THE RELATIVE STRENGTH OF THE RELATIONSHIP BETWEEN HYPERMENTALIZING AND BORDERLINE PERSONALITY DISORDER IN THE CONTEXT OF OTHER DISORDERS: A META-ANALYTIC REVIEW

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ABSTRACT

A hypermentalizing deficit, or a tendency to over attribute mental states to others, has been identified for Borderline Personality Disorder (BPD). However, associations between other disorders and hypermentalizing call the specificity of this deficit to BPD into question. The aims of the current study were to use meta-analytic methods to 1) evaluate the relative strength of the hypermentalizing deficit associated with BPD in the context of other disorders, and 2) assess the impact of moderators on the relationship between hypermentalizing and psychopathology. The Movie for the Assessment of Social Cognition (MASC), an ecologically valid experimental task, was used as the measure of hypermentalizing. Meta-analyses and moderator analyses were performed with 10 studies (n = 1,471) investigating the relationship between BPD and hypermentalizing and 30 studies (n = 3,339) investigating the relationship between non-BPD psychopathology and hypermentalizing. Results indicate that the extant literature does not support the specificity of hypermentalizing to BPD as defined by a significantly stronger association for BPD (r = 0.26; 95% CI = [0.12, 0.39]) than for non-BPD psychopathology (r = 0.22; 95% CI = [0.11, 0.31]). However, overlap between BPD and the general factors of psychopathology and personality pathology indicates the possibility that the association between non-BPD psychopathology and hypermentalizing may be explained by this overlap; BPD features present in other psychopathology may be behind the association, even if the construct of BPD is completely subsumed by the general factors. Additionally, age significantly moderated the association between non-BPD psychopathology and hypermentalizing, while percent female moderated the association between BPD and hypermentalizing. Concerns

regarding lack of race reporting, predominately Caucasian samples, and the MASC's potential bias against non-Caucasian individuals limit the generalizability of current results to non-Caucasian racial groups.

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Introduction

Mentalizing and BPD

Borderline Personality Disorder (BPD) is a serious mental disorder characterized by a pattern of chaotic relationships, identity disturbance, difficulties in emotion regulation, and impulsivity. This disorder is highly prevalent, especially among those receiving services for physical and mental health. While prevalence for BPD in the general population has been recorded at 1.4-5.9% (Grant et al., 2008; Lenzenweger et al., 2007), prevalence among clinical populations is much higher. These prevalence rates range from over 20% in psychiatric outpatient samples (Korzekwa et al., 2008) to over 40% in high users of inpatient settings (Comtois & Carmel, 2016) and over 60% among patients in forensic services (Blackburn et al., 1990; Ruiter & Greeven, 2000). Additionally, people with BPD are at a highly increased risk for suicide: approximately 50 times that of the general population (Skodol, Gunderson, Pfohl, et al., 2002).

Impairment in interpersonal functioning is a key characteristic of BPD. These interpersonal difficulties manifest as conflict throughout the social network of individuals with BPD, which contain more terminated relationships, such as former romantic partners and cut-off friendships, than the social networks of individuals without BPD (Clifton et al., 2007). In existing romantic relationships, BPD symptoms predict greater levels of dysfunction, including stress, conflict, partner dissatisfaction, and abuse (Daley et al., 1999). These interpersonal deficits translate to increased frequency of conflict with parents, siblings, and friends (Skodol, Gunderson, McGlashan, et al., 2002). The centrality of interpersonal

problems to BPD is demonstrated through the nine criteria for BPD in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), two of which explicitly relate to interpersonal difficulties: (1) frantic efforts to avoid real or imagined abandonment, and (2) a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation. However, most of the remaining criteria have implicit implications for interpersonal relationships (American Psychiatric Association, 2013; Sharp, 2014). For example, suicidal behavior, gestures, and threats (criterion 5) of individuals with BPD often have interpersonal triggers (Brodsky et al., 2006). Because of the centrality, and frequent lethality, of these interpersonal problems for individuals with BPD, social cognition has been identified as a potentially important factor for understanding the disorder (Sharp & Fonagy, 2008).

Research supports the idea that social cognitive deficits may underlie the interpersonal problems associated with BPD, but mixed results suggest that such deficits are only apparent when the social-cognitive task is complicated or highly emotionally arousing. For example, meta-analytic studies of emotion recognition in BPD indicate that individuals with BPD do not show impairment compared to healthy controls in recognizing emotions for negative or positive faces, but when uncertainty is introduced via neutral or ambiguous faces, individuals with BPD are less accurate than healthy controls (Daros et al., 2013; Mitchell et al., 2014; Richman & Unoka, 2015). Individuals with BPD also demonstrate less accurate emotion recognition than healthy controls when complexity is introduced, such as when faces are merged with prosodic information, but show no impairment when the stimulus is simplified by presenting the faces and prosodic information separately (Minzenberg et al., 2006). This same pattern emerges in other social cognitive constructs, including empathy and

trust, indicating that the social cognitive impairment associated with BPD is only apparent in the face of highly complicated or emotionally charged tasks (Sharp & Vanwoerden, 2015). In addition to being triggered by complex tasks, the social cognitive impairment characteristic of individuals with BPD seems to be marked by an over-attribution of intentions and thoughts to others.

Inaccurate social cognition by individuals with BPD tends to be characterized by going beyond the evidence and making inaccurate assumptions (Sharp & Vanwoerden, 2015). In emotion recognition tasks with neutral or ambiguous faces, individuals with BPD demonstrate a negative response bias (Mitchell et al., 2014). A similar negative bias was found in impressions of characters in movies; individuals with BPD ascribed more negative qualities to the characters than did healthy controls (Arntz & Veen, 2001). This negative bias seems to generalize to mental representations; studies using projective assessment techniques have demonstrated that individuals with BPD have more malevolent object worlds than healthy controls and tend to attribute motivation to others in illogical ways (Westen et al., 1990). In summary, the social cognitive deficits associated with BPD seem to only manifest in the face of complex and emotionally arousing stimulus and are characterized by an overattribution of mental states beyond what there is evidence for. In the interest of having a coherent theoretical framework by which to understand these findings, Sharp suggested that the construct of hypermentalizing may be helpful (Sharp, 2014; Sharp & Vanwoerden, 2015).

Sharp and colleagues put forward a model to understand the unique social cognitive problems encountered by those with BPD. This model combines mentalizing and social-information processing theory to understand the nature of the social cognitive deficits characteristic of BPD. Mentalization refers to one's imaginative capacity to reflect on the

mental states of oneself and others and is a central mechanism behind much of social cognition and interpersonal interactions (Allen et al., 2008). Optimal mentalizing is a balance between several polarities, one of which is especially relevant here—automatic versus controlled mentalizing. Automatic mentalizing happens quickly and without attention, while controlled mentalizing is slow and requires intention (Satpute & Lieberman, 2006). Optimal mentalizing requires flexibility across this pole contingent upon the demands of the situation. Sharp's model posits that individuals with BPD struggle to integrate across this pole and instead rely on one or the other in isolation. This integration is not necessary in situations that are not emotionally taxing but is vital in situations that are complicated or emotionally arousing. As a result, in such situations, individuals with BPD demonstrate mentalizing that is not context-specific. In other words, automatic and controlled mentalizing occur at inappropriate times. In this state, mentalizing is not reduced, but rather increased; an over attribution of mental states far beyond what there is evidence for, or hypermentalizing, dominates (Sharp & Vanwoerden, 2015). This is in line with the previously discussed evidence that suggests that for individuals with BPD, social cognitive deficits occur in the context of complex or emotionally arousing stimulus and are characterized by going beyond the evidence and making assumptions. Hypermentalizing is described as "making excessively convoluted inferences on the basis of others' social cues" (Fonagy et al., 2015). For example, if a friend appears to be sad, an individual who is hypermentalizing may think "she hates spending time with me." The tendency of individuals with BPD to hypermentalize has been examined in studies using the Movie for the Assessment of Social Cognition (MASC; Dziobek et al., 2006). The MASC is a widely used and ecologically valid measure of mentalizing that categorizes errors in mentalizing into those indicating no mentalizing,

those indicating reduced mentalizing (or hypomentalizing), and those indicating hypermentalizing. Studies using the MASC have provided strong evidence for a hypermentalizing impairment associated with BPD.

