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Mistrustful and Misunderstood: A Review of Paranoid Personality Disorder

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Abstract

Purpose of review—Paranoid Personality Disorder (PPD) has historically been neglected by science out of proportion to its prevalence or its association with negative clinical outcomes. This review provides an update on what is known about PPD regarding its prevalence, demographics, comorbidity, biological mechanism, risk factors, and relationship to psychotic disorders.

Recent Findings—PPD has long been the subject of a rich and prescient theoretical literature which has provided a surprisingly coherent account of the psychological mechanism of non-delusional paranoia. Available data indicate that PPD has a close relationship with childhood trauma and social stress. Descriptive data on a sample of 115 individuals with Paranoid Personality Disorder is examined in comparison with a group of individuals with Borderline Personality Disorder. The descriptive data largely confirm previously identified relationships between Paranoid Personality Disorder and childhood trauma, violence, and race. We identify important similarities to and differences from Borderline Personality Disorder.

Summary—PPD continues to be an important construct in the clinic and the laboratory. Available data lead to a reconsideration of the disorder as more closely related to trauma than to schizophrenia.

Keywords

Paranoid Personality Disorder; Borderline Personality Disorder; Paranoia; Childhood Trauma; Corticotropin-Releasing Hormone; Personality Disorder

INTRODUCTION

Paranoid Personality Disorder (PPD) seems destined to be misunderstood. It was once theorized to be associated with schizophrenia due to the phenomenological similarity of suspiciousness to paranoid delusion, but the evidence for this association is not strong.

Compliance with Ethics Guidelines

Conflict of Interest

Dr. Royce Lee declares that he has no conflicts of interest.

Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

Clinically, it is overlooked, or its symptoms are accounted for by comorbid personality disorders. Science has neglected PPD; individuals with PPD are slow to volunteer for research studies and it has not been a priority for research funding. And yet, the clinical reality is that PPD is a severe, relatively common clinical problem that is difficult to treat. Fortunately, our understanding of PPD has improved as research has accrued.

The reader can be directed to several excellent reviews of PPD. In a discussion of PPD in light of the transition from DSM-IV to 5, an argument was made to rethink PPD as the dimensional representation of trait-like suspicious hostility, rather than a categorical syndrome [1]. A recent review by Carroll [2] stressed its place in a pathway leading to violence in clinical and forensic settings. Bernstein and Useda [3] [3] provide a scholarly review of the rich psychological literature on PPD, and recalibrate the relationship between DSM-IV/5 descriptions and what is found in the population.

This review of PPD proposes that examining PPD in the context of childhood trauma provides a useful framework with which to integrate psychological theories regarding PPD, empirical data, and clinical practice. We will summarize the current and past literature on PPD. Then we will provide descriptive data on one of the largest samples of PPD samples to date. We will conclude by identifying important unanswered questions regarding these mistrustful and misunderstood humans.

Nosology

In the psychiatric nosology, DSM-IV and 5 describe PPD as a disorder of suspicious, unforgiving, ruminative, and jealous traits [4] [5]. In addition to suspiciousness, ICD-10 PPD includes traits of excessive self-importance and hostility, and further proposes subtypes of the expansive, fanatic, querulant and sensitive paranoid personality [6]. In all nosologies, PPD excludes psychotic symptoms, including paranoid delusions and hallucinations, commonly encountered in classic psychiatric syndromes like schizophrenia, schizoaffective, and psychotic states of bipolar disorder. Paranoid thoughts may also develop in Alzheimer's Dementia [7] and after acquired brain injury [8]. However, these conditions occur with distinct presentations and longitudinal course. An intriguing correlate of PPD is recognized in the forensic literature regarding persistent litigants, who can clog court systems with unending and unwinnable litigation (Lester et al., 2004).

Dimensional systems of diagnosis are under development that provide a glimpse of the future of the PPD diagnosis in the clinic. The DSM5 research criteria [9] and NIMH Research Domain Criteria (rDOC) system [10] largely succeed in capturing the essential features of PPD. The dimensional traits proposed in the DSM 5 have been confirmed to map onto the Five Factor Model (FFM) constructs in pairwise fashion, with negative affectivity/ neuroticism, antagonism/ agreeableness, detachment/ extraversion, disinhibition/ conscientiousness, psychoticism/ openness to experience [11]. FFM constructs are implicitly biological, with assumed genetic contributions. The rDOC system is explicitly biological, with the ambition of creating a brain-based nosology from the "bottom up". Facets of PPD can be recognized in Negative Valence Systems and Systems for Social Processes. How

these can be integrated into an approach in the clinic will depend on the results of ongoing research.

Why is Paranoid PD Important?

