Personality Pathology and Cognitive Aging: The Role of Interpersonal Stress

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WASHINGTON UNIVERSITY IN ST. LOUIS

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Personality Pathology and Cognitive Aging: The Role of Interpersonal Stress
by
Patrick J. Cruitt

A dissertation presented to
The Graduate School
of Washington University in
partial fulfillment of the
requirements for the degree
of Doctor of Philosophy

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Patrick J. Cruitt

Washington University in St. Louis

August 2021
Dedicated to Molly Cruitt, whose love and support made this endeavor possible.
ABSTRACT OF THE DISSERTATION

Personality Pathology and Cognitive Aging: The Role of Interpersonal Stress

by

Patrick J. Cruitt

Doctor of Philosophy in Psychological and Brain Sciences

Washington University in St. Louis, 2021

Professor Thomas Oltmanns, Chair

Research on the relationship between normal-range personality and cognitive aging has demonstrated consistent, but modest, effects. The current investigation seeks to increase our understanding of unhealthy cognitive aging by examining the maladaptive extremes of personality. Borderline and avoidant personality disorder (PD), but not obsessive-compulsive PD, were hypothesized to show prospective associations with cognitive aging. Interpersonal stress was expected to mediate these relationships. The current investigation tested these hypotheses in two longitudinal studies of older adulthood: the Alzheimer’s Disease Research Center cohort (ADRC, N = 434, M_{age} = 69.95, 56% women) and the St. Louis Personality and Aging Network study (SPAN, N = 1,058, M_{age} = 65.92, 54% women). The ADRC study administered a battery of neuropsychological tests to assess cognitive ability/memory. Borderline PD was measured with a composite derived from the NEO Five Factor Inventory. The SPAN study administered self-, informant, and interview measures of the three PDs, a free recall memory task, and an informant report measure of cognitive problems. Interpersonal stress was operationalized as interpersonal stressful life events and perceived social support. Borderline PD
features exhibited cross-sectional correlations with memory (ADRC: $r = -.11$; SPAN: all $rs = -.08$), general cognitive ability (ADRC: $r = -.11$), and cognitive problems ($rs$ ranged from .15 to .39). These features also prospectively predicted changes in cognitive problems (Std. $bs = .13$ and .15), but not in memory or cognitive ability. Avoidant and obsessive-compulsive PD exhibited little association with cognitive aging. Neither interpersonal stress variable mediated any effect of personality pathology on cognitive aging. These findings suggest that borderline PD features may interfere with cognitive maintenance interventions. Furthermore, they argue for the development of PD treatments adapted for the context of later life.
Chapter 1: Introduction

A growing body of literature suggests that a particularly maladaptive pattern of personality traits for cognitive health includes low conscientiousness, high neuroticism, and possibly low agreeableness (Curtis, Windsor, & Soubelet, 2015; Low, Harrison, & Lackersteen, 2013; Luchetti, Terracciano, Stephan, & Sutin, 2016; Terracciano, Stephan, Luchetti, Albanese, & Sutin, 2017; Terracciano et al., 2014). These effects have been relatively modest, and known behavioral risk factors for cognitive decline do not fully account for the variance attributable to personality traits (Terracciano et al., 2017). Despite the generally maladaptive pattern of personality traits identified as predictors, few studies have examined the links between personality pathology and cognitive decline, and those that have are cross-sectional and limited in scope (Cruitt & Oltmanns, 2018b; Dondu, Sevincoka, Akyol, & Tataroglu, 2015; Henriques-Calado & Duarte-Silva, 2019; Nicholas et al., 2010; Pilleron et al., 2015; Prior et al., 2016). Personality pathology contributes unique variance above and beyond Big Five traits to the prediction of a variety of later life outcomes (Cruitt & Oltmanns, 2018a; Gleason, Weinstein, Balsis, & Oltmanns, 2014), and measures of pathology capture additional information at the extreme ends of underlying, latent personality dimensions (Samuel, Simms, Clark, Livesley, & Widiger, 2010; Suzuki, Samuel, Pahlen, & Krueger, 2015). Additionally, the emphasis in the clinical literature on examining maladaptive processes as potential therapeutic targets offers insight into what biopsychosocial mechanisms may be at play in the relationship between personality and cognitive aging. Therefore, incorporating personality pathology into predictive models of cognitive aging outcomes has the potential to both improve the detection of at-risk individuals and help identify mediating factors.
Much of the research on personality and cognitive aging has examined dementia diagnoses or declines in global cognitive abilities. However, cognitive aging is not a unitary phenomenon, and different cognitive abilities show different trajectories in both healthy and pathological aging (Karr, Graham, Hofer, & Muniz-Terrera, 2018; Salthouse, 2004, 2018). For example, declines in verbal memory appear to begin prior to the onset of declines in other cognitive abilities, such as visuospatial processing or executive function. Therefore, it is important to study the relationship between personality and changes in the particular cognitive abilities, such as memory, that for many individuals serve as early symptoms of cognitive decline. Previous evidence suggests that rates of decline in episodic memory are related to the personality traits of neuroticism and conscientiousness (see Curtis, Windsor, & Soubelet, 2015 for a review). The results for conscientiousness are relatively consistent. For example, one study found that higher conscientiousness is associated with improved recall over time (Hock et al., 2014). Another study demonstrated that higher conscientiousness is also associated with a slower rate of decline in episodic memory specifically during terminal decline (i.e., the acceleration in cognitive decline observed prior to death; Wilson, Boyle, Yu, et al., 2015). The evidence with regard to neuroticism is more mixed. Some studies have found an association between neuroticism and episodic memory declines (Wilson et al., 2003; Wilson, Schneider, Boyle, et al., 2007), whereas others have found no relationship (Arbuckle, Maag, Pushkar, & Chaikelson, 1998; Hultsch, Hertzog, Small, & Dixon, 1999; Jelicic et al., 2003; Wetherell, Reynolds, Gatz, & Pedersen, 2002). Examining personality constructs that extend beyond the normal-range Big Five domains, such as personality pathology, may help resolve these ambiguities in the literature. Therefore, the current investigation seeks to address this gap by exploring both the prospective relationship
between personality pathology and global cognitive ability (or cognitive problems) as well as personality pathology’s prospective relationship with the specific domain of memory.

1.1 Personality Pathology and Cognitive Aging
To examine the role of personality pathology in cognitive aging, it is first important to define personality pathology and consider which of its manifestations are most likely to affect later-life cognitive outcomes. Just like normal-range personality, personality pathology represents relatively stable individual differences in patterns of thinking, feeling, and behaving. The core distinction is that personality pathology is inflexible across situations, inappropriate for the individual’s culture or developmental stage, and leads to distress or impairment. However, the best way to characterize clinically meaningful personality features that fit these criteria is the subject of much debate. Section II of the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5), includes ten categorical diagnoses of specific personality disorders (PDs), each defined by a set of symptoms (American Psychiatric Association, 2013). These symptoms have fostered an extensive research literature, including a burgeoning focus on their expression in later life (Cruitt & Oltmanns, 2018a; Van Alphen et al., 2015). However, categorical diagnoses as a whole are subject to a host of limitations, including heterogenous presentations, excessive comorbidity, and instability compared to other mental disorders (Clark, 2007; McDavid & Pilkonis, 1996; Zimmerman, Rothschild, & Chelminski, 2005).

Although the categorical diagnoses themselves are problematic, the symptoms that compose these diagnoses tend to be strong indicators of personality dysfunction when assessed dimensionally. To address the limitations of the Section II model and provide a way to conceptualize previous research on these symptoms, hybrid dimensional/categorical diagnostic models have been proposed in Section III of the DSM-5 that translate six of the PDs into
combinations of specific impairments in personality functioning and maladaptive trait profiles (Hopwood, Thomas, Markon, Wright, & Krueger, 2012; Morey, Benson, & Skodol, 2016). For the most part, these maladaptive traits represent pathological extensions of the Big Five, allowing researchers interested in aging to generalize from the normal-range personality and cognition literature to generate hypotheses regarding specific PDs. In a similar vein, examining measures of Section II PD symptoms may yield insights about the role of particularly pathological combinations of personality traits in cognitive aging that would be obscured in studies of normal-range personality (Cruitt, 2020). The current investigation capitalizes on these interconnections by using traditional symptom-based measures of specific PDs to replicate and extend previous findings from the normal-range personality literature, with an aim toward drawing conclusions that can then be translated across multiple conceptualizations of personality pathology.

Out of the six PDs with hybrid models in Section III of the DSM-5, three in particular seem like good candidates for exploring the role of personality pathology in cognitive aging: Borderline (BPD), avoidant (AVPD) and obsessive-compulsive personality disorder (OCPD).\(^1\) All three of these disorders are considered to a certain extent to be defined by negative affectivity, the pathologically high end of neuroticism, suggesting that they may exhibit similar prospective associations as neuroticism with cognitive ability and memory. Of these, BPD is the most closely related to negative affectivity and is also characterized by disinhibition (the pathologically low

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\(^1\) The other three specific PDs with hybrid models are schizotypal, narcissistic, and antisocial PD. Early waves of the St. Louis Personality and Aging Network (SPAN) study (see below) found extremely low rates of schizotypal PD (Oltmanns et al., 2014), indicating that it would be difficult to detect an effect of these symptoms on cognitive outcomes. Narcissistic and antisocial PD are primarily disorders of antagonism (i.e., low agreeableness). Overall, agreeableness has not consistently shown associations with cognitive ability, although some evidence exists of a modest association with Alzheimer’s disease (Curtis et al., 2015; Terracciano et al., 2014). Antisocial PD is also associated with disinhibition (i.e., low conscientiousness), but as with schizotypal PD these particular features are extremely rare in later life making it difficult to detect an effect on cognitive aging outcomes in a general population.
end of conscientiousness). This is consistent with the associations found between BPD symptoms and Big Five traits (Samuel & Widiger, 2008; Saulsman & Page, 2004), and BPD symptoms demonstrate similar negative health effects to these traits across the lifespan (see Dixon-Gordon, Whalen, Layden, & Chapman, 2015 for a review). One might further expect these negative consequences to extend to cognitive decline. A previous study found higher rates of current and retrospectively-reported premorbid BPD in patients with Alzheimer’s disease (Henriques-Calado & Duarte-Silva, 2019). Furthermore, this study found that current Cluster B PD features, which include BPD features, contributed unique variance to the prediction of Alzheimer’s disease diagnosis when controlling for age, cognitive ability, and other PD features. This finding aligns with those of another study showing that BPD features assessed from multiple sources consistently contribute unique variance to the prediction of observer-rated cognitive problems, after controlling for other PDs (Cruitt & Oltmanns, 2018b). Although controlling for multiple PDs in a single model make interpretation with regard to specific disorders complicated due to extensive comorbidity, it appears that BPD is a natural starting place to begin examining the longitudinal associations between particular manifestations of personality pathology and cognitive aging outcomes.

As with BPD, the conceptualization of AVPD/OCPD as disorders related to negative affectivity argues for a relationship between these disorders and unhealthy cognitive aging. Furthermore, there is some evidence that these symptoms are relatively more prevalent than BPD among older adults (Oltmanns, Rodrigues, Weinstein, & Gleason, 2014). With regard to AVPD, this is despite the fact that age bias in its criteria may result in underdiagnosis in this age group (Balsis, Gleason, Woods, & Oltmanns, 2007; Balsis, Woods, Gleason, & Oltmanns, 2007; Oltmanns & Balsis, 2011). After BPD, AVPD is the PD most closely linked with neuroticism (Samuel &
Widiger, 2008; Saulsman & Page, 2004). However, a second major component of AVPD is detachment, or pathologically low extraversion (American Psychiatric Association, 2013). Normal-range extraversion at the domain level is generally unassociated with cognitive ability or memory declines in older adulthood, or with risk for dementia (Curtis et al., 2015; Terracciano & Sutin, 2019). As such, one might expect that AVPD symptoms would show a weaker effect on cognitive outcomes than BPD symptoms, which are associated with a pattern of maladaptive traits more closely linked with negative cognitive aging outcomes.