In support of a hypermentalizing impairment for individuals with BPD, many studies using the MASC have found that individuals with BPD demonstrate more hypermentalizing thoughts than healthy controls (Andreou et al., 2015; Goueli et al., 2019; Normann-Eide et al., 2019; Quek et al., 2018) and psychiatric controls (Normann-Eide et al., 2019; Sharp et al., 2013). Furthermore, hypermentalizing has been shown to be positively associated with BPD traits (Duval et al., 2018; Quek et al., 2018; Sharp et al., 2016; Somma et al., 2019). Only one study has not found this increase in hypermentalizing compared to psychiatric controls (Vanwoerden, 2016), one compared to healthy controls (Ha, 2016), and only two have not found a significant positive association between hypermentalizing and BPD traits (Fossati et al., 2017, 2018). Additionally, no study has found any difference in reduced mentalizing between individuals with BPD and healthy controls.

Mentalizing and Other Psychopathology

While the association between hypermentalizing and BPD is well supported, other disorders have demonstrated associations with hypermentalizing on the MASC. Individuals with Autism Spectrum Disorder (ASD; Isaksson et al., 2019; Lahera et al., 2014; Martinez et al., 2017), schizophrenia (Martinez et al., 2017; Montag et al., 2011; Vaskinn et al., 2015), Attention-Deficit/Hyperactivity Disorder (ADHD; Isaksson et al., 2019), social anxiety disorder (SAD; Hezel & McNally, 2014; Washburn et al., 2016), psychogenic nonepileptic seizures (Schönenberg et al., 2015), and persistent somatoform pain disorder (Schönenberg et al., 2014) have also demonstrated increased hypermentalizing compared to healthy controls.

In contrast, several studies have not supported the link between hypermentalizing and these disorders. For instance, three studies fail to demonstrate differences in hypermentalizing in healthy controls compared with individuals with schizophrenia (Andreou et al., 2015; Engelstad et al., 2019; Peyroux et al., 2019), two fail to demonstrate differences in hypermentalizing between healthy controls and individuals with social anxiety (Ballespí et al., 2019; Lenton-Brym et al., 2018), and one fails to demonstrate differences with healthy controls compared to individuals with ADHD (Abdel-Hamid et al., 2019). Additionally, while individuals with BPD tend to only have increases in hypermentalizing scores, hypomentalizing scores are also elevated for individuals with ASD (Isaksson et al., 2019; Lahera et al., 2014; Martinez et al., 2017) and schizophrenia (Andreou et al., 2015; Engelstad et al., 2019; Montag et al., 2011; Peyroux et al., 2019; Vaskinn et al., 2015). Further, hypermentalizing on the MASC is associated with affective components of psychopathy (Sharp & Vanwoerden, 2014), conduct disorder assessed dimensionally (Fossati et al., 2017), internalizing symptoms not specific to any disorder (Gambin et al., 2015), and suicidal ideation and attempts (Hatkevich et al., 2019). This evidence calls the specificity of the hypermentalizing impairment to BPD into question.

Potential Moderators

To understand the differences in mentalizing ability across these studies, we also need to consider other factors that may impact mentalizing performance, specifically age, gender, and race. A previous meta-analysis of studies using the MASC found that in adults, age negatively predicts total score and women perform significantly better than men; however, the relationships between these variables and hypermentalizing score were not investigated (Wacker et al., 2017). Other studies have found that men make more hypermentalizing errors

on the MASC than women do in adult clinical samples (Fossati et al., 2017) and adolescent community samples (Poznyak et al., 2019; Taylor et al., 2013). The results for age are mixed, with one study finding that younger adults make more hypermentalizing errors than older adults in a community sample (Lecce et al., 2018), while others have found no relationship between hypermentalizing and age for clinical adults (Fossati et al., 2017) or community adolescents (Poznyak et al., 2019; Taylor et al., 2013).

No study has yet investigated the impact of race on MASC scores, but other studies have found that neural response in social cognitive networks decrease when the individual being mentalized is of a different race than the individual who is mentalizing them (Avenanti et al., 2010; Xu et al., 2009). Because all four characters in the MASC are Caucasian, it is possible that the race of the participant might impact their performance on the MASC such that Caucasian participants perform better than non-Caucasian participants due to an in-group advantage. Because these three participant characteristics may impact results of the MASC, it is important to account for the resultant variance in understanding differences among studies.

Current Study

With these considerations in mind, the goal of the current study was to use a meta-analytic approach to test the specificity of the hypermentalizing impairment to BPD. While the specificity has been tested in individual studies using psychiatric controls (Normann-Eide et al., 2019; Sharp et al., 2011, 2013), there are several advantages to using meta-analytic methods. Because meta-analyses combine samples from multiple studies, the generalizability of results is greater, mixed results can be resolved by the consideration of multiple moderators, and publication bias can be accounted for. While meta-analyses have clarified the relationship between BPD and specific social cognitive abilities, such as emotion

recognition (Mitchell et al., 2014; Richman & Unoka, 2015) and theory of mind (Nemeth et al., 2018) these meta-analyses do not fully investigate the hypermentalizing impairment associated with BPD. Those investigating emotion recognition did not use tasks with sufficient complexity to elicit the social cognitive deficits associated with BPD, nor were they able to distinguish between hypermentalizing and hypomentalizing, as they included tasks that were not designed to differentiate the two. Further, no meta-analyses have compared the performance of individuals with BPD to that of individuals with other disorders. Thus, the specificity of these deficits to BPD has not yet been investigated meta-analytically. Recently, it has been suggested that BPD represents the typical features common to all personality pathology in the form of maladaptive self-and interpersonal function (Sharp, 2019; Sharp et al., 2015). Therefore, if meta analyses were to identify hypermentalizing as unique to BPD, it is possible that hypermentalizing indicates personality pathology in general.

To address the above gaps, a literature review was conducted to identify studies that (1) investigate the association of hypermentalizing with any form of psychopathology, and (2) use the MASC, which was selected because it is a widely used task that separates suboptimal mentalizing into hyper- and hypomentalizing and is relatively ecologically valid and emotionally salient. Additionally, because hypermentalizing is theorized to both distinguish individuals with BPD from those with other psychopathology as well as be associated with the severity of BPD traits, we included studies that investigate the association of mentalizing performance with the severity of psychopathological traits as well as those that compare the mentalizing performance of a group with psychopathology to that of a

healthy controls group. Meta-analytic techniques allow us to synthesize the findings of these studies and draw conclusions about their significance.