Out of proportion with the scant attention dedicated to it, PPD powerfully predicts important adverse outcomes in the treatment of personality disordered patients. Epidemiological data from the United States indicates the PPD is a significant cause of disability [12]. Australian epidemiological research confirms the American findings, with PPD contributing to disability independent of the effects of other personality disorders [13]. Persons with PPD, when not disabled, stop working earlier than non-personality disordered individuals [14]. In clinical populations, it is one of the strongest predictors of aggressive behavior ([15], reviewed in [16]). In the forensic realm, PPD is associated with violence and stalking [17] as well as excessive litigation [18]. In the clinic individuals with PPD are prone to depression and have a negative prognosis, despite intensive psychiatric treatment [19] [20]. The risk of suicide and suicide attempts in PPD remains obscure, with little or no data available regarding this important clinical problem. However, because it is often comorbid with other personality disorders that are associated with elevated suicide risk, one can infer the risk of suicide in PPD individuals to be high, if only due to comorbidity with conditions such as Borderline Personality Disorder (BPD) [21].

Prevalence and Demographics

Estimates of the prevalence of PPD range from 1.21% to 4.4%. In an epidemiological survey of the Australian population, 10,641 respondents were assessed by telephone interview. 6.5% of the adult population was diagnosed with a personality disorder, with PPD making up 1.2% of the population [22]. A study from Norway found a 2.4% prevalence [23]. In a sample of 43,093 adults in the United States, the Epidemiologic Survey on Alcohol and Related Conditions found that 14.8% Americans, or 30.8 million individuals, had personality disorder. PPD was the second most prevalent personality disorder (4.4%), after Obsessive Compulsive Personality Disorder (OCPD) [12]. Even accounting for the possibility of methodological issues that might reduce the accuracy of these estimates, PPD is more common than expected given the scant attention it receives in the clinic or laboratory.

Prevalence in psychiatric clinics ranges from 2 – 10% and 10 – 30% in psychiatric inpatient hospitals [3]. Although some work has suggested a lower prevalence in hospital settings [24] [14], the high prevalence in prison populations, 23%, supports the validity of higher prevalence rates (Ullrich et al., 2008).

Regarding gender differences, epidemiological research finds higher rates in women [12] while clinical samples find higher rates in men [3]. Demographic risk factors include low income (OR = 3.55) and being Black, (OR = 2.15), Native American (OR = 3.12) or Hispanic (OR = 1.43). Additional risk factors include relationship history, with PPD being associated with having been widowed, divorced, or separated (OR = 1.94) or never married (OR = 2.03) [12]. In clinical settings, Africa-Americans are more likely to be diagnosed with

PPD [25]. These differences are probably best explained by the effects of trauma and stress [26]. Overall, the demographic patterns found in PPD paint a picture of a disorder that is found in the disadvantaged and subdominant. These raise the hypothesis of social stress as an etiological factor, which the data, as reviewed in sections following, generally confirm. Indeed, perceived racism is correlated with levels of non-psychotic paranoia in African Americans [27].

Very little data is available regarding the role of culture in PPD. Paranoid, mistrustful thinking can be found across cultures and lifespans. Available data suggest that levels of mistrust are similar between children living in England and Hong Kong [28]. On the other hand, there is some evidence suggests that cultural behaviors can modify the pattern of symptom presentation in culture specific ways (Nakamura et al., 2002). Further work is needed in this area.

History

Perhaps the first systematic description of PPD comes from Kraepelin, who posits that PPD is a diminished form of dementia precox. To him, *Paranoid personality* is characterized by mistrust, interest in secret motives, irritability and discontent, fault-finding, feelings of being treated unjustly and of being oppressed, and excessive evaluation of the self [29]. Although current evidence does not support the concept of PPD as a premorbid syndrome of schizophrenia, Kraepelin's description of the disorder is essentially identical to current descriptions of PPD. Important contributions also came from Bleuler, who pointed out that *paranoid personality* is not associated with fully fledged delusions [30], an idea that is predictive of the currently supported link between PPD and delusional disorder. Meyer detected a specific cognitive rigidity in PPD [31] that presages contemporary thought about it. In 1923, Schneider linked PPD to psychopathic behaviors, further dividing psychopathy into a combative versus eccentric subtype [32].

PPD has been present in the DSM since 1952 [33]. In the twentieth century, scholarship regarding PPD came in two bodies of work, a theoretical but clinically grounded psychodynamic formulation of PPD, and an empirical psychology of paranoid anxiety.

