fuente et al., 1997; Juengling et al., 2003; Soloff et al., 2003) of the prefrontal brain in BPD patients. Impaired executive functioning, that is difficulty in mental set shifting, information updating and monitoring, inhibition of prepotent responses and planning may be related to BPD symptoms like identity diffusion, impulsivity, self-injury, emotional lability, irritability, poor self-control, lack of self-direction, chronic feelings of emptiness, dissociative symptoms, rigidity and difficulty in shifting attention. Strategies that target to teach executive function skills like cognitive remediation programs have consistently been reported to improve neurocognitive deficits in patients with schizophrenia (Wykes and Spaulding, 2011) and with depression (Elgamal et al., 2007) would be beneficial for BPD patients too. It is a further question on how evidence-based psychotherapies of BPD affects the executive functions of the patients.

The largest deficit was found in the area of decision making that points to the dysfunction of orbitofrontal cortex (OFC) and the anterior cingulate cortex (ACC) (Carrasco et al., 2012; Rusch et al., 2010). Impaired decision making may be related to BPD patients' difficulties in withholding responses in order to gain larger rewards, for example, in impulsive spending, sex, substance abuse and binge eating and this delay aversion may be related to impaired processing of punishment cues in the face of high reward situations that may reflect an imbalance between the appetitive and aversive motivational states when they are excited by the available reinforcement signals. They have this problem even in tasks when they received explicit cognitive feedback regarding the negative consequences of their behavior (Svaldi et al., 2012). In the treatment of individuals with BPD it is not enough to explicitly explain the deleterious long term consequences of their

impulsive behaviors, what they already know anyway, but to develop new strategies that combine the inhibition of immediate satisfaction of their needs in the face of high reward situations and the ability to switch their attention from the present situation to a temporally extended viewpoint and to build up mentally available future attachment and/or autonomy related rewards that help overcome the immediate effects of the present urge. These aims are all targeted by the evidence-based treatments of BPD: dialectical behavior, mentalization based, transference focused and schema therapy (Bateman & Fonagy, 1999; Linehan, 1994; Yeomans et al., 2002; Young, 2000). The large magnitude of effect size for memory is in line with the decreased volume of hippocampus (Ruocco, Amirthavasagam, & Zakzanis, 2012), and the fronto-limbic dysregulation of it (Minzenberg et al., 2007; Salavert et al., 2011) among BPD patients and may underlie the BPD symptoms of identity disturbances and dissociations. The small effect sizes for visuospatial abilities may relate to parietal dysfunctions in BPD patients (Zago & Tzourio-Mazoyer, 2002). Slower cognitive processing speeds are likely to depend on white matter damage and aberrant network connectivity, which constrain the communication and coordination among cortical nodes of brain wide networks (Carrasco et al., 2012; Minzenberg et al., 2007; Turken et al., 2008).

Measurement of BPD patients' neuropsychological functioning may play an important role in therapy planning. For example, in a randomized control study of four treatment condition of BPD it was found that higher executive control and visual memory performances predict the number of weeks in treatment, which was used as a treatment completion measure (Fertuck et al., 2012). Neuropsychological impairments identified in our study may be also good candidates for predictors of treatment effectiveness in future research.

Our secondary aim was to address the role played by a number of potential moderators influencing cognitive performance in BPD patients. Age, gender, sex and race of participants did not have significant effect on the cognitive performance of BPD patients. Maybe the delayed age-related maturation of the BPD's brain (Houston, Ceballos, Hesselbrock, & Bauer, 2005; New et al., 2013) and the age-related symptom reduction of BPD patients (Zanarini, Gunderson, & Frankenburg,

1989) wipe out the general findings of age-related worsening of cognitive performance (Voineskos et al., 2012). The result that both sexes had similar level of cognitive performance is consistent with previous studies among those that reported equal cognitive performance among sexes. Other socio-demographic variables with significant differences were that patients with higher education and patients with more educated parents were performing better than patients with lower education and patients with less educated parents. These results are consistent with previous studies that reported better cognitive performance among more educated participants among healthy populations and among different patient groups (Acevedo and Loewenstein, 2007). Better cognitive performance among subjects with more educated parents were found previously in many studies and explained by different potential mechanism as richer vocabulary, higher SES status, hereditary mechanism (Ardila et al., 2005). In addition, we found no evidence that exposure to anti-depressant medication influenced cognitive functioning in BPD patients.

Theoretically, medication with dopaminergic and/or noradrenergic effects may improve, but medications with anti-cholinergic and sedative side effects impair cognitive function. In our analysis, different types of anti-depressants were pooled together. The results of previous studies on the anti-depressants' effects on cognitive performance were inconclusive (Biringer et al., 2009).