Meta-analysis allows for effect sizes from many studies to be either combined or compared. When combining effect sizes, the effect size of the association between two variables is extracted from each study, weighted according to its standard error and the estimated population variance, and included in the calculation of a weighted average. This weighted average represents the overall effect size of the association between these two variables according to the extant literature. On the other hand, comparing effect sizes allows for heterogeneity among these effect sizes to be accounted for. To compare effect sizes, moderators—or variables that may account for higher or lower effect sizes—are included in the model. In other words, moderators are variables that impact the strength of the association between the two variables in question. To evaluate the impact of these moderators on the effect size, meta-regression is used; moderators are conceptualized as predictors of the effect size. A significant model indicates that the moderators adequately explain the heterogeneity among effect sizes, while a significant moderator indicates that that moderator explains significant heterogeneity in the model.

For the current study, the relationship between psychopathology and hypermentalizing were investigated meta-analytically. For studies that investigate the group differences in mentalizing between healthy controls and a psychopathological group, the effect size represents the difference in mentalizing between these two groups. For studies that investigate the association between severity of psychopathology and mentalizing, the effect size represents the correlation between these two variables. The association between psychopathology and hypermentalizing was investigated separately for BPD and for non-

BPD psychopathology. Because there is a high comorbidity between BPD and other PDs, BPD is potentially represented in these other disorders (Sharp et al., 2015). Therefore, according to this viewpoint, it would not be appropriate to include other personality pathology in the non-BPD analyses and doing so may diminish the difference in effect size. However, as this is still a topic of debate, we conducted the non-BPD analyses both with and without PDs included. When PDs are included, these analyses are referred to as non-BPD, and when PDs are excluded, these analyses are referred to as non-PD. Additionally, moderator analysis were conducted. To explain heterogeneity among these effect sizes, sample characteristics (age and gender) were entered as moderators. The aims of the current study are as follows:

1. *Evaluate the relative strength of the hypermentalizing deficit associated with BPD in the context of other disorders.* The first aim was to use meta-analytic techniques to support previous literature that states that hypermentalizing is more strongly associated with BPD than with any other disorder. We investigated this aim using individual meta-analyses for BPD, non-BPD psychopathology, and non-PD psychopathology. Effect sizes for the association between hypermentalizing and each type of psychopathology were computed, and the effect size for BPD was compared to that for non-BPD psychopathology and non-PD psychopathology. Therefore, we hypothesized that BPD would be more strongly associated with hypermentalizing than either non-BPD psychopathology or non-PD psychopathology.
2. *Assess the impact of moderators on the relationship between hypermentalizing and psychopathology.* The second aim is to understand the influence of potential sources of heterogeneity; specifically, the sample characteristics of age, gender, and race. To

investigate this aim, we conducted moderator analyses on each category (BPD, non-BPD, and non-PD). The models and individual moderators were evaluated for significance. We had no a priori hypotheses because previous literature is inconclusive.

Methods

Measure of Mentalizing

The MASC (Dziobek et al., 2006) is a video-based assessment that evaluates everyday use of mentalizing. Participants watch a 15-minute film about four people getting together for dinner. The film is stopped at 45 points, during which the participants answer a multiple-choice question regarding a character's thoughts and feelings. Participants choose from 4 answer options: an accurate choice, a "hypermentalizing" choice that ascribes mental states with little to no evidence, a "hypomentalizing" choice that only partially matches the accurate mental states, and a "no mentalizing" choice unrelated to internal states. The number of times each type of answer was selected is calculated, resulting in 4 mentalizing scores: one indicating the frequency of accurate mentalizing and three indicating the frequency of each type of error. Additionally, four control questions unrelated to mentalizing are asked. The control score can be calculated by adding together the number of control items answered correctly and indicates general comprehension and attention. The MASC has been found to be a reliable tool for assessing mentalizing deficits in healthy adults (Dziobek et al., 2006), as well as adults and adolescents with psychopathology (Fossati et al., 2018). Only studies using the MASC have been included in the present study for several reasons: 1) as an experimental task, it does not rely on self-report; 2) it is a an ecologically valid task with sufficient complexity to require integration across the automatic vs. controlled pole, and thus

expose the deficits expected to be associated with BPD; and 3) it utilizes the framework of mentalizing that separates suboptimal mentalizing into hypomentalizing and hypermentalizing, allowing for the differences between these two types of responses to be detected.

Literature Search Procedures

We conducted a comprehensive literature review by searching PsycINFO, Social Sciences Citation Index (SSCI), PubMed, and ProQuest Dissertations and Theses databases. The search was conducted for articles published from January 2006 to the December 2019. The start point was selected by considering the year that the MASC was published. To find all potentially relevant articles, searches were conducted using the term "movie for the assessment of social cognition." Manuscripts that were identified as having cited the MASC validation paper were also reviewed. All articles generated by the literature search were then be assessed to determine eligibility for inclusion in the meta-analysis. Additionally, reference lists of included studies were searched for further relevant studies.

Inclusion and Exclusion Criteria

Studies met the following inclusion criteria: (a) the study utilized the MASC; (b) the study reported either a group comparison of hypermentalizing scores between individuals with a psychological diagnosis and healthy controls or an association between hypermentalizing scores and a continuous measure of a psychological disorder; (c) the article was written in English; (d) an effect size was reported or sufficient information was included to calculate an effect size; (e) the article presented data that were not reported by another study included in the review.

Coding Procedure

A coding form was created to systematically record relevant information from each article. Study characteristics included on the coding form were (a) report information (full bibliographic reference); and (b) publication type (peer reviewed journal article, dissertation, or unpublished manuscript). Coded sample characteristics included (a) total N; (b) mean age; (c) percent female; and (d) percent Caucasian. Information related to psychopathology and its measurement included (a) specific DSM disorder; (b) type of comparison (correlation, group difference, or both). Finally, Pearson's correlation, *r*, was used as the effect size for all statistical analyses. Effect size was recorded for the association between psychopathology and MASC hypermentalizing score. A second coder coded 20% of the sample for reliability analyses.

Data analytic strategy

Analyses were conducted in Excel and in R using the dmetar, meta, and metafor packages. For all aims, random-effects modeling was used for meta-analytic calculations. Random-effects modeling was chosen because it assumes that we have a random sample of studies that are distributed around a mean population effect size, which allows for results to be generalized to a population of studies. On the other hand, fixed-effects modeling, which assumes that effect sizes represent a "true effect size" plus or minus some error, merely describes the current data and does not allow for generalization. Pearson's correlation, *r*, was recorded for the association between hypermentalizing and psychopathology in each study as the measure of effect size. For studies in which effect size is reported in another metric or effect size is not reported but sufficient information is provided to calculate an effect size, effect sizes were calculated or transformed to *r* using standard procedures. Positive effect

sizes represent a positive association between hypermentalizing and psychopathological symptoms, or instances where a psychopathological group had higher hypermentalizing scores than a healthy control group. Negative effect sizes represent a negative association between hypermentalizing and psychopathological symptoms, or instances where a psychopathological group had lower hypermentalizing scores than a healthy controls group. For studies that report multiple effect sizes (i.e. multiple measures of psychopathology), pooled effect size and standard error were calculated. Effect sizes were then converted to Z_r using Fisher's transformation to control for the skewed distribution of *r*. Results of the meta-analysis were converted back to *r* when reported for ease of interpretation. Heterogeneity was calculated for each diagnostic category, and publication bias was investigated using Egger's linear regression. Egger's linear regression tests for funnel plot asymmetry, which is indicative of publication bias.