Out of the three specific disorders in question, OCPD has the most mixed evidence with regard to cognitive aging. Unlike AVPD and BPD, which appear to be endorsed less commonly in older adulthood relative to younger adulthood, some evidence suggest that OCPD features are more commonly endorsed as people age (Abrams & Horowitz, 1999; Balsis, Gleason, et al., 2007). Although one study found no evidence of increased retrospectively-reported OCPD symptoms in AD patients (Nicholas et al., 2010), others have found that retrospective OCPD diagnoses were more prevalent in this population (Dondu et al., 2015; Henriques-Calado & Duarte-Silva, 2019). Some limitations of these latter studies make it difficult to draw firm conclusions about whether OCPD symptoms represent risk factors for other cognitive aging outcomes. First, premorbid symptoms were reported retrospectively, preventing strong causal inferences. Second, in one case appears that informants were used to supplement patient report to guard against unreliable responding (Dondu et al., 2015). Some evidence suggests that informants endorse higher rates of PD symptoms than do targets (Oltmanns et al., 2014), although other studies report contradictory findings (Sleep, Lamkin, Lynam, Campbell, & Miller, 2019). If OCPD diagnosis was more dependent on informant report than self-report in the AD patients compared to controls, it may have led to a biased estimate of the prevalence of previous OCPD in the AD patients. Informants
may attribute certain OCPD features, such as rigidity and stubbornness, to problems due to changes in cognitive ability or memory. Therefore, although there is some evidence suggesting a link between OCPD and cognitive decline, alternative explanations have yet to be fully ruled out.

Although there are no additional studies that I am aware of examining the explicit link between OCPD and cognitive aging, additional evidence suggests that OCPD is not a strong risk factor for negative cognitive outcomes in later life. A primary feature of OCPD is rigid perfectionism, which is theorized to represent the pathologically high end of Big Five conscientiousness (American Psychiatric Association, 2013), and meta-analytic studies confirm that OCPD symptoms exhibit a moderate, positive correlation with conscientiousness (Samuel & Widiger, 2008; Saulsman & Page, 2004). As mentioned above, higher conscientiousness has been found to prospectively predict slower rates of decline in general cognitive ability and episodic memory (Chapman et al., 2012; Wilson, Boyle, Yu, et al., 2015; Wilson, Schneider, Arnold, Bienias, & Bennett, 2007). Therefore, the question remains whether maladaptively high conscientiousness as manifested in OCPD demonstrates a paradoxical negative association with cognitive aging outcomes or if it counteracts the potential negative effect of other maladaptive OCPD features.

The current study seeks to address this question by studying the prospective association between explicit, multisource measures of OCPD symptoms and cognitive aging outcomes over time. In examining these maladaptive extremes of personality, the goal is to identify potential mechanisms that may partially account for the association between personality and cognitive decline and represent future targets for research and intervention.

1.2 The Role of Interpersonal Stress
One such mechanism that might help link personality pathology with cognitive aging is interpersonal functioning. Positive social functioning in the form of social engagement is one of
the consistent factors linked to healthy cognitive aging (Smith, 2016). At the same time, poor interpersonal functioning in the form of interpersonal stress is associated with cognitive decline. Longitudinal evidence indicates that stressful life events (SLEs), including interpersonal SLEs, predict risk for dementia later in life (Johansson et al., 2013), and the frequency of negative social interactions is associated with risk for developing mild cognitive impairment (Wilson, Boyle, James, et al., 2015). In addition, negative social interactions averaged across time were associated with more rapid decline in episodic, semantic, and working memory. Given these findings, researchers have suggested that personality may provide important context for understanding the role of social functioning on cognitive abilities in later life (Hill & Payne, 2017). It may be that personality pathology is an important precursor to interpersonal stress, through which it then negatively affects cognitive aging. Yet, there is currently little to no research directly examining interpersonal stress as a potential mediator of the relationship between personality and cognitive decline.

Evidence in support of this idea comes from work demonstrating strong associations between PD symptoms and interpersonal stress. This is particularly true with regard to BPD symptoms. The social networks of individuals with higher levels of BPD features are characterized by greater frequency of negative interactions and poorer social support (Beeney, Hallquist, Clifton, Lazarus, & Pilkonis, 2018). Furthermore, BPD features prospectively predict increased rates of interpersonally-oriented, dependent SLEs (Conway, Boudreaux, & Oltmanns, 2018; Gleason, Powers, & Oltmanns, 2012; Powers, Gleason, & Oltmanns, 2013). These findings suggest that BPD may impact cognitive aging through two pathways: increased interpersonal SLEs and lower

---

2 For the sake of simplicity, positive and negative interpersonal functioning are presented here as two sides of the same coin. However, similar to positive and negative affect, positive and negative interpersonal functioning may exist on somewhat independent dimensions (Schuster, Kessler, & Aseltine, 1990). The implications of this for individualized cognitive maintenance interventions will be revisited in the discussion.
perceived social support. Similarly, AVPD also negatively impacts interpersonal functioning, though perhaps not in the same volatile manner. Whereas AVPD does not predict increased rates of interpersonal SLEs, it is associated with interpersonal dysfunction and lower social support (Wilberg, Karterud, Pedersen, & Urnes, 2009). Therefore, AVPD may also predict cognitive aging through decreased perceived social support, although not through interpersonal SLEs. Finally, OCPD is also characterized by interpersonal dysfunction (Cain, Ansell, Simpson, & Pinto, 2015). However, the mixed evidence reviewed above makes it unclear whether OCPD would influence cognitive aging outcomes through either of the proposed pathways.

1.3 The Current Investigation
Given the clear intersection of personality pathology, interpersonal stress, and cognitive decline, the current dearth of research that examines all three of these constructs is unfortunate. This gap in the literature may be due, in part, to the challenges associated with assessing personality pathology and cognitive decline in later life. Assessments for both domains are time and effort intensive, and both benefit immensely from consideration of multiple sources of information, including informants. The current study addresses this gap in the literature by integrating findings across two longitudinal datasets of community-dwelling older adults. The Knight Alzheimer’s Disease Research Center (ADRC) in St. Louis has cultivated a pool of participants with detailed cognitive assessments at multiple time points, and has already contributed meaningfully to our understanding of the relationship between normal-range personality and cognitive decline (Duchek, Balota, Storandt, & Larsen, 2007; Schultz et al., 2017). Meanwhile, the St. Louis Personality and Aging Network (SPAN) is the first major, longitudinal study to examine personality pathology as assessed from multiple different perspectives (self, informant, and interview) in community-dwelling older adults (Oltmanns et al., 2014). These
complementary datasets represent two sides of the same coin. The ADRC dataset has comprehensive assessments of cognitive aging coupled with a brief self- and informant report personality inventory (from which a BPD composite can be drawn), whereas SPAN has extensive interview, self- and informant report personality measures (encompassing all three relevant diagnoses) and two brief screening instruments for cognitive decline. By comparing findings across these two longitudinal datasets, the current study is able to draw more robust conclusions about the relationship between personality pathology and cognitive aging than would be possible by examining either set of data in isolation.

1.4 Specific Aim 1
The first aim of the current study was to examine the cross-sectional and prospective relationships between personality pathology and cognitive aging outcomes in both the ADRC and SPAN samples. Below, I describe my hypotheses, broken down by sample.

1.4.1 ADRC Hypotheses
Hypothesis 1.1 Based on the previous literature, I predicted that BPD traits would show cross-sectional associations with general cognitive ability and memory. In particular, I anticipated that latent BPD traits (obtained from a principal components analysis of self and informant report) would show negative, cross-sectional associations with free recall and a general cognitive composite that incorporates measures of processing speed, executive functioning, memory and verbal fluency. I also expected mean BPD traits to be higher in a group of individuals with a clinical dementia rating (CDR) of 0.5 or greater compared to those in a CDR 0 group.

Hypothesis 1.2 With regard to the prospective relationship between BPD traits and cognitive aging outcomes, I hypothesized that BPD traits would prospectively predict residualized change
in performance on a free recall task as well as the general cognitive composite, controlling for baseline age, gender, race, and education.

1.4.2 SPAN Hypotheses

**Hypothesis 1.3** As in the ADRC sample, I predicted that latent BPD traits would show a negative, cross-sectional association with immediate free recall and a positive correlation with informant reported cognitive problems. I also expected that the BPD traits would prospectively predict residualized change in these cognitive aging outcomes (controlling for covariates), again paralleling the analyses in the ADRC sample.

**Hypothesis 1.4.** Relative to the ADRC study, SPAN obtained more comprehensive and explicit measures of PD symptomatology. As such, I was able to examine interviewer, self- and informant report of BPD symptoms, as well as AVPD and OCPD. I predicted that the BPD symptom-based scores would show similar cross-sectional associations as those obtained from the trait-based assessment. In addition, I hypothesized that AVPD would show a negative correlation with immediate free recall and a positive association with informant reported cognitive problems. Due to the discrepancies in the literature described above, it was difficult to make specific predictions with regard to OCPD features. However, I predicted that it would be unrelated to the cognitive aging outcomes.

**Hypothesis 1.5.** Finally, I expected that BPD and AVPD symptom-based measures would prospectively predict residualized change in immediate free recall and informant reported cognitive problems, controlling for baseline age, gender, race, education, and household income. I did not expect OCPD to prospectively predict residualized change in either outcome.
1.5 Specific Aim 2
My second specific aim was to examine the potential role of interpersonal SLEs and (lack of) perceived social support in mediating the relationship between personality pathology and cognitive aging. As only the SPAN sample had data regarding interpersonal SLEs and perceived social support, the following hypotheses were tested in that sample.