Finally, we addressed the question of whether the cognitive performance was associated with other psychiatric illnesses, which commonly co-occur with BPD. Results of these analyses revealed that cognitive deficits were related to co-morbid personality disorders, major depression, eating disorders and any substance abuse disorders and were unrelated to co-morbid anxiety disorders and PTSD. We should take it into consideration that high co-morbidity rate in BPD may be an artifact of the categorical system of DSM and it may just reflect that more symptom dimensions are affected. To solve this categorical system generated ‘co-morbidity problem’ researchers developed dimensional constructs like Kernberg's personality organization model (Kernberg, 1985), dimensional model for diagnosing personality disorders listed in the DSM-5 (APA, 2013) chapter called

Emerging Measures and Models or the Research Domain Criteria (RDoC) project launched by NIMH (Insel et al., 2010). Symptom dimension-based research on neuropsychological disturbances could reveal more specific associations between symptoms and neuropsychological functioning. However, the authors of the original papers followed the DSM-IV concept of diagnoses that were determined by structured clinical interviews. Most of the paper reported percent of co-morbid diagnostic categories and did not report symptom severity, or symptom dimensions so we could not analyze their associations with neuropsychological domains. Our results are partly consistent with the idea that the more severe the case, that is the presence of more symptoms and/or more symptom dimensions leads to higher level of cognitive impairment (McDermott and Ebmeier, 2009), although the presence of co-morbid anxiety disorders and PTSD did not affect the cognitive performance and this suggests that a simple explanation as more symptom more severe cognitive impairment may not be sufficient. Furthermore, when we assessed the differences among each neuropsychological domain, we found specific associations between different co-morbid disorders and specific neuropsychological domain.

Specifically, BPD patients with any co-occurring dramatic cluster personality disorder were more impaired in decision making and executive functioning than samples performed worse than patients with only BPD and less percentage of co-occurring cluster B disorders. These results are consistent with the previous results of meta-analyses that found executive function deficit (Morgan and Lilienfeld, 2000) and hot and cold executive function deficit (De Brito et al., 2013) among anti-social personality disorder patients, neuropsychological impairment and mild difficulties with reversal learning (Ruocco et al., 2009) among dramatic personalities. The impairment in mental set shifting, information updating and monitoring, inhibition of prepotent responses and planning and the difficulties in inhibition of immediate satisfaction of their needs in the face of high reward situations in order to gain larger rewards may be a general diathesis for dramatic personality disorders. A dimensional assessment of personality disorder may also help clarify the relationship between symptom domains and neuropsychological performance, to avoid the difficulties imposed by categorical

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diagnoses, which often yield significant diagnostic heterogeneity within a personality disorder diagnosis (Ruocco et al., 2009). Furthermore, BPD samples with a higher percentage of co-occurring major depression were more impaired in memory than BPD samples with a lower percentage of MD. In a recently published meta-analysis it was found that cognitive impairment in the areas of executive functions, attention and memory is a core feature of depression even in remission (Rock et al., 2014). Our results show that co-morbid MD worsened the performance only in the memory domain of BPD patients. Currently depressed BPD patients worsened by memory performance may impair the effectiveness of therapeutic interventions which need high memory loads. Further- more, samples with a higher percentage of any co-occurring eating dis- order were more impaired in executive functioning and memory than the samples which had a higher percentage of any co-occurring eating disorder with BPD and performed worse than with a lower percentage. This result is not surprising as previous meta-analyses in eating disorders indicated that anorexia nervosa was impaired in executive functioning and bulimia across different domains of memory and impulsivity and both types of eating disorder are impaired in decision making (Zakzanis et al., 2010). Furthermore, samples with a higher percentage of any co-occurring eating disorder were more impaired in memory, visuospatial abilities, and VIQ, however, they were better than BPD with a lower percentage of co-occurring any substance abuse disorder in processing speed. The results of a previous meta-analysis on the specific and generalized effects of substance abuse on neuropsychological performance that identified common and substance specific neuropsychological impairments (Fernández-Serrano et al., 2011) would be helpful in the interpretation of our results. However, the fact that most of the BPD studies did not detail the types of co-occurring substance abuse and that most substance abuser BPD patients simultaneously use and abuse more than one substance, creates a great challenge in interpreting neuropsychological findings in BPD patients with any co-occurring sub- stance use. To sum up we may say that the identified neuropsychological impairments generally constitute a severe diathesis (Tyrer et al., 2007) that makes BPD patients vulnerable to a broad range of symptoms, and it is a further question whether these symptoms that

phenomenologically fits to one or other DSM-IV syndromes are the same as the original disorders and are subject to the same type of causal explanations and respond similarly to the same type of causal interventions.