To evaluate the hypotheses associated with Aim 1, separate meta-analyses for the association between hypermentalizing scores and psychopathology were conducted for each diagnostic category (BPD, non-BPD, and non-PD). First, effect sizes for the association between hypermentalizing and each category were calculated, resulting in three individual pooled effect size estimates: one for BPD, one for non-BPD, and one for non-PD. Then, these effect sizes were compared using their confidence intervals; non-overlapping confidence intervals indicate statistically significant differences between two effect sizes. To address Aim 2, moderator analysis was also conducted for all three categories. Moderators were excluded for analyses in which fewer than 10 studies reported the statistic, as advised by Fu et al (2011). When not excluded, the following moderators were evaluated individually: (a) mean age; (b) percent female; and (c) percent Caucasian.

Results

Descriptive information

Figure 1 shows the process of selecting studies from those identified in the literature search. Of 400 unique articles identified, 36 were selected as meeting the inclusion criteria. Of the 36, 5 reported an effect size for only the association between BPD and hypermentalizing, 26 reported an effect size for only the association between a non-BPD psychopathology and hypermentalizing, and 5 studies reported both an effect size for the association between BPD and hypermentalizing and an effect size for the association between a non-BPD psychopathology and hypermentalizing. Of the 31 studies reporting associations between non-BPD psychopathology and hypermentalizing, 5 report on a personality pathology. Thus, 10 studies are included in the BPD analyses, 31 are included in the non-BPD analyses, and 26 are included in the non-PD analyses. Studies included in the BPD analyses are presented in Table 1, and studies included in the non-BPD and non-PD analyses are presented in Table 2. 20% of the studies were coded by a second rater to determine coder reliability. Among the relevant coded variables (N, % female, % Caucasian, age mean, and effect size), reliability was almost perfect (all *r*s > .9). Additionally, discrepant codes were re-coded until perfect agreement was reached.

The 10 studies and 1,471 participants included in the BPD analyses are presented in Table 1. All studies are peer-reviewed journal articles, with the exception of one dissertation (Ha, 2016). Mean sample age is 21.99. Mean age by study ranges from 15.03-38.58; however, there is a gap in this range from 17.7-27.9, as studies tend to include only adults or only adolescents. 65.4% of the pooled sample is female, which ranges from 57.5%-100% by study. No study included more men than women. Only 2 studies reported racial demographic

information of the participants (20%), so race was not included as a moderator in the BPD analyses for aim 2. Both studies in which race was reported had similarly high proportions of Caucasian participants (range: 91.8-92.5).

The non-BPD analyses include 31 studies and 3,339 participants and are presented in Table 2. All studies are peer-reviewed journal articles, with the exception of one thesis (Divilbiss, 2009) and one dissertation (Wastler, 2019). Mean sample age is 26.89, which ranges from 15.37-46.75 by study. 54% of the combined sample is female, which ranges from 0-100% by study. Like studies included in BPD analyses, race was only reported in 6 (19.3%) of articles and thus was not included in moderator analyses for aim 2. For studies where race was reported, 82.61% of participants were Caucasian (range: 64.67%-92.5%). Highly represented psychological disorders include schizophrenia (10 studies), Major Depressive Disorder (MDD; 6 studies), SAD (4 studies), ASD (4 studies), ADHD (4 studies), Bipolar disorder (2 studies), and GAD (2 studies).

The 26 studies and 2,666 participants included in the non-PD analyses are also presented in Table 2; those excluded from these analyses are marked with an asterisk. With these articles excluded, mean age for the sample is 25.99 (range: 15.37-46.75), 55.38% were female (range: 25-100), and 80.74% (range: 64.67-88.2) of participants in studies reporting race were Caucasian. Again, race was excluded from moderator analyses in Aim 2 due to low reporting (5 studies; 19.2%).

Relative strength of hypermentalizing deficits (Aim 1)

Meta-analytic results for all three sets of studies (BPD, non-BPD, and non-PD) are reported in Table 3. For studies reporting the association between BPD and hypermentalizing, random-effects modeling indicated a small and significant positive

association between hypermentalizing and BPD (see Figure 2; r = 0.26, SE = .07, 95% CI = [0.12, 0.39]). Heterogeneity among the included effect sizes was significant ($Q(9) = 60.39$, $p<.01$) and high in magnitude (I^2 = 85.1%). This high degree of heterogeneity between studies supports our decision to use random-effects models and conduct moderator analyses Egger's test for funnel plot asymmetry was performed to evaluate the possibility of publication bias; non-significant results (B_0 = 1.57, 95% CI [-0.73, 6.08], t = 0.69, p = .51) do not provide support for the presence of publication bias.

For studies reporting the association between non-BPD psychopathology and hypermentalizing, random-effects modeling indicated a small and significant positive association between hypermentalizing and psychopathology (see Figure 3; r = 0.22, SE = .05, 95% CI = [0.11, 0.31]). Heterogeneity among the effect sizes was also significant ($Q(30)$ = 161.36, $p<.01$) and high in magnitude (I^2 = 81.4%). To address the possibility of publication bias, Egger's linear regression was performed. Results were non-significant (B_0 = 2.31, 95% CI [-0.44, 5.05], t = 1.65, p = .11), which does not indicate the presence of publication bias. When studies including personality pathology were excluded, the association between hypermentalizing and non-personality psychopathology was small, positive, and significant (see Figure 4; r = 0.25, SE = .05, 95% CI = [0.16, 0.34]). There was still significant ($Q(25)$ = 145.76, $p<.01$) and large (I^2 = 82.8%) heterogeneity. Egger's test was again non-significant (B_0 = 2.21, 95% CI [-0.73, 5.15], t = 1.44, p = .16).

Overlapping confidence intervals for the effect size of the association between BPD and hypermentalizing (r = 0.26; 95% CI = [0.12, 0.39]) and for the effect size of the association between non-BPD psychopathology and hypermentalizing (r = 0.22; 95% CI = [0.11, 0.31]) do not indicate a significant difference between these associations. When

personality pathology is excluded from the non-BPD analyses, (r = 0.25; 95% CI = [0.12, 0.39]), the confidence interval still overlaps with that of the BPD analyses, indicating that even when personality pathology is excluded, there is not a significant difference in the associations between psychopathology and hypermentalizing for BPD versus non-BPD psychopathology.

Moderator analysis (Aim 2)

Results of moderator analyses are presented in Table 3. For studies included in the BPD analyses, when percent female was entered as a moderator, the model predicted a significant amount of heterogeneity among the effect sizes ($Q_{Model}(1) = 37.18$, $p < .01$). While a significant amount of residual heterogeneity remained ($Q_{Residual}(8) = 17.23$, $p = .03$), the model explained 96.8% of the variance. The predicted association (r) between hypermentalizing and BPD is 0.11 for 60% female and 0.47 for 80% female. When age was entered as a moderator, the model did not predict a significant amount of heterogeneity among the effect sizes ($Q_{Model}(1) = 2.64$, $p < .01$).