*Hypothesis 2.1.* I predicted that the number of interpersonal SLEs that were reported at follow-up to have occurred between the initial and follow-up assessments would partially mediate the previously established prospective relationships between BPD (but not AVPD) and residualized change in informant reported cognitive problems and immediate free recall, controlling for baseline age, gender, race, education, and household income. I did not make predictions regarding OCPD, as I did not anticipate OCPD to be related to changes in the cognitive aging outcomes.³

*Hypothesis 2.2.* In addition, I expected perceived social support to partially mediate the previously established prospective relationships between BPD and AVPD and residualized change in informant reported cognitive problems and immediate recall, controlling for baseline age, gender, race, education, and household income. Again, I did not make predictions regarding OCPD.⁴

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³ I determined ahead of time that if OCPD symptoms did predict cognitive aging outcomes, mediation analyses would be conducted in an exploratory manner.
⁴ These hypotheses and the methods used to test them were preregistered at [https://osf.io/auc5g](https://osf.io/auc5g)
Chapter 2: Method

2.1 Participants
The current study used data from two independent samples of adults from the St. Louis metropolitan area. The ADRC sample initially consisted of 1,211 adults (56% women; aged 30 to 101, \( M = 69.95 \)). Of these, 434 participants had personality data and neuropsychological data at one time point (ADRC Time 1), and 424 had neuropsychological data for at least one time point afterward (ADRC Time 2). On average, these waves were collected 1.8 years apart. As for the SPAN sample, data for the current analyses come primarily from the third and fourth in-lab assessments (hereafter referred to as SPAN Time 1 and SPAN Time 3), which occurred on average 2.27 years apart. I also used an assessment of perceived social support obtained during an at-home wave that occurred mid-way between these two in-lab assessments (which will be called SPAN Time 2). Out of 1,630 participants at the initial assessment, 1,058 participated at SPAN Time 1 (54% women). At SPAN Time 1, participants ranged in age from 60 to 73 (\( M = 65.92 \)). Of the participants with SPAN Time 1 data, 974 had relevant data from SPAN Time 3 for the analyses using free recall as the outcome, and 424 had data for the analyses using informant reported cognitive problems as assessed by the Ascertain Dementia Eight-Item Informant Questionnaire (AD8).\(^5\)

2.2 ADRC Measures

2.2.1 NEO Five-Factor Inventory
A short form of the 240-item NEO Personality Inventory-Revised (NEO PI-R), the NEO Five-Factor Inventory (NEO FFI) consists of 60 items assessing the five domains of the five-factor

\(^5\) This latter number was lower than anticipated based on informant completion rates in previous waves of data collection, a limitation that is noted in the discussion.
model (FFM; Costa & McCrae, 1992). Both self- and informant report versions were administered in the ADRC sample. Given the use of the NEO FFI to measure personality in large epidemiological studies, Few et al. (2016) developed a 24-item BPD composite score (FFI-BPD), for use as a proxy of personality pathology when conducting secondary analyses on these richly characterized datasets. The development study found that the median correlation between the FFI-BPD and explicit measures of BPD symptomatology across six samples was .60, similar to the previously validated NEO-BPD composite score derived from the full NEO PI-R (Miller, Bagby, Pilkonis, Reynolds, & Lynam, 2005). The FFI-BPD exhibited similar correlations as BPD features assessed through other methods with psychiatric comorbidities and etiological factors (e.g., childhood abuse), as well as a similar heritability (Few et al., 2016). Therefore, it appears that the FFI-BPD adequately captures this maladaptive personality pattern and is a valid tool for examining the association between BPD and cognitive decline. Descriptive statistics for the FFI-BPD composite scores are presented in Table 1. Items on the NEO-FFI are responded to on a scale from 0 (Strongly Disagree) to 4 (Strongly Agree), as such scores on the FFI-BPD composite have an absolute range of 0 to 4. Self- and informant reported FFI-BPD scores were correlated, \( r(393) = .40 \), 95% CI [.31, .48].

2.2.2 Clinical Dementia Rating

A global measure of dementia severity, the Clinical Dementia Rating (CDR) scale was developed at Washington University in St. Louis to adequately capture the full range of dementia severity using behavioral and psychometric data (Berg, 1988; Hughes, Berg, Danziger, Coben, & Martin, 1982; Morris, 1993). A CDR score is assigned to participants on the basis of an approximately 90-minute interview procedure that obtains independent information from both the participant and a close other. CDR scores of 0, 0.5, 1, 2, and 3 correspond to diagnoses of no
Table 1

Descriptive Statistics for Personality Measures in Both ADRC and SPAN Samples

<table>
<thead>
<tr>
<th>Personality Measures</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
<th>Cronbach’s Alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADRC FFI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-report Borderline</td>
<td>1.33</td>
<td>0.40</td>
<td>0.21-2.54</td>
<td>0.80</td>
</tr>
<tr>
<td>Informant report Borderline</td>
<td>1.22</td>
<td>0.47</td>
<td>0.13-3.08</td>
<td>0.86</td>
</tr>
<tr>
<td>SPAN FFI</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Self-report Borderline</td>
<td>1.34</td>
<td>0.38</td>
<td>0.33-3.08</td>
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</tr>
<tr>
<td>Informant report Borderline</td>
<td>1.41</td>
<td>0.50</td>
<td>0.29-3.58</td>
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</tr>
<tr>
<td>SPAN MAPP</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-report Borderline</td>
<td>0.46</td>
<td>0.39</td>
<td>0.00-3.67</td>
<td>0.68</td>
</tr>
<tr>
<td>Informant report Borderline</td>
<td>0.57</td>
<td>0.55</td>
<td>0.00-2.89</td>
<td>0.79</td>
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<tr>
<td>Self-report Avoidant</td>
<td>0.65</td>
<td>0.57</td>
<td>0.00-3.71</td>
<td>0.82</td>
</tr>
<tr>
<td>Informant report Avoidant</td>
<td>0.66</td>
<td>0.67</td>
<td>0.00-4.00</td>
<td>0.85</td>
</tr>
<tr>
<td>Self-report Obsessive-Compulsive</td>
<td>1.19</td>
<td>0.54</td>
<td>0.00-3.50</td>
<td>0.65</td>
</tr>
<tr>
<td>Informant report Obsessive-Compulsive</td>
<td>1.43</td>
<td>0.64</td>
<td>0.00-3.50</td>
<td>0.66</td>
</tr>
<tr>
<td>SPAN SIDP</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Borderline</td>
<td>0.07</td>
<td>0.17</td>
<td>0.00-1.71</td>
<td>0.71</td>
</tr>
<tr>
<td>Avoidant</td>
<td>0.10</td>
<td>0.26</td>
<td>0.00-2.14</td>
<td>0.81</td>
</tr>
<tr>
<td>Obsessive-compulsive</td>
<td>0.22</td>
<td>0.25</td>
<td>0.00-1.63</td>
<td>0.58</td>
</tr>
</tbody>
</table>

Note. ADRC = Alzheimer’s Disease Research Center. SPAN = St. Louis Personality and Aging Network. FFI = NEO Five-Factor Inventory. MAPP = Multisource Assessment of Personality Pathology. SIDP = Structured Interview for DSM-IV Personality.
dementia, very mild dementia, mild dementia, moderate dementia, and severe dementia, respectively. Out of the 434 participants at ADRC Time 1, 58 (13.36%) had a CDR score of 0.5, with the remaining participants having a score of 0. At ADRC Time 2, 12.26% (n = 52) had a CDR score of 0.5 and 1.18% (n = 5) had a score of 1, with the rest again receiving a score of 0.

2.2.3 Free Recall
I used the free recall portion of the selective reminding task to measure episodic memory performance (Grober, Buschke, Crystal, Bang, & Dresner, 1988). Participants were presented with pictures of 16 items alongside a cue word indicating the category to which the item belonged. Participants engaged in three recall trials, starting each with a 20 second interference task (counting backwards from 97 by 3s). Within each trial, free recall was measured first by allowing participants 90 seconds to recall as many items as they could. Then, assessors administered the cued recall portion by presenting the category cue for each item, telling participants the non-recalled items after 10 seconds (this part of the task was only included in the general cognitive composite described below for the purpose of the current analyses). Free recall scores represent the number of items recalled during the free recall portion of all three trials, with an absolute range of 0 to 48. Descriptive statistics can be found in Table 2.

2.2.4 General Cognitive Composite
I generated a general cognitive composite from performance on the free and selective reminding, animal naming, and trails A and B tasks by averaging z-scores for each test. These measures were selected because they provided good coverage of memory, verbal fluency, processing speed and executive functioning abilities. Free and selective reminding (see above) consists of both the free recall score and the total number of items recalled across both the free and cued recall portions of the task. Animal naming is a self-explanatory verbal fluency task in which
Table 2
Descriptive Statistics for Cognitive Aging Measures in Both ADRC and SPAN Samples

<table>
<thead>
<tr>
<th>Cognitive Aging Measures</th>
<th>ADRC/SPAN Time 1</th>
<th>ADRC Time 2/SPAN Time 3</th>
<th>Test-Retest Correlation [95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Range</td>
</tr>
<tr>
<td>ADRC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free Recall</td>
<td>29.93</td>
<td>6.75</td>
<td>1-46</td>
</tr>
<tr>
<td>Selective Reminding</td>
<td>47.63</td>
<td>1.75</td>
<td>21-48</td>
</tr>
<tr>
<td>Animal Naming</td>
<td>21.07</td>
<td>5.68</td>
<td>2-37</td>
</tr>
<tr>
<td>Trails Making A</td>
<td>32.45</td>
<td>11.88</td>
<td>10-104</td>
</tr>
<tr>
<td>Trails Making B</td>
<td>80.57</td>
<td>35.14</td>
<td>19-240</td>
</tr>
<tr>
<td>SPAN</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free Recall</td>
<td>5.9</td>
<td>2.03</td>
<td>0-13</td>
</tr>
<tr>
<td>Cognitive Problems</td>
<td>0.54</td>
<td>1.23</td>
<td>0-8</td>
</tr>
</tbody>
</table>

*Note. ADRC = Alzheimer’s Disease Research Center. SPAN = St. Louis Personality and Aging Network.*
participants name animals for one minute, with scores representing how many different animals they were able to generate (Goodglass & Kaplan, 1983). Trailmaking A and B assess processing speed and executive functioning abilities (Armitage, 1946). Part A consists of drawing a line between 25 numbered circles in order, whereas Part B has participants alternate between numerically and alphabetically ordered circles. Scores represents time to complete the task, in seconds (time is capped at a maximum of 150 and 300 seconds in A and B, respectively). Trails A and B were negatively weighted in the composite, as higher scores on these tasks indicate greater impairment. Descriptive statistics for all of the neuropsychological tasks are presented in Table 2.

2.3 SPAN Measures

2.3.1 NEO Personality Inventory-Revised
The full 240-item NEO PI-R used in the current analyses was administered at the first wave of data collection, using both self- (Form S) and informant (Form R) report versions (Costa & McCrae, 1992). The FFI-BPD composite was calculated for comparison purposes with the ADRC sample. Descriptive statistics for the FFI-BPD composite scores are presented in Table 1. Self-report and informant report were correlated \( r(838) = .41, 95\% \text{ CI } [.35, .46] \).

2.3.2 Structured Interview for DSM-IV Personality
The Structured Interview for DSM-IV Personality (SIDP-IV) is an 80-item semistructured diagnostic interview for the PD symptoms contained in the fourth and fifth editions of the *DSM* (American Psychiatric Association, 2013; Pfohl, Blum, & Zimmerman, 1997). Trained graduate students, full time research staff and a few advanced undergraduate students administered the SIDP-IV at SPAN Time 1. Interviewers rated each item on a scale from 0 (*not present*) to 3 (*strongly present*) and were trained to frame probe questions in a way that was relevant to the
context of later life in order to make ratings. Mean scores for each of the specific PD diagnoses (BPD, AVPD, and OCPD) were calculated (see Table 1 for descriptive statistics and internal consistency). Interrater reliability was calculated at the baseline assessment of the SPAN study by having independent raters watch a randomly selected subsample of 265 interviews. One-way random, average measures intraclass correlation coefficient for the BPD, AVPD, and OCPD scores at baseline were .77, .86, and .62, respectively.

2.3.3 Multisource Assessment of Personality Pathology
The Multisource Assessment of Personality Pathology (MAPP) is also an 80-item measure of the PD features described in the DSM, translated into lay terms and administered in both self- and informant report versions. The MAPP data used in the current analyses were obtained at SPAN Time 1. Care was taken in developing the MAPP that each item was relevant to the context of later life. Participants and informants rated each item from 0 (I am/He or she is never like this) to 4 (I am/He or she is always like this). Composite scores for each of the relevant diagnoses for the current analyses were calculated. Descriptive statistics are presented in Table 1.

2.3.4 The Ascertain Dementia Eight-Item Informant Questionnaire
The AD8 is a screening instrument for cognitive decline that assesses informant reported cognitive problems due to changes over the last several years (Galvin et al., 2005). Each of the eight items has three response options: “YES, a change,” “NO, no change,” or “N/A, Don’t know.” A total score of cognitive change is obtained by summing “YES” responses. In a community sample, a cutoff of two or more demonstrated 85% sensitivity and 86% specificity in discriminating between individuals with a Clinical Dementia Rating (CDR) of 0 and a CDR of 0.5 or greater (Galvin et al., 2005). Out of the 842 participants with complete AD8 data at SPAN
Time 1, 120 (14.25%) received a score of two or more. Table 2 contains descriptive statistics for the AD8.