Reliability and Measurement

The reliability of the PPD diagnosis has been addressed in series of studies, each of which has been fairly small. Reliability is measured with Cohen's Kappa, which is a ratio, from 0 to 1, of the relationship between the observed rate of agreement of diagnosis to the rate expected by chance. A Kappa of 1 is a rating with perfect agreement. Kappa and IC values < 0.40 are considered poor, between 0.41 and 0.75 are fair, and above 0.75 are excellent [34]. As displayed in Table 1 below, based on the evidence availability, DSM defined PPD has low to fair inter-rater reliability and low test-retest reliability. We found six studies that reported Kappa values; 1: the Collaborative Longitudinal Personality Disorders Study, finding lower PPD reliability than borderline personality disorder (BPD) [35]; 2: a study in military recruits [36]; 3: a study using the International Personality Disorder Exam, also finding PPD to have lower reliability than BPD [13]; 4. An early study using the DSM-III

criteria setting the floor on reliability (kappa = 0.35) [37]; 5: a study using the SIDP III [38] and a study using the Dutch version of the SCID-II [39].

Given the very small sample sizes used to calculate Kappa scores for PPD, it is difficult to come to firm conclusions about the reliability of diagnosis. However, the data do not make a strong case for it. Potentially making matters worse, clinicians are less likely to detect PPD than semi-structured research interviews [40]. This finding would suggest that the inter-rater reliability in the clinic is most likely even lower than that found in the research setting. Thus, the categorical description of PPD would need substantial revision to maximize its usefulness in the clinic. Interestingly, evidence from the same studies found higher reliability for PPD related dimensional trait behaviors. As reviewed in the following sections, this comes at a time when the empirical psychological and biological sciences also support a dimensional behavioral trait model.

Dimensions

Given the relatively low inter-rater reliability of categorically defined PPD compared to its dimensional equivalent, the ability of dimensional systems to capture the essence of PPD is an important issue. A study examining the taxonometric structure of PPD in 731 patients from the Collaborative Longitudinal Study of Personality Disorders found evidence that PPD is dimensional rather than categorical ([41]. The lack of clear demarcation between cases and non-cases detected in taxonometric analysis could help explain the low inter-rater reliability found using clinical diagnostic criteria. On the other hand, a Korean study examining the frequency distribution of personality disorder criteria found that PPD exhibits a cubic distribution [42]. These results suggest that PPD is intermediate between disorders that are categorical, such as Antisocial and Schizoid, and those that are more clearly dimensional, such as Obsessive-Compulsive.

Consensus on the best dimensional representation of PPD is lacking. Evidence to date can be broadly categorized as research dividing PPD into lower order behavioral traits, versus research examining paranoia itself as a dimension. We will first review studies examining lower order behavioral traits.

In a Norwegian study of 930 personality disordered patients, 114 patients with DSM-IV PPD were identified [43]. Supporting the dimensional approach, there was no distinct boundary between cases and non-cases, as evidenced by a large of number of individuals satisfying some diagnostic criteria but remaining sub-threshold. Confirmatory factor analysis found evidence for two underlying traits, suspiciousness and hostility, moderately correlated with each other (r = .69). The authors propose that suspiciousness and hostility represent independent, latent factors underlying PPD. Doubts about the loyalty of friends was the most highly endorsed symptom, while doubts about the fidelity of a loved one was the least endorsed. These results are supported by previous work examining PPD in the context of the Five Factor Model of personality traits, finding that PPD is negatively correlated with agreeableness and positively correlated with neuroticism [44], possibly along with low extraversion [46]. While understanding PPD as a disorder of low agreeableness and high neuroticism is helpful, this set of traits is not unique to PPD. In fact, meta-analysis reveals

that high neuroticism and low agreeableness emerge as traits underlying personality disorder in general [45]. Thus, PPD could be seen as being closely related to Avoidant, Borderline, Obsessive-Compulsive, Antisocial, and Narcissistic Personality Disorder [3]. This would suggest that the Five Factor Model may not be sufficient to characterize clinically important aspects of personality disorder psychopathology relevant to PPD.

A body of work has examined paranoia as a dimension in non-psychiatric populations. Work in community samples finds that paranoid thoughts are common, with 12.6% of young New Zealanders endorsing paranoid features [47] and 1/3 of the UK population endorsing suspicious thoughts [48]. In a study from 7,281 UK individuals from the Adult Psychiatric Morbidity Survey (APMS, 2007), 18.6 reported mild, non-bizarre paranoia. A smaller percentage (1.8%) endorsed feeling that there is a plot to cause them serious harm, representing more severe paranoia of delusional intensity [49]. Another study using a UK sample, this time the British National Psychiatric Morbidity Survey, found evidence for an exponential distribution of paranoid symptoms along a continuum, with a few endorsing more severe symptoms (roughly 2%) and many endorsing mild symptoms (20–30%) [50]. This study was notable for using individual items from the Psychosis Screening Questionnaire (PSQ) and paranoia-related personality disorder criteria from the SCID. The rationale for this approach is that paranoia may exist on a continuum, from interpersonal sensitivity, to mistrust, to ideas of reference, to fixed delusions. The results supported individual clusters, or subtypes of paranoia. The results also supported a continuum model of increasing severity, in which the most severely paranoid individuals, who endorsed paranoid delusions, endorsed all of the affective and interpersonal symptoms of the less severe groups. Such an interpretation would remarkably be consistent with Kraepelin’s view of paranoia as emerging from abnormal personality, rather than expression of schizophrenia (Kendler, 1988).