For studies included in the non-BPD analyses, the model including percent female as a moderator did not predict significant heterogeneity ($Q_{Model}(1) = 0.00$, $p = .95$). The model including mean age as a moderator predicted significant heterogeneity among the effect sizes ($Q_{Model}(1) = 5.81$, $p = .02$), accounting for 19.61% of the variance. There was still a significant amount of residual heterogeneity unexplained by the moderators ($Q_{Residual}(28) = 143.20$, $p < .01$). The predicted association (r) between hypermentalizing and psychopathology is 0.11 at a mean sample age of 20 and 0.29 at 35.

When PDs are excluded, there is still not significant heterogeneity predicted by the model including percent female ($Q_{Model}(1) = 0.35$, $p = .55$). The model including mean age

still predicted significant heterogeneity ($Q_{Model}(1) = 9.05$, $p < .01$) and had significant residual heterogeneity ($Q_{Residual}(23) = 108.62$, $p < .01$). The model accounted for 33.00% of the variance. Predicted values for the association between hypermentalizing and psychopathology (*r)* were 0.12 for a mean sample age of 20 and 0.36 at 35.

Discussion

Review and discussion of main findings

The aims of the current study were to 1) evaluate the relative strength of the hypermentalizing deficit associated with BPD in the context of other disorders and 2) assess the impact of moderators on the relationship between hypermentalizing and psychopathology. Both aims were assessed meta-analytically by combining and comparing studies that evaluated an effect size for the association between psychopathology and hypermentalizing score on the MASC.

Specificity of hypermentalizing to BPD. Our hypotheses that BPD would have a stronger association with hypermentalizing than non-BPD psychopathology were not supported; while a small, positive association between hypermentalizing and BPD was confirmed via meta-analysis, a small, positive association between hypermentalizing and other psychopathology was also found via meta-analysis. Given the similarity in magnitude and the overlap in confidence intervals for these two associations, the current study did not support the hypothesis that the association between BPD and hypermentalizing would be stronger than the association between other psychopathology and hypermentalizing, even when other personality pathology is excluded. In other words, we did not find evidence for the specificity of hypermentalizing to BPD.

While no evidence was found in the current study to support the specificity of hypermentalizing to BPD, comparing the strengths of the associations is only one way to test for specificity. The current study does not control for the presence of BPD features among non-BPD psychopathology. Thus, it is possible that the strength of the resultant association between non-BPD psychopathology and hypermentalizing is partially or entirely attributable to BPD features present in other psychopathology. This possibility is supported by evidence that BPD is strongly related to both the p-factor (the common factor of psychopathology, potentially representing severity or risk of developing any form of psychopathology; Caspi et al., 2014; Gluschkoff et al., 2020) and the general factor of personality pathology (suggested to represent severity, affective dysregulation, or Criterion A of the alternative model; Sharp et al., 2015; Wright et al., 2016). Thus, BPD features may be common to all psychological disorders and may account for the association between non-BPD psychopathology and hypermentalizing. In order to investigate this possibility, future research comparing BPD and non-BPD psychopathology in their association with hypermentalizing should control for BPD features present in other psychopathology. If after controlling for BPD features, there is no longer an association between other psychopathology and hypermentalizing, hypermentalizing may still be specific to BPD. This possibility would suggest that, while hypermentalizing may be present in individuals with other disorders, it can be explained by BPD features. This would support the model of hypermentalizing put forth by Sharp et al (2014), which suggests that BPD features and hypermentalizing are recursively related to one another, but would emphasize an understanding of this model that accounts for dimensionality and comorbidity.

The idea that BPD features account for hypermentalizing associated with the general factor presupposes the existence of the BPD construct; however, another interpretation of the close associations among BPD, the p-factor, and the general factor of personality pathology is that BPD may be completely subsumed by one or both general factors (Gluschkoff et al., 2020; Sharp et al., 2015). In other words, it is possible that some combination of two or all three of these constructs represent the same latent variable. To determine the relationship between this factor or group of factors and hypermentalizing compared to specific factors associated with other psychopathology, it is necessary to first conduct latent variable modeling to derive and identify the presence of general and specific factors. Once identified, the associations between these factors and hypermentalizing can be investigated. Should hypermentalizing prove uniquely associated with the BPD factor, regardless of whether that factor represents BPD uniquely, or whether it additionally represents a general factor of psychopathology and/or personality pathology, hypermentalizing may still be specific to the BPD construct. The hypothetical finding of an association between hypermentalizing and one or both general factors may be important in our understanding of the nature of these factors. For example, it would provide support for the possibility that these factors represent self-other dysfunction (Criterion A of the alternative model) and/or affective dysregulation, as both of these are implicated in the model of hypermentalizing put forth by Sharp et al. (2014). Additionally, it would suggest that hypermentalizing may be an important etiological factor behind risk for and severity of psychopathology in general.

Moderator analyses. We did not have any a priori hypotheses for the moderator analyses, but important differences between significant moderators for BPD and non-BPD psychopathology were noted. Percent female, but not mean age, was found to significantly

moderate the association between BPD and hypermentalizing; the higher proportion female, the stronger the association between BPD and hypermentalizing. In other words, for females, the association between BPD and hypermentalizing is stronger than for males. On the other hand, mean age, but not percent female, was found to significantly moderate the association between other psychopathology and hypermentalizing; the older the sample, the stronger the association between psychopathology and hypermentalizing. In other words, as age increases, psychopathology and hypermentalizing become more strongly associated.

The finding that the association between BPD and hypermentalizing is stronger for samples with a higher percentage of women may indicate that the strength of this association is stronger for men than for women, but an insufficient body of research and a possible third variable limit the conclusiveness of this finding. Past research has indicated that men are more likely to hypermentalize than women (Fossati et al., 2017; Poznyak et al., 2019; Taylor et al., 2013); however, the current finding suggests that this relationship may be influenced by BPD symptoms. An increase in BPD symptoms for women may lead to a greater increase in hypermentalizing than the same increase in BPD symptoms for men. One possible explanation for this moderating effect is gender differences in stress sensitivity (Sharp, 2014). Past research has indicated that females have greater levels of stress sensitivity than males (Oldehinkel & Bouma, 2011). Thus, it is possible that for females, the same increase in BPD symptoms leads to a larger increase in level of perceived stress in the context of complex social interactions. With an increased level of stress, integration across the automatic and controlled poles of mentalizing is increasingly impaired, leading to an increased tendency to hypermentalize (Sharp & Vanwoerden, 2015).

This moderating effect of gender on the association between BPD and hypermentalizing is qualified by a few limitations. First, the extant literature does not include any studies with fewer than 57% female. Thus, generalizations of these results to samples with more men than women are not strongly supported. Additionally, moderators may stand in for a third variable which is the true moderator. In this case, a likely possibility is setting; men with BPD are less likely to use pharmacotherapy and psychotherapy services despite a similar level of distress, whereas rates of hospitalization are similar among men and women with BPD (Sansone & Sansone, 2011), thus samples from psychotherapy settings may overrepresent women. Therefore, it is possible that the true moderator is the setting; among individuals at lower acuity settings, the association between BPD and hypermentalizing may be stronger than the association among individuals at higher acuity settings. However, there is insufficient evidence to determine if the force behind this moderation is gender, setting, or another third variable. Future research should investigate the relationship between BPD, gender, and hypermentalizing, especially in samples with higher percentages of men.