### 2.3.5 Immediate Free Recall
Immediate free recall was tested in the SPAN sample using a list of sixteen, verbally presented words at each wave. Word lists were selected from a set of six, which were previously developed for use in a study on dementia. Participants received different lists at consecutive waves. Lists were balanced with each other in terms of word length, frequency, and concreteness. Administrators provided the words at a three-second rate of presentation and gave the participants two minutes to recall all of the words from the list that they could remember. Scores represent the number of words recalled, with an absolute range from 0 to 16. See Table 2 for descriptive statistics.

### 2.3.6 List of Threatening Experiences Questionnaire
The List of Threatening Experiences Questionnaire (LTE) assesses the presence or absence of twelve different life events that are typically associated with negative long-term outcomes (Brugha, Bebbington, Tennant, & Hurry, 1985). The LTE data used in the current analyses were collected at SPAN Time 3. Trained interviewers asked whether the participant experienced each item in the last six months, and participants responded either yes or no. Separately, the interviewers asked about the time period since the date of the participant’s last in-lab follow-up (for the purposes of the current analyses, SPAN Time 1), excluding the last six months (Mean duration = 19.80 months, $SD = 8.26$, Range = 3-106)⁶ Interviewers would follow-up with probe questions to ensure that the event actually occurred to the participant personally and in the

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⁶ Due to an error in training, a small subset of participants was asked about the period since the intervening at-home follow-up (SPAN Time 2). As such, some events may have been missed, but there is no reason to suspect systematic error.
appropriate time frame, and was a major, discrete event (Paykel, 1983). This process protected against over-reporting biases associated with personality pathology, as individuals with more BPD features have shown higher rates of adjustment after the follow-up interview (Gleason et al., 2012; Harkness & Monroe, 2016). Interpersonal SLEs consisted of separation due to marital difficulties, breaking off a steady relationship, and serious problem or conflict with a close other (Powers et al., 2013). Of the 424 participants who had complete data for the mediation analyses using the LTE-Q, 16.27% (n = 69) reported at least one interpersonal SLE, with nine participants reporting two events. Participants reported a total of 61 conflicts with a close other, 16 break ups, and 3 separations due to marital difficulties. Due to the highly skewed nature of the data, interpersonal SLEs were coded either as present (1) or absent (0) rather than adding up the number of events.

2.3.7 Quality of Relationship Inventory
At SPAN Time 2, participants completed the seven-item general support subscale of the Quality of Relationship Inventory (QRI; Pierce, Sarason, & Sarason, 1991). Items on this subscale assess perceptions of support from relationships other than a person’s spouse or partner, for example, “To what extent could you turn to at least one of these people for advice about problems?” Participants are instructed to respond to items on a 4-point Likert-style scale, from 1 (Not at all) to 4 (Very much). Composite scores for the QRI general support subscale ranged from 1 to 4 (M = 3.14, SD = 0.69). Cronbach’s alpha for the subscale was .92.
2.4 Data Analytic Plan
All analyses were conducted in R (R Core Team, 2018). Excluding the overall correlational analyses, I predetermined that I would run 19 confirmatory statistical tests.\(^7\) As such, I adopted a Bonferroni corrected alpha threshold of \(p < .003\).

2.4.1 Specific Aim 1 Analytic Plan
**ADRC Hypotheses.** First, I imputed missing data for the self- and informant reported FFI-BPD scores based on participant’s scores on the other measure using the missMDA package (Josse & Husson, 2016).\(^8\) I then submitted these variables to a principal components analysis using the FactoMineR package (Le, Josse, & Husson, 2008). I decided a priori that a single latent factor score was to be extracted if it accounted for at least 60% of the variance in the measures and no more than two dimensions received eigenvalues of greater than 1. If a single factor failed to emerge, tests were to be run with both the self- and informant reported FFI-BPD separately, and the focus of the interpretation would be on those findings that are consistent across both measures.

To test Hypothesis 1, I used correlational analyses to obtain an estimate of the cross-sectional relationship between BPD and performance on free recall and the general cognitive composite in the ADRC sample. I ran a t-test to determine if BPD traits are higher in the CDR 0.5 group relative to the CDR 0 group at ADRC Time 1. For Hypothesis 2, I ran two linear regression models predicting free recall and general cognitive composite at ADRC Time 2 from the FFI-

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\(^7\) This number assumed that analyses were to be conducted with a single latent factor score in all cases, rather than treating each individual measure of the same construct as a separate test (see below). In cases where the single latent factor failed to meet my a priori fit criteria, I focus my interpretation of the results on findings that are relatively consistent across measures of the same construct, treating them as a family of tests.

\(^8\) Imputing missing data for the purpose of the principal components analyses was not mentioned in the preregistration, but this procedure followed the guidelines for using the PCA function of the FactoMineR package.
BPD. In addition to initial levels of the outcomes from ADRC Time 1, I entered gender, initial age, and education as covariates in each of the models.

**SPAN Hypotheses.** As for the SPAN hypotheses, I first conducted the correlational analyses between the latent FFI-BPD factor (again obtained through principal components analysis using the procedure described above) and the two cognitive aging measures (AD8 and immediate free recall). I also ran linear regression analyses predicting AD8 and immediate free recall measures at SPAN Time 3 from the FFI-BPD factor and the same covariates used in the ADRC analyses. This step allowed me to test the replicability of findings across samples and enhance the generalizability of further analyses using SPAN data. Next, I submitted the intercorrelations among the SIDP-IV, self-report MAPP, and informant report MAPP scales of each of the three PDs of interest (BPD, AVPD, and OCPD) separately to principal components analyses to obtain latent factor scores for each. I correlated these latent factor scores with AD8 and immediate free recall, then ran linear regression analyses predicting residualized change in these measures from each of the PD factor scores separately (controlling for covariates). As with the FFI-BPD measures, if the factor failed to meet my a priori criteria I planned to run the same results with the individual PD measures instead.

### 2.4.2 Specific Aim 2 Analytic Plan

To test the hypotheses associated with my second specific aim, I performed path analyses using the lavaan package in R (Rosseel, 2012). I entered BPD and AVPD measures separately into two models for each outcome, one with interpersonal SLEs as the mediator and one with perceived social support as a mediator (again controlling for demographic covariates). As mentioned above, no analyses with OCPD measures were planned in advance, as I did not anticipate OCPD to show associations with the cognitive aging outcomes. However, I decided that I would
examine these relationships in an exploratory manner if they were found. In each of the path analyses I used a bootstrapping procedure to obtain an estimate for the indirect pathway, with the number of iterations equal to 5000.
Chapter 3: Results

3.1 Specific Aim 1 Results

3.1.1 ADRC Results

First, self- and informant report FFI-BPD scores were submitted to a principal components analysis. The two dimensions yielded two dimensions with eigenvalues of 1.38 and 0.62, respectively. The first dimension accounted for 69.16% of the variance. As such, I proceeded to use the resulting latent factor scores for all analyses. The FFI-BPD factor score had a range of -2.66 to 4.18 ($M = 0.00, SD = 1.18$).

Cross-sectional correlations between the FFI-BPD factor, CDR score and neuropsychological measures at the first assessment are presented in Table 3. The FFI-BPD factor exhibited the expected negative associations with free recall and the general cognitive composite, although the effects were weak, both $r(427) = -.11$, 95% CI [-.20, -.01]. Consistent with my hypothesis, the FFI-BPD discriminated between those with a CDR score of 0 ($M = -0.10$) and those with a 0.5 ($M = 0.62$, $t [72.84] = -4.14, p < .001$).

Next, I ran a linear regression model predicting residualized change in free recall and general cognition from FFI-BPD factor scores, initial age, years of education, and gender. All variables except for gender were standardized for ease of interpretation. Contrary to hypotheses, FFI-BPD factor scores did not contribute unique variance to the prediction of either outcome at ADRC Time 2 (Table 4). The only significant predictors in both models based on the Bonferroni corrected alpha threshold of $p < .003$ were initial levels of the outcome.
### Table 3

**Correlations Between FFI-BPD Factor, CDR, and Neuropsychological Measures at ADRC Time 1**

<table>
<thead>
<tr>
<th>Variables</th>
<th>FFI-BPD Factor</th>
<th>CDR</th>
<th>General Cognition</th>
<th>Free Recall</th>
<th>Selective Reminding</th>
<th>Animal Naming</th>
<th>Trail Making A</th>
<th>Trail Making B</th>
</tr>
</thead>
<tbody>
<tr>
<td>FFI-BPD Factor</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CDR</td>
<td>.21***</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>General Cognition</td>
<td>-.11*</td>
<td>-.46***</td>
<td>-.74***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free Recall</td>
<td>-.11*</td>
<td>-.42***</td>
<td>-.46***</td>
<td>.43***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Selective Reminding</td>
<td>-.12*</td>
<td>-.36***</td>
<td>-.59***</td>
<td>.43***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Animal Naming</td>
<td>-.02</td>
<td>-.26***</td>
<td>-.65***</td>
<td>.41***</td>
<td>.22***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trails Making A</td>
<td>.04</td>
<td>.29***</td>
<td>-.66***</td>
<td>-.30***</td>
<td>-.10*</td>
<td>-.28***</td>
<td>.52***</td>
<td></td>
</tr>
<tr>
<td>Trails Making B</td>
<td>.06</td>
<td>.22***</td>
<td>-.70***</td>
<td>-.31***</td>
<td>-.21***</td>
<td>-.27***</td>
<td>.52***</td>
<td>.52***</td>
</tr>
</tbody>
</table>

**Note.** Ns range from 429 to 434. FFI-BPD = NEO Five-Factor Inventory Borderline Personality Disorder composite score. CDR = Clinical Dementia Rating. ADRC = Alzheimer’s Disease Research Center sample.

* p < .05. *** p < .001.
Table 4

*Summary of Regression Analyses Predicting ADRC Time 2 Free Recall and General Cognition from FFI-BPD*

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Free recall</th>
<th>General cognition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Std. b</td>
<td>SE</td>
</tr>
<tr>
<td>Intercept</td>
<td>-0.02</td>
<td>0.05</td>
</tr>
<tr>
<td>FFI-BPD Factor</td>
<td>-0.05</td>
<td>0.03</td>
</tr>
<tr>
<td>Time 1 Age</td>
<td>-0.07</td>
<td>0.04</td>
</tr>
<tr>
<td>Years of Education</td>
<td>-0.07</td>
<td>0.03</td>
</tr>
<tr>
<td>Gender</td>
<td>0.06</td>
<td>0.07</td>
</tr>
<tr>
<td>Time 1 Level of Outcome</td>
<td>0.73</td>
<td>0.04</td>
</tr>
</tbody>
</table>

*Note.* All variables were standardized with the exception of gender. ADRC = Alzheimer’s Disease Research Center. FFI-BPD = NEO Five-Factor Inventory Borderline Personality Disorder composite score.

aN = 398, $R^2 = .55$. bN = 398, $R^2 = .71$.

*p < .003*
3.1.2 SPAN Results

Next, I sought to replicate and extend the findings from the ADRC sample in the SPAN sample. The self- and informant report FFI-BPD scores were submitted to a principal components analysis. Eigenvalues for the first two factors were 1.47 and 0.53, and the first factor accounted for 73.32% of the variance in the two measures. As such, the principal components analysis met my a priori criteria to proceed with using the latent factor score for the remaining analyses. Factor scores in the SPAN sample ranged from -4.20 to 7.30 ($M = 0, SD = 1.16$).