This meta-analysis is limited by several factors. Although we have uncovered an association between neuropsychological deficits and BPD, the studies included were cross-sectional. To solve the issues around the state or trait nature of personality disorder and co-occurring first axis symptoms and different cognitive impairments, a longitudinal follow-through cohort study with repeat measure in both symptoms and cognitive domains is required. Future research should focus on a study design which includes participant groups with a range of severity levels along first and second axis symptom dimensions and uses common measures, which would provide a better comparison across groups and a clearer indication of the way in which severity of BPD is related to cognitive impairment. The number of studies included in the analysis was relatively small with only 27; some cognitive domains (decision making, language and VIQ) were only covered in two studies. In total, 53 papers were originally identified through the literature search. However, a large number had to be excluded. Reasons included the absence of clinical diagnosis, absence of healthy or any control groups, clinical group mixed with BPD and anti-social personality disorder, little statistical information for calculating effect size and clinical groups with patients having a mean age under 18.

Moreover, though meta-regression is helpful in drawing results and conclusions, meta-regression explores whether covariates clarify any of the heterogeneity of treatment effects between studies, and therefore is not reasonable to assume that all the heterogeneity is explained. The findings from meta-regressions are observational and have a weaker interpretation than the causal relationships consequent from randomized evaluations. Our meta-analyses accounted for this using a random effects model, but in meta-regression it is across several varied studies and does not have the advantage of randomization to reinforce a causal understanding. Though we accounted for the potential of gender and age being unmatched by using a random effects model and by selecting the

input data as matched groups, post data only, it is important to note that some studies considered age and gender matched control groups, and some did not. Taken together, it is important to note that these are observational in nature.

In conclusion, this meta-analysis indicates that, there are consistent impairments in cognitive functioning in BPD patients as compared with matched controls. Significant deficits are observed in the decision making, memory, executive functioning, processing speed, verbal intelligence, visuospatial abilities, and attention while no differences were observed in the overall intellectual ability (i.e., full-scale IQ), non-verbal intelligence and language domains. Age, sex, race and anti-depressant treatment did not influence cognitive performance of the BPD subjects, while BPD patients with more education and with parents of a higher educational level had better neuropsychological functioning. In addition, samples with a higher percentage of co-morbid personality disorders, major depression, eating disorders, any substance abuse disorders respectively performed worse than patients with a lower percentage of such co-morbid disorders; however, anxiety disorders and PTSD co-morbidity did not affect the cognitive performance of the BPD group. These results contribute to understanding the BPD psychopathology supporting neuropsychological deficits as among the core features of the disorder and co-occurring disorders affect the cognitive performance of BPD patients.

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6. Conclusions

Taken together, the meta-analyses have extended our knowledge of neurocognitive functioning in BPD. We can use these findings to create new, specific treatment modalities as these findings may be related to symptom dimensions. In addition, we could create new research paradigms to further understand this. Clinicians can now learn how to interact and treat those with borderline personality disorder more efficiently by targeting and treating the deficits found and highlighting in treatment the impairments. Since there are several symptom dimensions of BPD, it could be helpful to target the specific neurocognitive deficits to symptoms and treating accordingly.

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7. Summary

Borderline personality disorder (BPD) is characterized by an intricate cognitive profile with impairments in some areas of cognitive functioning and other areas having enhanced abilities in neuropsychological functioning. Despite this well-known phenomenon that has been evidenced in the literature and research studies, there have been few quantitative analyses that have investigated subsets of the cognitive profile in BPD, but none have looked at specific moderator variables such as demographic and clinical variables. Taken this together, the dissertation looks at the three most studied core cognitive elements within in BPD, a) mental state decoding, or the ability to attribute mental states to social partners from perceivable social information b) neuropsychological functioning, and c) autobiographical memory or the memory system that contains personal memories and knowledge of self-related past events. In addition, the dissertation considers the influence of co-occurring disorders, due to their high comorbidity with BPD. In order to do such, three separate meta-analytic reviews are performed which highlight the deficits and enhanced cognitive profile in BPD. Moreover, the dissertation considers moderator variables such as domains of cognitive profile, continuous and categorical variables (i.e., age, percentage of sample male, race, education), and co-occurring disorders as to how they relate to BPD. Results are instrumental in informing treatment and further research. Conclusions can be applied to developing further modalities in research, teaching, and treatment.