The finding that the relationship between psychopathology and hypermentalizing becomes stronger as age increases may be accounted for via a developmental psychopathology lens. It is important to note that this finding should not be generalized outside of the range of ages included in this meta-analysis (ages 15-46). Over the course of development from adolescence to adulthood, typically developing individuals experience a decrease in hypermentalizing (Lecce et al., 2018). This decrease may not occur for individuals with psychopathology (Fossati et al., 2017). Thus, as mentalizing develops over the course of adolescence and young adulthood, mentalizing deficits in individuals with psychopathology may become more pronounced in contrast with their more typically

developing peers, resulting in an association between psychopathology and hypermentalizing that increases with age. This finding is limited by the cross-sectional nature of the studies included in these analyses, as well as of the studies investigating the relationship between hypermentalizing and age. To confirm the typical and psychopathological trajectories of the development of hypermentalizing, future studies using longitudinal data are necessary. As with all moderators, the strength of this finding is additionally limited by the possibility that age stands in for the effect of a third variable that is truly behind the effect.

General limitations and recommendations for future research

Extant literature. Meta-analytic studies are limited by the available empirical basis. The biggest limitation of the current literature is treatment of race. Racial demographic information was rarely reported, and when it was, samples were predominately Caucasian. Lack of diversity in sampling limits the generalizability of these results to non-Caucasian racial groups, and lack of race reporting prohibited the inclusion of race as a moderator, meaning that potential differences in the association between psychopathology and hypermentalizing due to race could not be examined. We recommend a standard of inclusion of racial demographics in reports of psychological research. Additionally, future research should include more diverse samples and investigate the associations between race, psychopathology, and hypermentalizing.

Other noteworthy gaps in the extant literature include a lack of BPD studies with less than 57% female or with mean ages between 17.7 and 27.9. These gaps, in addition to differences in overall mean age and percent female between the BPD and non-BPD studies call into question the validity of comparisons between the two. Given the significance of age as a moderator for non-BPD and percent female as a moderator for BPD, differences in

overall levels of these two demographics indicate that comparisons between the two may not be equivalent. Methods to account for this lack of equivalence in future research are discussed in the following section.

Finally, two outliers potentially skew the estimations of the mean effect sizes: one in the BPD category (Goueli et al., 2019) and one in the non-BPD/PD categories (Maurage et al., 2011). These outliers may positively skew estimates of mean effect sizes. However, given the presence of outliers for all diagnostic categories, we do not anticipate that this potential skew has impacted comparisons made between these categories.

Dependence. We have already discussed the implications of dependence due to overlap between BPD and the general factor of psychopathology, but these analyses are also impacted by dependence due to the inclusion of some studies in both BPD and non-BPD analyses. This means that some groups of participants are included in both sets of analyses, for example. Because traditional meta-analytic techniques assume that effect sizes are independent, comparisons between BPD and non-BPD results using these techniques should be interpreted with caution. To account for this dependency as well as the differences in demographic characteristics discussed in the previous section, future research should include multilevel meta-analysis. Multilevel meta-analysis allows for dependence to be modeled by grouping outcomes within studies (Moeyaert et al., 2017). Additionally, these analyses could include BPD and non-BPD studies in the same model, allowing for control of mean-level differences in demographics using tests of moderation and interaction.

Publication bias. While we found no evidence of publication bias, according to some estimates, the number of included studies is too small to be sufficiently powered to detect all types of publication bias using Egger's linear regression, especially in the BPD group (Sterne & Egger, 2005). However, because the significant heterogeneity of the sample ruled out other

tests of publication bias, such as failsafe N, Egger's is the most appropriate test for this body of literature. Additionally, given that many studies did not present hypermentalizing as the main outcome, we estimate only a minor risk of undetected publication bias in this literature.

MASC. Because the current meta-analysis is based on data using only used one measure of mentalizing, we are limited by the properties of that measure. The MASC was chosen because it is uniquely a commonly used, complex, ecologically valid, experimental task that separates suboptimal mentalizing into hypermentalizing and hypomentalizing. Thus, it is consistent with the framework of mentalizing previously used to detect mentalizing deficits in BPD. Despite these strengths, it should be noted that the MASC represents only one theory of mentalizing and may be biased against non-Caucasian participants due to the entirely Caucasian cast.

Conclusions

The current study indicates that the extant literature does not support the specificity of hypermentalizing to BPD as defined by a significantly stronger association. However, overlap between BPD the general factor of psychopathology, and the general factor of personality pathology indicates the possibility that the association between non-BPD psychopathology and hypermentalizing may be explained by this overlap; BPD features present in other psychopathology may be behind the association, even if the construct of BPD is completely subsumed by the general factors. Findings that gender moderated the association between BPD and hypermentalizing and that age moderated the association between general psychopathology and mentalizing require further research to clarify their implications; however, gender may stand-in for a third variable (e.g. setting), while age differences in association strength may be explained using a developmental framework.

Additionally, significant concerns regarding lack of race reporting, predominately Caucasian samples, and the MASC's potential bias against non-Caucasian individuals limit the generalizability of current results to non-Caucasian racial groups.

Tables and Figures

Figure 1. Identification process for eligible studies

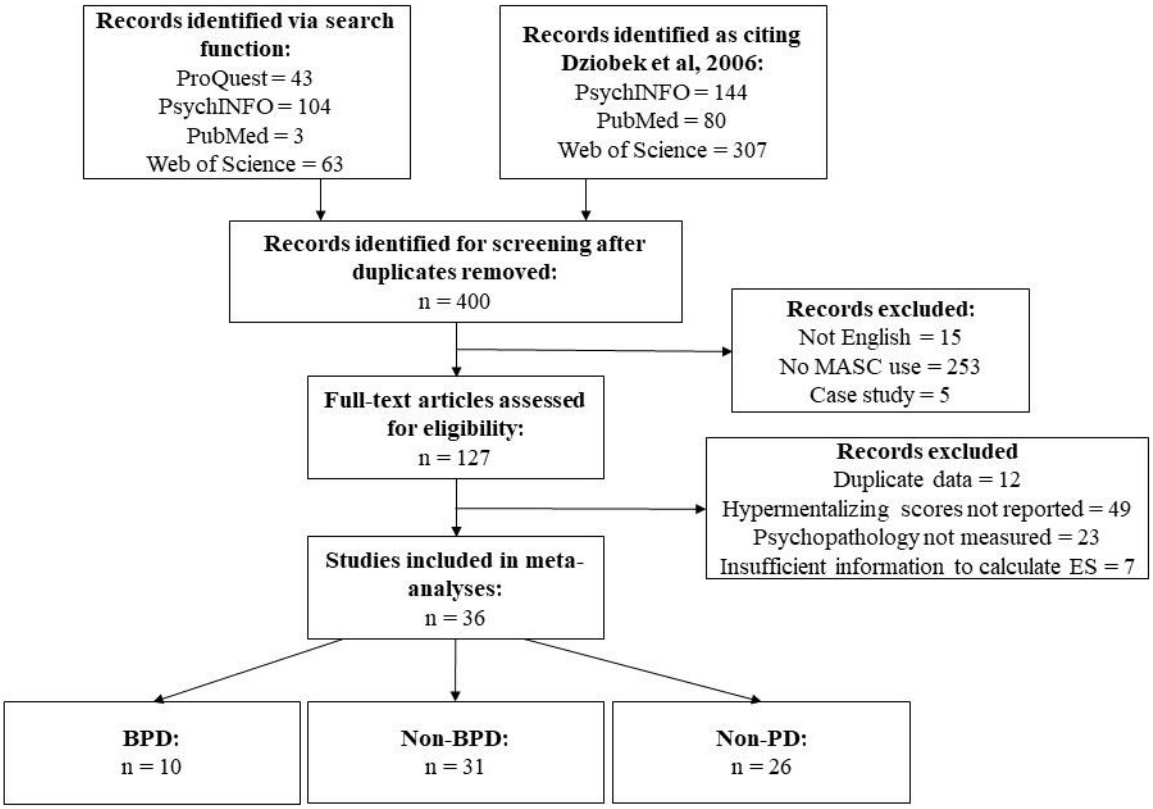


Table 1. Descriptive information and effect sizes for studies included in the BPD analyses.