Cross-sectional correlations between the SPAN PD measures and cognitive aging outcomes are presented in Table 5. Consistent with my hypotheses and with the results from the ADRC sample, FFI-BPD factor scores at SPAN Time 1 showed a weak, negative association with number of words recalled on the immediate free recall task, $r(1002) = -.08$, 95% CI [-.14, -.02], and a moderate, positive association with informant reported cognitive problems on the AD8, $r(701) = .33$, 95% CI [.26, .40]. Surprisingly, the correlation between AD8 scores and recall was weak and the 95% confidence interval included zero, $r(661) = -0.07$, 95% CI [-.14, .01].

As with the ADRC analyses, I ran linear regression models predicting residualized change in informant reported cognitive problems and immediate free recall from FFI-BPD factor scores, baseline age, years of education, and gender (as before, all variables were standardized except for gender). The results for these models are found in Table 6. FFI-BPD factor scores showed no effect on change in free recall performance, Std. $b = -0.03$, 95% CI [-0.08, 0.02]. The results regarding informant reported cognitive problems as measured by the AD8 were in the expected direction, with higher FFI-BPD factor scores predicting increased AD8 scores at SPAN Time 3, Std. $b = 0.07$, 95% CI [0.01, 0.13]. However, the effect was weak and failed to reach the corrected statistical significance threshold of $p < .003$. 

Table 5
Correlations of SPAN Time 1 Personality Disorder Measures with Cognitive Aging and Interpersonal Stress Measures

<table>
<thead>
<tr>
<th>Variables</th>
<th>BPD</th>
<th>AVPD</th>
<th>OCPD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FFI-BPD</td>
<td>Self</td>
<td>Informant</td>
</tr>
<tr>
<td>Cognitive Aging</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free Recall(^a)</td>
<td>-.08*</td>
<td>-.08**</td>
<td>-.08*</td>
</tr>
<tr>
<td>Cognitive Problems(^b)</td>
<td>.33***</td>
<td>.15***</td>
<td>.39***</td>
</tr>
<tr>
<td>Interpersonal Stress</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stressful Life Events(^c)</td>
<td>.19***</td>
<td>.12*</td>
<td>.26***</td>
</tr>
<tr>
<td>Perceived Social Support(^d)</td>
<td>-.26***</td>
<td>-.26***</td>
<td>-.20***</td>
</tr>
</tbody>
</table>

Note. \(^a\)Ns range from 825 to 1048. \(^b\)Ns range from 691 to 703. Cognitive problems measured using the Ascertain Dementia Eight-item Informant Questionnaire. \(^c\)Ns range from 411 to 416. Stressful life events measured using List of Threatening Events Questionnaire at SPAN Time 3, covering the time period since SPAN Time 1. \(^d\)Ns range from 739 to 891. Perceived social support was measured using the general support subscale of the Quality of Relationship Inventory at SPAN Time 2.

\(^*p < .05. \(^{**p < .01. \(^{***p < .001.\)
Table 6

Summary of Regression Analyses Predicting SPAN Time 3 Free Recall and Cognitive Problems from FFI-BPD

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Free recall</th>
<th>Cognitive problems</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Std. b</td>
<td>SE</td>
</tr>
<tr>
<td>Intercept</td>
<td>0.11</td>
<td>0.04</td>
</tr>
<tr>
<td>FFI-BPD Factor</td>
<td>-0.03</td>
<td>0.02</td>
</tr>
<tr>
<td>Time 1 Age</td>
<td>-0.05</td>
<td>0.03</td>
</tr>
<tr>
<td>Years of Education</td>
<td>0.16</td>
<td>0.03</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.16</td>
<td>0.06</td>
</tr>
<tr>
<td>Time 1 Level of Outcome</td>
<td>0.25</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Note. All variables were standardized with the exception of gender. SPAN = St. Louis Personality and Aging Network. FFI-BPD = NEO Five-Factor Inventory Borderline Personality Disorder composite.

aN = 974, R² = .13. bN = 424, R² = .24
*p < .003
The next step was to examine the relationships among the self-report, informant report and structured interview PD measures included in the SPAN sample (Table 7) and determine if the borderline, avoidant, and obsessive-compulsive PD scales from these sources could each be characterized by a single latent dimension. For the BPD measures, the eigenvalues for the three dimensions were 1.69, 0.79, and 0.52. These dimensions accounted for 56.41%, 26.25% and 17.34% of the variance in the measures, respectively. The eigenvalues for the AVPD measures were 1.72, 0.83, and 0.45, and percentage of variance accounted for by each dimension was 57.37%, 27.55%, and 15.09%. Finally, the principal components analysis of the OCPD measures exhibited similar results, with eigenvalues of 1.58, 0.86, and 0.56, and dimensions accounting for 52.68%, 28.53%, and 18.79% of the variance. All three principal components analyses showed mixed results with regard to my a priori criteria for using the latent factor, with one dimension obtaining an eigenvalue greater than one but accounting for less than 60% of the variance. In keeping with my a priori criteria, I ran all analyses with each individual measure separately. However, given the mixed results with regard to my criteria, I also present the analyses with the latent factor scores to supplement the interpretation of the individual measure analyses.

Starting with the BPD symptom measures, I first examined the cross-sectional correlations with free recall and cognitive problems (Table 5). Just like the FFI-BPD, self-, informant and interview BPD symptom measures were all cross-sectionally correlated -0.08 with free recall at SPAN Time 1 ($df = 979, 790, 992$; 95% CIs [-.14, -.02], [-.15, -.01], [-.14, -.02], respectively). Next, I ran linear regression models predicting immediate free recall at SPAN Time 3, controlling for covariates and free recall performance at SPAN Time 1. As with the analysis predicting cognitive problems from FFI-BPD factor scores, the direction of the effect for self-report and interview BPD symptom scores was in the expected direction (Table 8). Higher scores
### Table 7
**Correlations Among Personality Disorder Measures at SPAN Time 1**

<table>
<thead>
<tr>
<th>Variables</th>
<th>BPD</th>
<th>AVPD</th>
<th>OCPD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FFI-BPD</td>
<td>Self</td>
<td>Informant</td>
</tr>
<tr>
<td>BPD</td>
<td>-</td>
<td>.47***</td>
<td>-</td>
</tr>
<tr>
<td>FFI-BPD</td>
<td></td>
<td>Self</td>
<td>.58***</td>
</tr>
<tr>
<td>Self</td>
<td></td>
<td>Informant</td>
<td>.39***</td>
</tr>
<tr>
<td>Informant</td>
<td></td>
<td>Interview</td>
<td></td>
</tr>
<tr>
<td>AVPD</td>
<td>Self</td>
<td>.34***</td>
<td>.60***</td>
</tr>
<tr>
<td></td>
<td>Informant</td>
<td>.42***</td>
<td>.22***</td>
</tr>
<tr>
<td>OCPD</td>
<td>Self-report</td>
<td>.21***</td>
<td>.48***</td>
</tr>
<tr>
<td></td>
<td>Informant</td>
<td>.24***</td>
<td>.13***</td>
</tr>
<tr>
<td></td>
<td>Interview</td>
<td>.27***</td>
<td>.28***</td>
</tr>
</tbody>
</table>

*Note. Ns range from 825 to 1058. SPAN = St. Louis Personality and Aging Network. BPD = Borderline personality disorder. AVPD = Avoidant personality disorder. OCPD = Obsessive-compulsive personality disorder. FFI-BPD = NEO Five-Factor Inventory Borderline Personality Disorder composite. Self and informant report obtained using the Multisource Assessment of Personality Pathology. Interview obtained using the Structured Interview for DSM-IV Personality.*

*p < .05. ***p < .001.
Table 8
Summary of Regression Analyses Predicting SPAN Time 3 Free Recall from BPD Symptoms

<table>
<thead>
<tr>
<th>Outcome: Free Recall</th>
<th>BPD measure: Factor&lt;sup&gt;a&lt;/sup&gt;</th>
<th>BPD measure: Self-report&lt;sup&gt;b&lt;/sup&gt;</th>
<th>BPD measure: Informant report&lt;sup&gt;c&lt;/sup&gt;</th>
<th>BPD measure: Interview&lt;sup&gt;d&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Std.  b</td>
<td>SE</td>
<td>t</td>
<td>95% CI</td>
</tr>
<tr>
<td>Intercept</td>
<td>0.11</td>
<td>0.04</td>
<td>2.70</td>
<td>[0.03, 0.19]</td>
</tr>
<tr>
<td>BPD measure</td>
<td>-0.05</td>
<td>0.02</td>
<td>-1.98</td>
<td>[-0.10, 0.00]</td>
</tr>
<tr>
<td>Time 1 Age</td>
<td>-0.05</td>
<td>0.03</td>
<td>-1.82</td>
<td>[-0.11, 0.00]</td>
</tr>
<tr>
<td>Years of Education</td>
<td>0.16</td>
<td>0.03</td>
<td>4.88*</td>
<td>[0.10, 0.23]</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.15</td>
<td>0.06</td>
<td>-2.50</td>
<td>[-0.27, -0.03]</td>
</tr>
<tr>
<td>Time 1 Free Recall</td>
<td>0.24</td>
<td>0.03</td>
<td>7.80*</td>
<td>[0.18, 0.31]</td>
</tr>
</tbody>
</table>

*Note. All variables were standardized with the exception of gender. SPAN = St. Louis Personality and Aging Network. BPD = Borderline personality disorder. Self and informant report obtained using Multisource Assessment of Personality Pathology. Interview represents Structured Interview for DSM-IV Personality.

<sup>a</sup>N = 974, R<sup>2</sup> = .13. <sup>b</sup>N = 952, R<sup>2</sup> = .13. <sup>c</sup>N = 771, R<sup>2</sup> = .13. <sup>d</sup>N = 964, R<sup>2</sup> = .13.

* p < .003.
on these instruments were associated with decreased performance on the free recall task (Both std. bs = -0.06, 95% CIs [-0.12, 0.00]), although once again this effect was weak and did not rise to the level of statistical significance. The effect for informant report symptoms was essentially zero (Std. b = -0.01, 95% CI [-0.08, 0.05]).

However, the analyses with informant reported cognitive problems as the outcome of interest told a different story. BPD symptom measures showed weak to moderate positive correlations with AD8 scores at SPAN Time 1: self-report, r(689) = .15, 95% CI [.08, .23]; informant report, r(695) = .39, 95% CI [.33, .45]; interview, r(692) = .26, 95% CI [.19, .33]. Furthermore, both self- and informant reported BPD symptoms predicted increases in cognitive problems over time (Table 9). For each one standard deviation increase in self- and informant reported BPD symptoms at SPAN Time 1, AD8 scores were expected to increase by 0.15 (95% CI [0.06, 0.24]) and 0.13 (95% CI [0.05, 0.22]) standard deviations, respectively. Conversely, the same analysis using the interview measure of BPD symptoms as the predictor found no effect (Std. b = 0.07, 95% CI [-0.01, 0.15]). Nevertheless, as a whole, the results suggest some support of my hypothesis that BPD symptoms would predict change in cognition over time, at least in terms of the cognitive problems that close others were observing in the target participants.