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8. Összefoglalás (Summary in Hungarian)

A borderline szemelyiségzavart (borderline personality disorder, BPD) egy összetett kognitív profil jellemzi, több területen visszamaradott kognitív teljesítménnyel, de emellett számos átlagon felüli neuropszichológiai képességgel. Bár ezen karakterjegyek kísérleti alátámasztást nyertek, és mára általánostan elfogadottak a szakirodalomban, kevés kvantitatív vizsgálat vett figyelembe demográfiai, valamint klinikai moderátorváltozókat. Ezen disszertáció a BPD-ben három leggyakrabban vizsgált kognitív modul szerepét tárgyalja: a) mentalizációs képesség, vagyis mások mentális állapotaira való következtetés képessége észlelhető szociális információk alapján, b) neuropszichológiai funkciók, c) autobiografikus memória, vagyis azon rendszer, amely a szelffel kapcsolatos emlékeket tartalmazza. Emellett a disszertáció más, a BPD-vel együtt előforduló zavarok hatását is vizsgálja. Három metaanalízisben összevetjük BPD-vel diagnosztizált egyének neuropszichológiai karakterisztikáit egeszséges kontrollcsoportéival. Potenciális moderátorváltozókat elemeztünk: kor, nemek aránya, iskolázottság, rassz, együtt-előforduló zavarok. Az eredmények hangsúlyozzák a kognitív funkciók klinikai relevanciáját borderline személyiségzavar esetén, valamint a demográfiai és klinikai moderátorváltozók figyelembevételének fontosságát jövőbeli vizsgálatok tervezésénél.

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10. Bibliography of the candidate's publications

10.1 Related to thesis

1. Beran, E., **Richman, M.J.,** & Unoka, Z. (2019) Autobiographical memory in borderline personality disorder: a meta-analysis interpreted in terms of the CaR-FA-X model. *Journal of Personality Disorders,* 33(6), 818-831, 2019, Impact Factor: 2.970
2. Unoka, Z., & **Richman, M.J.** (2016) Neurocognitive deficits in BPD patients and the moderator effects of co-occurring mental disorders: a meta-analysis. *Clinical Psychology Review, 44,* 1-12, Impact Factor: 9.904
3. **Richman, M.J., &** Unoka, Z. (2015) Mental state decoding impairment in major depression and borderline personality disorder: meta-analysis. *British Journal of Psychiatry*. *207*, 483–489. Impact Factor: 7.233

10.2 Not related to thesis

4. Buchman-Wildbaum, T., **Richman, M. J.,** Váradi, E., Schmelowszky, Á., Griffiths, M. D., Demetrovics, Z., & Urbán, R. (2020). Perceived loss among people living with mental disorders: Validation of the personal loss from mental illness scale. *Comprehensive psychiatry*, *96*, 152146. Impact Factor: 2.586
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10.2.1 Popular Science Articles

15. Agoston, C., Urban, R, **Richman, M.J.,** & Demetrovics, Z (2018, July) Caffeine Use Disorder – Should We Consider It as a Mental Disorder? *Science Trends*, Humans. doi.org/10.31988/SciTrends.21601

10.2.2 Published Conference Abstracts

16. Demetrovics, Z. & **Richman, M.J**. (2018) Behavioral addictions: Research directions and developments. Keynote. *Journal of Behavioral Addictions*: Suppl. 1 pp. 2-2., 1
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10.2.3 Thesis and Summer Projects

20. **Richman, M.J.,** (2018) Rumination in borderline personality disorder: meta-analysis. *Eötvös Loránd University MA.* Supervisor: Zsolt Demetrovics, DSc, ELTE Institute of Psychology
21. **Richman, M.J.,** (2014) Subtypes of identity disturbance and their association with the course of borderline personality disorder over 16 years of prospective

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10.2.4 Manuscripts in Review/ Revision/ Prep

24. Maleki, G., Zabihzadeh., A., **Richman, M.J.,** Demetrovics, Z., & Mohammadnejad, F. (accepted) Decoding and reasoning mental states in major depression and social anxiety disorder: Moderating role of attachment. *BMC Psychiatry*
25. Kun, B., Takács Z.K., **Richman, M.J.,** Griffiths, M.D., Demetrovics Z. (in review) Work addiction and personality: A meta-analytic study. *Journal of Behavioral Addictions,*
26. **Richman, M.J.,** Unoka, Z., Dudas, R., & Demetrovics, Z., (in preparation) Rumination in borderline personality disorder: Meta-analysis
27. Buchman-Wildbaum, T., Unoka, Z., Vizin, G., Dudas, R., Demetrovics, Z., & **Richman, M.J.** (in revision) Shame in borderline personality disorder: Meta-analysis. *Journal of Personality Disorders*

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