Study	N	Mean age	% female	% Caucasian	Effect size (*r*)
(Andreou et al., 2015)	82	31.28	64.5	NR	0.284
(Duval et al., 2018)	150	17.94	66.4	92.5	0.210
(Fossati et al., 2017)	181	38.58	57.5	NR	-0.030
(Fossati et al., 2018)	59	37.02	64.4	NR	0.020
(Goueli et al., 2019)	60	17.7	100	NR	0.794
(Ha, 2016)	40	15.03	78	NR	0.036
(Normann-Eide et al., 2019)	158	27.9	61.9	NR	0.198
(Quek et al., 2018)	51	15.39	84.33	NR	0.426
(Sharp et al., 2016)	259	15.42	63.1	91.8	0.239
(Somma et al., 2019)	431	16.76	63.1	NR	0.239

Table 2. Descriptive information and effect sizes for studies included in the non-BPD analyses.

Study	N	Mean age	% female	% Caucasian	Effect size (*r*)	Type of psychopathology
(Abdel-Hamid et al., 2019)	60	35.16	50	NR	0.225	ADHD
(Andreou et al., 2015)	74	31.28	64.5	NR	0.248	SCH
(Ballespí et al., 2019)	113	21.1	85.6	85	0.138	SAD
(Brockmeyer et al., 2016)	50	24.14	100	NR	0.239	Anorexia
(Divilbiss, 2009)	52	NR	65.5	87.9	0.214	SCH
(Duval et al., 2018)*	150	16.94	66.4	92.5	0.085	Narcissistic PD
(Engelstad et al., 2019)	125	32.81	26	NR	0.254	SCH
(Fossati et al., 2017)*	181	38.58	57.5	NR	0.056	PDs
(Fossati et al., 2018)*	59	37.02	64.4	NR	0.054	PDs
(Hatkevich et al., 2019)	391	15.37	62.7	88.2	0.117	MDD, GAD, ADHD
(Hezel & McNally, 2014)	80	23.3	76.25	NR	0.314	SAD
(Isaksson et al., 2019)	192	19.39	52	NR	0.275	ASD, ADHD, NDDs
(Kocsis-Bogár et al., 2017)	86	23.6	72	NR	0.045	SCH
(Lahera et al., 2014)	48	22.47	25	NR	0.361	ASD
(Lenton-Brym et al., 2018)	110	19.73	66.37	NR	0.005	SAD
(Martinez et al., 2017)	75	23.22	17.33	NR	0.399	ASD, SCH
(Maurage et al., 2011)	64	46.75	48.44	NR	0.918	Alcohol Use Disorder
(Montag et al., 2010)	58	41.85	60.3	NR	0.201	Bipolar I, MDD
(Montag et al., 2011)	160	38.75	45	NR	0.300	SCH
(Newbury-Helps et al., 2017)*	125	34.7	0	NR	-0.082	Antisocial PD
(Normann-Eide et al., 2019)*	158	27.9	61.9	NR	0.178	PDs
(Peyroux et al., 2019)	100	30.4	28.3	NR	0.057	SCH
(Poznyak et al., 2019)	89	15.43	60.7	NR	0.078	GAD, MDD, SOM, SCH, ADHD, CD, ASD
(Preller et al., 2014)	168	30.03	28.6	NR	0.266	Cocaine Use Disorder
(Santos et al., 2017)	62	40.87	54.8	NR	0.240	Bipolar
(Schönenberg et al., 2014)	38	46.63	100	NR	0.430	Persistent Somatoform Pain Disorder
(Schönenberg et al., 2015)	30	32.27	80	NR	0.363	Psychogenic Nonepileptic Seizures
(Vaskinn et al., 2015)	162	29.19	38.9	NR	0.121	SCH
(Washburn et al., 2016)	119	19.28	68.07	64.71	0.063	MDD, SAD
(Wastler, 2019)	116	19.09	70.69	64.67	0.123	SCH, MDD
(Wolkenstein et al., 2011)	44	36.5	56.82	NR	0.059	MDD

Note: *Excluded in the non-PD analyses; ADHD=Attention Deficit/Hyperactivity Disorder, SCH=Schizophrenia, PD=Personality Disorder, MDD=Major Depressive Disorder, GAD=Generalized Anxiety Disorder, NDD=general Neurodevelopmental Disorders, ASD=Autism Spectrum Disorder, SOM=Somatic disorders, CD=Conduct Disorder, SAD=Social Anxiety Disorder

Table 3. Summary table of meta-analyses of associations between hypermentalizing and psychopathology

	BPD	Non-BPD	Non-PD
Random-effects model			
Effect size *r*	0.26***	0.22***	.25***
95% CI	[0.12, 0.39]	[0.13, 0.32]	[0.16, 0.34]
Heterogeneity			
Q(df)	60.36 (9)***	161.36(30)***	145.76(25)***
I^2	85.1%	81.4%	82.8%
Moderator effects			
Q_{Age}(df)	2.64(1)	5.81(1)*	9.05(1)**
Q_{Gender}(df)	37.18(1)***	0.00(1)	0.35(1)

Note: * p<.05, **p<.01, ***p<.001

Figure 2. Forest plot for random-effects meta-analysis of BPD and hypermentalizing

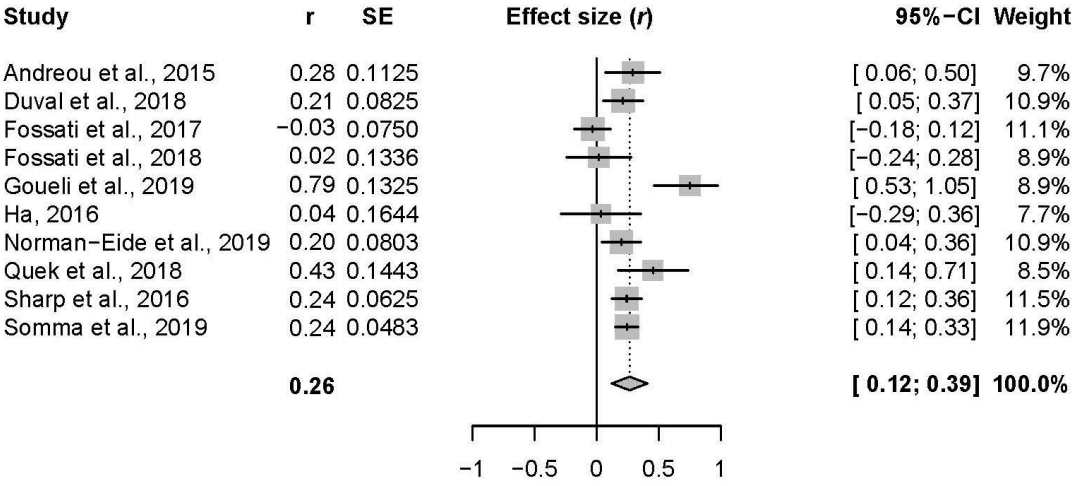


Figure 3. Forest plot for random-effects meta-analysis of non- BPD psychopathology and hypermentalizing