In contrast with the BPD findings, the AVPD symptom measures showed near-zero cross-sectional correlations with free recall at SPAN Time 1: self-report, r(979) = .02, 95% CI [-.04, .08]; informant report, r(787) = -.03, 95% CI [-.10, .04]; interview, r(992) = -.03, 95% CI [-.10, .03]. Given the lack of zero-order correlations at SPAN Time 1, it is unsurprising that the results of the linear regression models do not support my hypothesis that AVPD would predict change in free recall performance (Table 10). Similarly, AD8 scores at SPAN Time 1 were uncorrelated with AVPD symptoms as measured by self-report, r(689) = .05, 95% CI [-.03, .12], and
Table 9
Summary of Regression Analyses Predicting SPAN Time 3 Cognitive Problems from BPD Symptoms

<table>
<thead>
<tr>
<th></th>
<th>Outcome: Cognitive Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BPD measure: Factor&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Std.</td>
</tr>
<tr>
<td>Intercept</td>
<td>-0.03</td>
</tr>
<tr>
<td>BPD measure</td>
<td>0.12</td>
</tr>
<tr>
<td>Time 1 Age</td>
<td>0.12</td>
</tr>
<tr>
<td>Years of Education</td>
<td>-0.09</td>
</tr>
<tr>
<td>Gender</td>
<td>0.16</td>
</tr>
<tr>
<td>Time 1 Cognitive Problems</td>
<td>0.39</td>
</tr>
</tbody>
</table>

Note. All variables were standardized with the exception of gender. SPAN = St. Louis Personality and Aging Network. BPD = Borderline personality disorder. Cognitive problems were measured using the Ascertain Dementia Eight-item Informant Questionnaire. Self and informant report obtained using Multisource Assessment of Personality Pathology. Interview represents Structured Interview for DSM-IV Personality.

<sup>a</sup>N = 424, R² = .25. <sup>b</sup>N = 420, R² = .25. <sup>c</sup>N = 421, R² = .25. <sup>d</sup>N = 419, R² = .24.

<sup>*</sup>p < .003.
Table 10

*Summary of Regression Analyses Predicting SPAN Time 3 Free Recall from AVPD Symptoms*

<table>
<thead>
<tr>
<th>Outcome: Free Recall</th>
<th>AVPD measure: Factor(^a)</th>
<th>AVPD measure: Self-report(^b)</th>
<th>AVPD measure: Informant report(^c)</th>
<th>AVPD measure: Interview(^d)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Std. (b)</td>
<td>SE</td>
<td>t value</td>
<td>95% CI</td>
</tr>
<tr>
<td>Intercept</td>
<td>0.11</td>
<td>0.04</td>
<td>2.80</td>
<td>[0.03, 0.19]</td>
</tr>
<tr>
<td>AVPD measure</td>
<td>0.03</td>
<td>0.02</td>
<td>1.14</td>
<td>[-0.02, 0.08]</td>
</tr>
<tr>
<td>Time 1 Age</td>
<td>-0.05</td>
<td>0.03</td>
<td>-1.55</td>
<td>[-0.10, 0.01]</td>
</tr>
<tr>
<td>Years of Education</td>
<td>0.17</td>
<td>0.03</td>
<td>5.25*</td>
<td>[0.11, 0.24]</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.16</td>
<td>0.06</td>
<td>-2.69</td>
<td>[-0.28, -0.04]</td>
</tr>
<tr>
<td>Time 1 Free Recall</td>
<td>0.25</td>
<td>0.03</td>
<td>7.89*</td>
<td>[0.19, 0.31]</td>
</tr>
</tbody>
</table>

*Note.* All variables were standardized with the exception of gender. SPAN = St. Louis Personality and Aging Network. AVPD = Avoidant personality disorder. Self and informant report obtained using Multisource Assessment of Personality Pathology. Interview represents Structured Interview for DSM-IV Personality.

\(^a\)\(N = 974, R^2 = .13\). \(^b\)\(N = 952, R^2 = .13\). \(^c\)\(N = 768, R^2 = .13\). \(^d\)\(N = 964, R^2 = .13\).

*\(p < .003\)
interview, \( r(692) = .02, 95\% \text{ CI} [-.06, .09] \). Informant reported AVPD symptoms did show the expected correlation with AD8 scores, \( r(692) = .28, 95\% \text{ CI} [.21, .34] \). In terms of predicting residualized change in cognitive problems, self-reported and informant reported AVPD symptoms demonstrated positive, but non-significant, associations with increased cognitive problems, whereas interview based AVPD symptoms showed no association (Table 11).

The final analyses associated with Specific Aim 1 examined the relationship between OCPD symptoms and the cognitive measures. As predicted, self-reported OCPD symptoms, \( r(979) = -.01, 95\% \text{ CI} [-.07, .06] \), and informant reported OCPD symptoms, \( r(792) = -.02, 95\% \text{ CI} [-.09, .05] \), showed no association with free recall at SPAN Time 1. Interview reported OCPD symptoms, on the other hand, exhibited a weak, negative correlation, \( r(992) = -.09, 95\% \text{ CI} [-.16, -.03] \), similar in magnitude to the correlations between the BPD symptom measures and free recall performance. None of the OCPD measures predicted residualized change in free recall between SPAN Time 1 and Time 3 (Table 12). With regard to informant reported cognitive problems, there existed weak, positive correlations with OCPD symptoms as assessed using informant report, \( r(697) = .18, 95\% \text{ CI} [.11, .25] \), and interview, \( r(692) = .08, 95\% \text{ CI} [.01, .15] \), but a near-zero correlation with self-report OCPD symptoms, \( r(689) = 0.04, 95\% \text{ CI} [-0.03, 0.11] \). Contrary to expectations, informant reported OCPD symptoms did predict change in cognitive problems over time (Table 13). For each one standard deviation increase in informant reported OCPD symptoms, AD8 scores at SPAN Time 3 were expected to increase by 0.14 standard deviations (95% CI [0.06, 0.21]). Neither self-report nor interview showed the same effect.
Table 11
Summary of Regression Analyses Predicting SPAN Time 3 Cognitive Problems from AVPD Symptoms

<table>
<thead>
<tr>
<th></th>
<th>Outcome: Cognitive Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AVPD measure: Factor(^a)</td>
</tr>
<tr>
<td></td>
<td>Std.</td>
</tr>
<tr>
<td>Intercept</td>
<td>-0.06</td>
</tr>
<tr>
<td>AVPD measure</td>
<td>0.07</td>
</tr>
<tr>
<td>Time 1 Age</td>
<td>0.10</td>
</tr>
<tr>
<td>Years of Education</td>
<td>-0.11</td>
</tr>
<tr>
<td>Gender</td>
<td>0.20</td>
</tr>
<tr>
<td>Time 1 Cognitive</td>
<td>0.43</td>
</tr>
</tbody>
</table>

*Note. All variables were standardized with the exception of gender. SPAN = St. Louis Personality and Aging Network sample. AVPD = Avoidant personality disorder. Cognitive problems were measured using the Ascertain Dementia Eight-item Informant Questionnaire. Self and informant report obtained using Multisource Assessment of Personality Pathology. Interview represents Structured Interview for DSM-IV Personality.

\(^a\)N = 424, R^2 = .24. \(^b\)N = 420, R^2 = .24. \(^c\)N = 420, R^2 = .24. \(^d\)N = 419, R^2 = .23.

\(^*p < .003\)
Table 12

Summary of Regression Analyses Predicting SPAN Time 3 Free Recall from OCPD Symptoms

<table>
<thead>
<tr>
<th>Outcome: Free Recall</th>
<th>OCPD measure: Factor&lt;sup&gt;a&lt;/sup&gt;</th>
<th>OCPD measure: Self-report&lt;sup&gt;b&lt;/sup&gt;</th>
<th>OCPD measure: Informant report&lt;sup&gt;c&lt;/sup&gt;</th>
<th>OCPD measure: Interview&lt;sup&gt;d&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Std. b</td>
<td>SE</td>
<td>t value</td>
<td>95% CI</td>
</tr>
<tr>
<td>Intercept</td>
<td>0.12</td>
<td>0.04</td>
<td>2.88</td>
<td>[0.04, 0.20]</td>
</tr>
<tr>
<td>OCPD measure</td>
<td>0.02</td>
<td>0.02</td>
<td>0.84</td>
<td>[-0.03, 0.07]</td>
</tr>
<tr>
<td>Time 1 Age</td>
<td>-0.05</td>
<td>0.03</td>
<td>-1.57</td>
<td>[-0.11, 0.01]</td>
</tr>
<tr>
<td>Years of Education</td>
<td>0.17</td>
<td>0.03</td>
<td>5.19*</td>
<td>[0.11, 0.23]</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.17</td>
<td>0.06</td>
<td>-2.81</td>
<td>[-0.29, -0.05]</td>
</tr>
<tr>
<td>Time 1 Free Recall</td>
<td>0.25</td>
<td>0.03</td>
<td>7.92*</td>
<td>[0.19, 0.31]</td>
</tr>
</tbody>
</table>

Note. All variables were standardized with the exception of gender. SPAN = St. Louis Personality and Aging Network. OCPD = Obsessive-compulsive personality disorder. Self and informant report obtained using Multisource Assessment of Personality Pathology. Interview represents Structured Interview for DSM-IV Personality. 

<sup>a</sup>N = 974, R² = .13.  
<sup>b</sup>N = 952, R² = .13.  
<sup>c</sup>N = 773, R² = .13.  
<sup>d</sup>N = 964, R² = .13.  

* p < .003
Table 13

Summary of Regression Analyses Predicting SPAN Time 3 Cognitive Problems from OCPD Symptoms

<table>
<thead>
<tr>
<th>Outcome: Cognitive Problems</th>
<th>OCPD measure: Factor(^a)</th>
<th>OCPD measure: Self-report(^b)</th>
<th>OCPD measure: Informant report(^c)</th>
<th>OCPD measure: Interview(^d)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Std. (b)</td>
<td>(SE)</td>
<td>(t) value</td>
<td>95% CI</td>
</tr>
<tr>
<td>Intercept</td>
<td>-0.04</td>
<td>0.05</td>
<td>-0.75</td>
<td>[-0.15, 0.07]</td>
</tr>
<tr>
<td>OCPD measure</td>
<td>0.07</td>
<td>0.03</td>
<td>2.11</td>
<td>[0.00, 0.14]</td>
</tr>
<tr>
<td>Time 1 Age</td>
<td>0.10</td>
<td>0.04</td>
<td>2.44</td>
<td>[0.02, 0.17]</td>
</tr>
<tr>
<td>Years of Education</td>
<td>-0.11</td>
<td>0.04</td>
<td>-2.65</td>
<td>[-0.20, -0.03]</td>
</tr>
<tr>
<td>Gender</td>
<td>0.17</td>
<td>0.08</td>
<td>2.12</td>
<td>[0.01, 0.33]</td>
</tr>
<tr>
<td>Time 1 Cognitive Problems</td>
<td>0.43</td>
<td>0.05</td>
<td>9.45(^*)</td>
<td>[0.34, 0.52]</td>
</tr>
</tbody>
</table>

\(^a\)Note. All variables were standardized with the exception of gender. SPAN = St. Louis Personality and Aging Network. OCPD = Obsessive-compulsive personality disorder. Cognitive problems were measured using the Ascertain Dementia Eight-item Informant Questionnaire. Self and informant report obtained using Multisource Assessment of Personality Pathology. Interview represents Structured Interview for DSM-IV Personality.  
\(^b\)N = 424, \(R^2 = .24\). \(^c\)N = 420, \(R^2 = .23\). \(^d\)N = 423, \(R^2 = .25\). \(^e\)N = 419, \(R^2 = .24\).  
\(^*\)\(p < .003\)
3.2 Specific Aim 2 Results

Once the relationships between personality pathology and cognitive aging outcomes had been established, the next step was to examine the potential role of interpersonal stress measures in mediating those relationships. As with the regression models, all variables were standardized before running each model, with the exception of gender and presence/absence of interpersonal stressful life events. Given that BPD and AVPD symptoms did not predict change in free recall, it is unsurprising that no mediating effects were found through interpersonal stressful life events or perceived social support with regard to that outcome. For reference, indirect effect estimates and 95% confidence intervals for the planned path analyses are presented in Table 14.