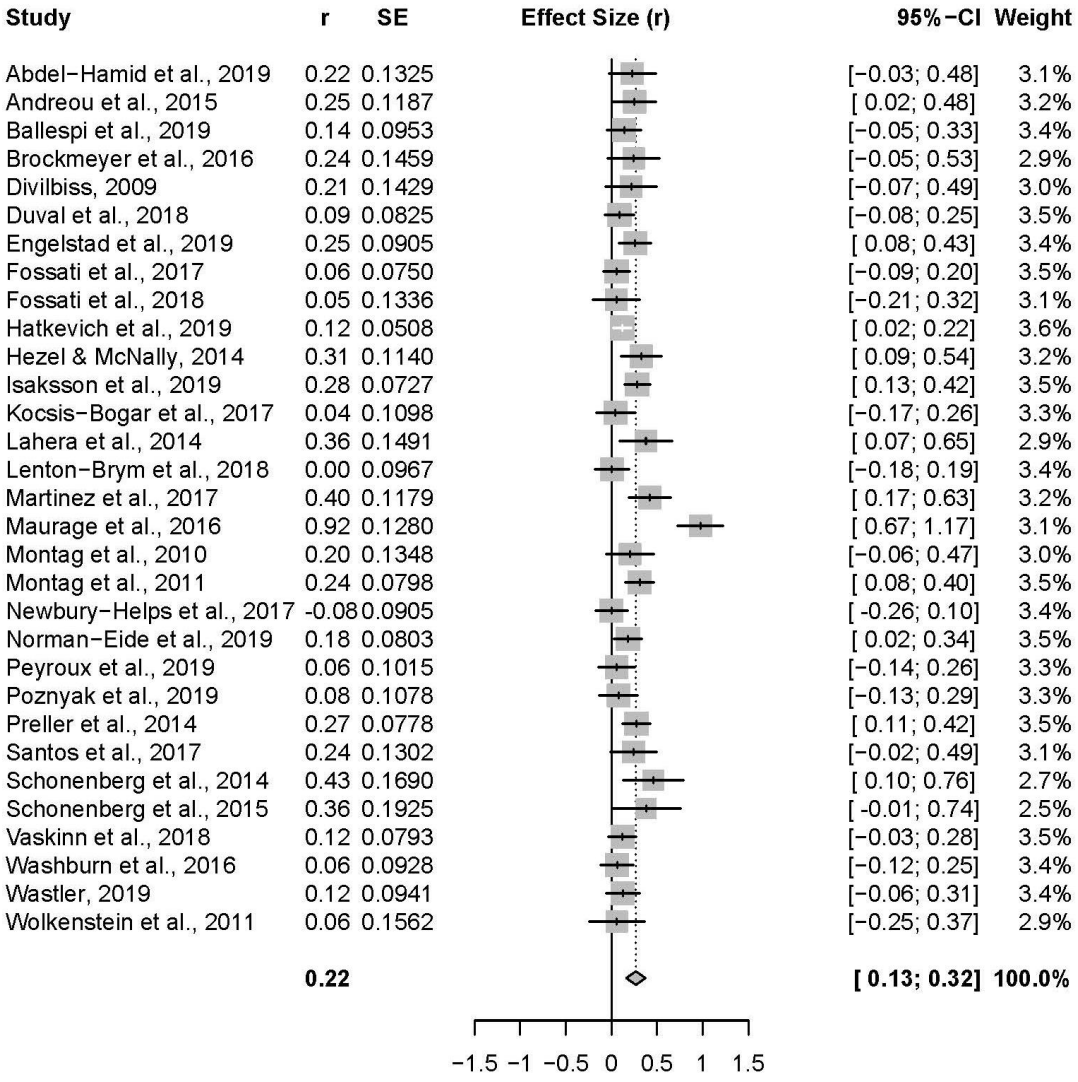
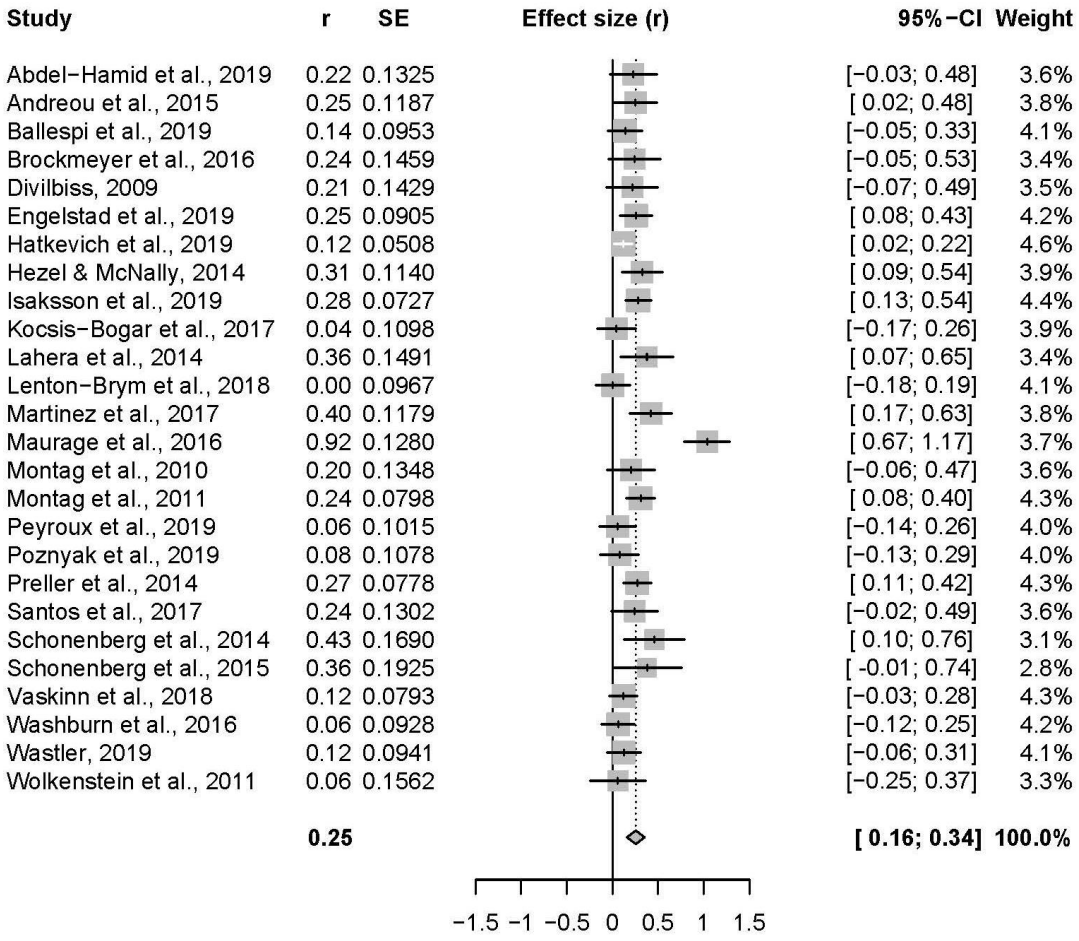


Figure 4. Forest plot for random-effects meta-analysis of non-PD psychopathology and hypermentalizing



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