Results for the planned path analyses with informant reported cognitive problems as the outcome are presented in Table 15. Figures 1 through 4 show the pathways for the PD variables that did predict change in cognitive problems, self- and informant reported BPD. There was no indirect effect of BPD symptoms as measured using either of these sources on cognitive problems through interpersonal stressful life events (self-report: $ab = -0.00$, 95% CI [-0.02, 0.01]; informant report: $ab = -0.01$, 95% CI [-0.04, 0.01]). Neither was there an indirect effect of self- or informant report BPD symptoms on cognitive problems through perceived social support (self-report: $ab = 0.01$, 95% CI [-0.00, 0.03]; informant report: $ab = 0.01$, 95% CI [-0.00, 0.03]). Table 15 also indicates the results of the exploratory test of whether the interpersonal stress measures mediated the relationship between informant reported OCPD symptoms and cognitive problems, and the path diagrams are shown in Figures 5 and 6. Again, there was no mediation effect through interpersonal stressful life events, $ab = -0.00$, 95% CI [-0.01, 0.00], or perceived social support, $ab = 0.01$, 95% CI [-0.00, 0.02]. As would be expected given the results of the
earlier regression analyses, none of the other PD variables showed an indirect effect on cognitive problems through interpersonal stressful life events or perceived social support.
Table 14

*Indirect Effects of PD Symptoms on Free Recall through Interpersonal Stress*

<table>
<thead>
<tr>
<th>PD Measure</th>
<th>Mediator: Interpersonal Stressful Life Events</th>
<th>Mediator: Perceived Social Support</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Std. ab [95% CI]</td>
<td>Std. ab [95% CI]</td>
</tr>
<tr>
<td>Borderline PD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-report</td>
<td>0.002 [-0.002, 0.008]</td>
<td>0.003 [-0.010, 0.017]</td>
</tr>
<tr>
<td>Informant report</td>
<td>0.004 [-0.005, 0.014]</td>
<td>0.001 [-0.013, 0.015]</td>
</tr>
<tr>
<td>Interview</td>
<td>0.007 [-0.003, 0.018]</td>
<td>0.001 [-0.007, 0.009]</td>
</tr>
<tr>
<td>Avoidant PD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-report</td>
<td>-0.000 [-0.003, 0.002]</td>
<td>-0.000 [-0.018, 0.017]</td>
</tr>
<tr>
<td>Informant report</td>
<td>0.001 [-0.002, 0.005]</td>
<td>-0.001 [-0.011, 0.009]</td>
</tr>
<tr>
<td>Interview</td>
<td>0.001 [-0.003, 0.006]</td>
<td>-0.002 [-0.016, 0.011]</td>
</tr>
</tbody>
</table>

*Note.* $N = 974$. Models partialled out initial level of the outcome, initial age, years of education, and gender from the outcome. All variables except for gender and presence/absence of interpersonal stressful life events were standardized. PD = Personality disorder. Self and informant report obtained using Multisource Assessment of Personality Pathology. Interview represents Structured Interview for *DSM-IV* Personality. Interpersonal stressful life events were assessed using the List of Threatening Events Questionnaire, and perceived social support using the Quality of Relationships Inventory.
### Table 15

**Indirect Effects of PD Symptoms on Cognitive Problems through Interpersonal Stress**

<table>
<thead>
<tr>
<th>PD Measure</th>
<th>Mediator: Interpersonal Stressful Life Events</th>
<th>Mediator: Perceived Social Support</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Std. $ab$ [95% CI]</td>
<td>Std. $ab$ [95% CI]</td>
</tr>
<tr>
<td>Borderline PD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-report</td>
<td>-0.004 [-0.019, 0.008]</td>
<td>0.013 [-0.002, 0.032]</td>
</tr>
<tr>
<td>Informant report</td>
<td>-0.010 [-0.036, 0.010]</td>
<td>0.011 [-0.001, 0.027]</td>
</tr>
<tr>
<td>Interview</td>
<td>-0.006 [-0.026, 0.012]</td>
<td>0.009 [-0.001, 0.025]</td>
</tr>
<tr>
<td>Avoidant PD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-report</td>
<td>-0.000 [-0.007, 0.004]</td>
<td>0.013 [-0.000, 0.032]</td>
</tr>
<tr>
<td>Informant report</td>
<td>-0.001 [-0.008, 0.005]</td>
<td>0.006 [-0.001, 0.017]</td>
</tr>
<tr>
<td>Interview</td>
<td>-0.001 [-0.008, 0.005]</td>
<td>0.017 [0.002, 0.038]</td>
</tr>
<tr>
<td>Obsessive-compulsive PD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Informant report</td>
<td>-0.000 [-0.006, 0.004]</td>
<td>0.005 [-0.001, 0.016]</td>
</tr>
</tbody>
</table>

*Note.* $N = 424$. Models partialled out initial level of the outcome, initial age, years of education, and gender from the outcome. All variables except for gender and presence/absence of interpersonal stressful life events were standardized. PD = Personality disorder. Self and informant report obtained using Multisource Assessment of Personality Pathology. Interview represents Structured Interview for *DSM-IV* Personality. Interpersonal stressful life events were assessed using the List of Threatening Events Questionnaire, and perceived social support using the Quality of Relationships Inventory.
Figure 1 Path analysis of effect of self-reported BPD symptoms on cognitive problems through interpersonal stressful life events

Note. N = 424. Model partialled out initial level of the outcome, initial age, years of education, and gender from the outcome. All variables except for gender and presence/absence of interpersonal stressful life events were standardized. BPD = Borderline personality disorder.

*p < .003

Figure 2 Path Analysis of effect of informant reported BPD symptoms on cognitive problems through interpersonal stressful life events

Note. N = 424. Model partialled out initial level of the outcome, initial age, years of education, and gender from the outcome. All variables except for gender and presence/absence of interpersonal stressful life events were standardized. BPD = Borderline personality disorder.

*p < .003
Figure 3 Path analysis of effect of self-reported BPD symptoms on cognitive problems through perceived social support

Note. $N = 424$. Model partialled out initial level of the outcome, initial age, years of education, and gender from the outcome. All variables except for gender were standardized. BPD = Borderline personality disorder.

*p < .003

Figure 4 Path analysis of effect of informant reported BPD symptoms on cognitive problems through perceived social support

Note. $N = 424$. Model partialled out initial level of the outcome, initial age, years of education, and gender from the outcome. All variables except for gender were standardized. BPD = Borderline personality disorder.

*p < .003
Figure 5 Path analysis of effect of informant reported OCPD symptoms on cognitive problems through interpersonal stressful life events

Note. N = 424. Model partialled out initial level of the outcome, initial age, years of education, and gender from the outcome. All variables except for gender and presence/absence of interpersonal stressful life events were standardized. OCPD = Obsessive-compulsive personality disorder.
*p < .003

Figure 6 Path analysis of effect of informant reported OCPD symptoms on cognitive problems through perceived social support

Note. N = 424. Model partialled out initial level of the outcome, initial age, years of education, and gender from the outcome. All variables except for gender were standardized. OCPD = Obsessive-compulsive personality disorder.
*p < .003
Chapter 4: Discussion

4.1 Specific Aim 1 Conclusions

Based on previous work on the relationship between personality (both normal-range and pathological) and cognitive abilities, I made several predictions regarding the concurrent and prospective associations between personality pathology and cognitive aging outcomes (Specific Aim 1). Overall, the current results provided a fair amount of support for these hypotheses. BPD features showed modest cross-sectional associations with poorer memory performance, worse overall cognitive ability, higher clinical dementia stage, and more informant reported problems due to changes in cognitive ability in both the ADRC and SPAN samples (Hypothesis 1.1, 1.3 and 1.4). Additionally, self- and informant report measures of BPD symptoms showed predictive utility with regard to changes in informant reported cognitive problems over time (Hypothesis 1.5). Contrary to my hypotheses, BPD features did not show a prospective relationship with changes in memory or general cognitive ability (Hypothesis 1.2 and 1.3). In terms of the other two manifestations of personality pathology, AVPD was for the most part unrelated to the cognitive aging outcomes, whereas OCPD showed some small associations with cognitive problems (Hypothesis 1.4). Neither AVPD nor OCPD generally exhibited a prospective relationship with free recall or cognitive problems, with the one exception of informant reported OCPD symptoms predicting cognitive problems (Hypothesis 1.5). In conclusion, BPD symptoms largely appear to perform as expected, with the major caveat that they did not predict change in neuropsychological measures of cognitive ability and memory over time. Meanwhile, although AVPD is similar to BPD in terms of its associations with negative affectivity/neuroticism, it did not exhibit the same pattern of relationships with cognitive outcomes. Finally, the current findings may offer some insight into the source of the ambiguity surrounding the relationship
between OCPD symptoms and cognition, as it appears that only informant report of OCPD symptoms was related to cognitive problems. These conclusions illuminate the role that personality pathology, particularly BPD, may play in the detection of risk for cognitive decline.

4.2 Specific Aim 2 Conclusions

In addition to enhanced prediction, the current investigation aimed to explore how personality pathology may add to our understanding of cognitive aging through the identification of potential mechanisms. As such, I sought to examine whether interpersonal stress mediated the relationship between personality pathology and cognitive changes (Specific Aim 2). The current findings uncovered no evidence of an indirect effect of personality pathology on cognitive aging through interpersonal stressful life events or perceived social support (Hypothesis 2.1 and 2.2). Put another way, the observed direct relationships between personality pathology and changes in cognitive problems were largely preserved when accounting for interpersonal stress. Interpersonal functioning cannot be ruled out as a possible contributor to the link between personality and poor cognitive outcomes in later life. However, the current analyses were not able to detect such an effect over a two-year time course using the measures available in these studies. Future research would need to consider whether this was a sufficiently long time course to observe such an effect. It might also benefit from examining other markers of interpersonal functioning beyond interpersonal stressful life events and perceived social support. In reality, it is unlikely that any one mechanism accounts for very much of the variance in individual differences in cognitive aging. As such, it is important to consider multiple mechanisms that stand to improve our understanding of how people differ in their trajectories of cognitive functioning across older adulthood.
4.3 Contributions to the Field
Overall, the current investigation meaningfully advances the existing literature on the relationship between personality pathology and cognitive aging. By comparing findings across two datasets with varying degrees of granularity in the assessment of personality pathology and cognitive ability, the current investigation offers a clearer understanding of this relationship than either study would provide on its own. In particular, the current investigation provides three major contributions to the field. First, the current analyses provide evidence that BPD features are associated with cognitive ability in later life and prospectively predict changes in informant reported cognitive problems. These findings add fuel to the growing recognition that BPD features, though less prevalent in later life, continue to exert a meaningful, negative impact on aging-related health and well-being outcomes (Beatson et al., 2016; Rosowsky & Gurian, 1992).

Of note, the prospective effect of BPD features on cognitive problems was not found when BPD was measured using the trait profile as measured by the NEO FFI. As the NEO FFI items are written primarily to capture the adaptive range of personality traits, it may be that clinical indicators of BPD capture additional information relevant to understanding cognitive difficulties in later life. Furthermore, there was no prospective association between BPD features and change in neuropsychological assessments of memory. The clinical literature has long recognized the value of informant report for early detection of risk for cognitive decline and dementia (Carr, Gray, Baty, & Morris, 2000; Jorm et al., 1996). One interpretation of this result is that informants were picking up on early signs of cognitive impairments in complex tasks of daily living that were not yet affecting performance on explicit tests of memory. The idea that clinical features of BPD yield important information about cognitive difficulties at the level of everyday tasks is an intriguing one that deserves further exploration.
The second major contribution of the current investigation to the literature is that it has helped identify the boundaries of the relationship between personality pathology and cognitive aging. For example, it found no evidence of a prospective relationship in either sample between personality pathology and neuropsychological measures of cognitive ability and memory over a period of approximately two years. Much of the previous work that used explicit measures of personality pathology has been cross-sectional in nature or asked for retrospective reports of participants’ premorbid personality (Cruitt & Oltmanns, 2018b; Dondu et al., 2015; Henriques-Calado & Duarte-Silva, 2019; Nicholas et al., 2010; Pilleron et al., 2015; Prior et al., 2016) and may overestimate the predictive utility of personality pathology for understanding decline in discrete cognitive abilities. At the same time, the follow-up period in question was shorter than typical studies of normal-range personality and cognitive ability (Curtis et al., 2015). Therefore, the possibility that this relationship plays out over a longer time course remains open. Beyond the temporal dynamics, the current investigation also establishes some boundaries with regard to potential mechanisms. It found no evidence that interpersonal stress mediated an effect of personality pathology on cognitive decline. As mentioned above, whether this mediating role could be detected with alternative measures over a longer time course remains to be determined. Nevertheless, the fact that the current investigation did not find a mediating role for interpersonal stress will need to be taken into account when designing future studies.

Finally, the current investigation provides an important methodological contribution to the field by once again confirming that the source of personality information matters. For example, BPD features as assessed by interview failed to show the same prospective effect on cognitive problems as when they were assessed using self- and informant report. Although all three sources contribute unique, valid information to the prediction of various outcomes (Cruitt &
Oltmanns, 2018b), previous research suggests that interview-based assessments of BPD features tend to be more conservative (Hopwood et al., 2008). As such, interview may provide more information at higher levels of dysfunction than is typically found in community samples of participants. Further evidence that the source of information about personality pathology matters comes from the OCPD findings. These results appear to add to the ambiguous conclusions in the literature (Dondu et al., 2015; Henriques-Calado & Duarte-Silva, 2019; Nicholas et al., 2010). Informant reported OCPD features demonstrated an association with informant reported cognitive problems, whereas self- and interview-based assessments did not. The latter result could be attributed to shared source variance. Close others may tend to describe difficult individuals in an overall negative manner, which may explain why previous studies that used a mix of self- and informant report to assess dementia patients based on severity may overestimate the association between OCPD and dementia status. This same bias may in part explain the findings with regard to BPD features, although the fact that self-report BPD features also showed an association with cognitive problems suggests that some variance in cognitive problems due to personality pathology goes beyond a shared source. Nevertheless, the current findings tell a cautionary tale, and argue for the use of multiple sources of information when examining the relationship between personality and cognitive decline.

4.4 Limitations
Although the current investigation contributes to our knowledge about personality pathology and cognitive aging, it does have a number of limitations. Given that much of the previous research on personality pathology and cognitive aging relied on cross-sectional and retrospective analyses, the current investigation’s ability to examine this relationship prospectively over two waves of data is a significant strength. However, there are at least three limitations stemming
from the use of only two waves of data. First, there was relatively high rank-order stability in
cognitive ability and low rates of conversion to more severe stages of dementia over the two
waves of data used in the current investigation from the ADRC sample. Incorporating additional
waves of data collection as these longitudinal studies unfold will allow for modeling trajectories
of change in cognitive ability over time. Furthermore, the average participant at this point in the
ADRC and SPAN studies was in their late sixties, which is relatively young to be exhibiting
symptoms of cognitive decline. Studying participants from early into late older adulthood will
increase the chances of detecting risk factors for later change. Finally, the assessment of the
interpersonal stress variables in SPAN that occurred between Time 1 and Time 3 did allow for
mediation analyses that followed an appropriate time course. Nevertheless, a more rigorous
analysis of mediation would involve measuring all three variables of interest at each of three
time points.

Another feature of the current investigation that may limit the generalizability of its conclusions
involves the use of the AD8. First, fewer informants completed SPAN Time 3 than anticipated,
leading to a much lower sample size for the AD8 analyses than the free recall analyses.
However, the sample size was comparable to the analyses using the ADRC data. Another
potential limitation of using the AD8 is that the time frame that informants are asked to consider
(“the last several years”) is deliberately vague to enhance its use as a screening measure for
cognitive decline. However, this leads to a lack of temporal precision and complicates the
interpretation of residualized change in the AD8. It is possible that informants were considering
overlapping periods of time when filling out the AD8 at both time points, potentially masking
change that did occur. Furthermore, there was a near-zero correlation between AD8 and the
immediate free recall task in SPAN. As mentioned above, this finding may be due to informants
picking up on impairments in complex daily tasks that represent how decline manifests in everyday functioning more so than an explicit memory task. However, this is an open empirical question and it may be that informant reports of cognitive problems are biased by the participant’s personality. Future research will need to examine whether informant reports of cognitive problems map on to daily cognitive impairments through more frequent and detailed assessments. Ideally, such work would account for personality functioning as well, to ensure that informants are not conflating problems due to personality and cognitive functioning.

4.5 Implications and Future Directions
Despite its limitations, the current investigation offers some important implications for the broader literature. These implications fall under three primary areas of study: 1) biopsychosocial mechanisms of cognitive aging, 2) interventions to improve cognitive aging, and 3) interventions for personality pathology in later life. With regard to the first, future research on potential biopsychosocial mechanisms may need to grapple with the current investigation’s failure to find evidence supporting a mediating role of interpersonal stress in the personality pathology-cognitive aging relationship. Interpersonal stress represents a particularly straightforward example of stress generation in personality pathology, and the current findings bear out this relationship. Increased stress, including interpersonal stress (e.g., in the context of marriage or caregiving), is associated with increased inflammation (Gouin, Glaser, Malarkey, Beversdorf, & Kiecolt-Glaser, 2012; Kiecolt-Glaser, 2018), which is in turn associated with increased rates of cognitive decline (Bradburn, Sarginson, & Murgatroyd, 2018; Dziedzic, 2006; Swardfager et al., 2010). Therefore, one might theorize a relatively clear causal pathway linking personality pathology, interpersonal stress, inflammation, and cognitive decline. The current investigation failed to find an effect for the first leg of this mediation. As the SPAN study unfolds and obtains
additional waves of stress and inflammation biomarker data it will become possible to fully test this proposed model. If it turns out that personality pathology does not influence cognitive aging through stress/inflammation, researchers will need to revisit the previous literature and generate alternative pathways.

Future research will also have the opportunity to draw out the current investigation’s implications for intervening in cognitive aging. Just as some evidence suggests that negative social interactions increase risk for cognitive decline (Wilson, Boyle, James, et al., 2015), other studies have established that engagement in positive social activities promotes cognitive maintenance in older adults (Hertzog, Kramer, Wilson, & Lindenberger, 2009; Smith, 2016). These activities, in turn, show meaningful associations with personality traits, such that any interventions that seek to enhance engagement in these activities need to account for individual differences (Hill & Payne, 2017). The current findings suggest that the extent to which poor interpersonal functioning, social engagement, and personality characteristics are interrelated in terms of their effect on cognitive aging still needs to be explored. For example, one question prompted by the current analyses might be whether the salutary effect of social engagement on cognitive aging is mitigated when it is accompanied by highly conflictual social relationships, as might be the case in BPD. Such a pattern could be compared to the effect of social withdrawal (as seen in AVPD), which limits the opportunity for both positive and negative social interactions. Exploring these questions could lead to divergent recommendations for individuals with various personality features in terms of cognitive maintenance strategies, rather than blanket suggestions promoting social engagement wholesale. This line of research will require extensive effort to obtain in-depth measures of social functioning alongside personality and cognitive aging.
but holds the potential to offer more individualized approaches to maintaining cognitive ability in later life.

Beyond interventions targeted toward cognitive aging outcomes, the current findings also contribute to our understanding of treating personality pathology in later life. Several case studies highlight the difficulty of distinguishing between personality pathology and dementia in the diagnostic process (Greve, Curtis, & Bianchini, 2007; Greve, Curtis, Bianchini, & Collins, 2004; Hellwig, Dykierek, Hellwig, Zweremmann, & Meyer, 2012; Helmes & Steward, 2010). The current results suggest that diagnostic clarity would be enhanced if the source of information about the patient’s behavior were taken into account. For example, informants may have difficulty distinguishing between OCPD features (e.g., rigidity and stubbornness) and problems due to cognitive changes (e.g., problems making decisions or difficulty learning how to use a new device). In addition, several reviews point to the strain put on the healthcare system by late-life personality pathology (Beatson et al., 2016; Rosowsky & Gurian, 1992), which is likely compounded by cognitive decline. There is currently a small, but growing, literature on the efficacy of PD treatments adapted to the context of later life (Khasho et al., 2019; Lynch et al., 2007; Van Dijk et al., 2019; Videler, Rossi, Schoevaars, Van Der Feltz-Cornelis, & Van Alphen, 2014). Future work will need to consider the ability of participants with cognitive decline to engage in these treatments, and the appropriate level of treatment to provide in that context (Videler et al., 2015). Research in this area will need to move beyond the ‘personality as risk factor’ approach adopted in the current investigation and study the manifestation of personality pathology within populations experiencing cognitive decline. In so doing, personality pathology interventions may be identified that hopefully not only slow cognitive aging, but also improve quality of life for those who are suffering in older adulthood.
In order to pursue this line of research, it is important to translate the current findings across different conceptualizations of personality pathology. A central premise of the current investigation is that PD features provide additional information at the maladaptive extremes of personality (Samuel et al., 2010; Suzuki et al., 2015). Extrapolating from this premise, conclusions drawn from measures of these symptoms may translate into insights regarding the underlying patterns of pathological personality traits. One way to understand the current findings in these terms is that BPD features offer particularly salient markers of the maladaptive combination of high neuroticism/negative affectivity and low conscientiousness/high disinhibition (American Psychiatric Association, 2013). As such, they are useful for understanding the role of personality in predicting negative outcomes common to both traits. Alternatively, some evidence suggests that BPD features are indicators of a general factor of personality pathology (Sharp et al., 2015). That is, BPD features may have less to do with an individual’s particular stylistic manifestation of personality pathology. They may instead better reflect the overall severity of personality dysfunction that an individual is experiencing. In that case, the current findings would suggest that it is personality dysfunction broadly speaking rather than specific traits (like negative affectivity) that is associated with increased cognitive problems over time. Future research may benefit from using dimensional personality pathology measures that attempt to cover the full range of the underlying latent traits.

Ultimately, the current investigation represents an incremental advancement of our understanding of the relationship between personality and cognitive ability in later life. The relationship between BPD features and cognitive problems appears similar in magnitude to previous findings with regard to normal-range personality traits (Curtis et al., 2015; Low et al., 2013; Luchetti et al., 2016; Terracciano et al., 2017, 2014). Nevertheless, the maladaptive
extremes of personality have an important role to play with regard to our approach to identifying potential mechanisms involved in cognitive aging and interventions aimed to promote cognitive maintenance and positive psychological functioning in later life. Researchers interested in pursuing this line of research will need to cut across the subdisciplines of clinical, personality, and aging psychology, fostering opportunities for collaboration that extend beyond the questions at hand and deepen our understanding of how to leverage individual differences to help people live well in later life.
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