The field would benefit from an instrument that more specifically characterizes paranoid ideation as found in PPD. Two such measures have been developed, although neither has been widely used. The Paranoid Personality Disorder Features Questionnaire (PPDFQ) measures six traits associated with PPD: suspiciousness, antagonism, introversion, hypersensitivity, hypervigilance, and rigidity [3]. Compared to the DSM-5 criteria, the PPDFQ provides equal emphasis to cognitive, social, and emotional features of the disorder. It additionally measures impairment associated with each trait. Unfortunately, the measure has not been utilized in any research studies beyond the initial work describing the instrument. The Ambiguous Intentions Hostility Questionnaire (AIHQ) was developed as a measure of the tendency to perceive hostile intentions in ambiguous situations. The AIHQ, when tested in a sample of college students, was unrelated to psychosis proneness, and demonstrated good interrater reliability [52]. Although intended for use in non-psychotic populations, it has been utilized to study hostile attribution in psychotic populations [53]. The AIHQ has seen wider dissemination than the PPDFQ. In a predominantly non-psychotic sample of online Korean job-seekers, hostile attribution, as measured by the AIHQ, is related to deficiencies in theory of mind [54].

Longitudinal Course and Comorbidity

Very little is known about the longitudinal course of PPD. We know that PPD traits decline by 46% from adolescence to early adulthood (Johnson et al., 2000). This conforms to the general pattern seen in personality disorder [56]. A small study following a clinical sample found that PPD showed short term stability relative to schizoid personality disorder. PPD did not show the same kind of clinical deterioration as was found with a comparison group of schizoid PD [24].

An estimated 75% of PPD cases have a comorbid personality disorder [57] [58]. Avoidant and BPD are the most frequently comorbid (48% and 48%), along with Narcissistic PD (35.9%) [59]. In forensic settings, the combination of PPD + Antisocial Personality Disorder was the second most common cluster, after Antisocial + Narcissistic Personality Disorder [60]. Substance abuse problems [61] and panic disorder are also frequent comorbidities [62].

Risk Factors

Childhood trauma has consistently been identified as a risk factor for PPD, in at least 4 cross-sectional studies and one longitudinal study. The longitudinal study found that childhood emotional neglect, physical neglect, and supervision neglect predicted PPD symptom levels in adolescence and early adulthood [63]. In adolescence, PPD has been cross-sectionally associated with elevated physical abuse in childhood and adolescence, but not sexual abuse [64]. In this study, patients with PPD were also more likely to have PTSD. In a study of psychiatric adult outpatients, PPD was found to associated with both sexual and physical abuse [65]. These relationships were found with other personality disorders as well, and were not specific to PPD. Childhood abuse was also related to PPD symptom level, suggesting a dose-response relationship, even when PPD symptoms were subthreshold for the diagnosis [66] [67]. Although these studies have focused on chronic trauma from caregivers, acute physical trauma in the form of childhood burn injury has also found to be a risk factor for adult PPD traits [68].

Brain trauma has been hypothesized to be a risk factor for paranoia [69]. Empirical, cross-sectional research finds that between 8.3 – 26% of brain injury patients meet PPD criteria [70] [71]. PPD was the second most common PD following TBI [71]. Longitudinal studies in this area are lacking, but are needed to establish the temporal sequence of the association. Another important question regarding the associating with brain injury and PPD is if the relationship is due to neural circuit dysfunction, or if a change in function as result of the injury alters social interactions. As an example of this, persons who are hard of hearing are more likely to develop paranoia, likely through increased difficulty with and stress from communication with others [72].

Theoretical Models of PPD

Psychodynamic

Based on the case of Schreber, who exhibited paranoid delusions, Freud theorized that paranoia is an externalizing defense against unconscious homosexual wishes [73]. There has

been little theoretical or empirical support for anxiety about homosexual urges as a cause of paranoia, but theoretical work has preserved the idea of paranoia representing an outward projection of inward conflict. These have varied from shame [74] to an intolerance to indifference [75]. Otto Kernberg classified PPD as a subtype of borderline character pathology, a “lower order” level of character organization characterized by minimal super-ego integration, excessive aggressive drives [76], and a tendency towards primitive mental processes such as splitting [77]. It is interesting that Kernberg saw both BPD and PPD as sharing characteristics with psychotic patients, but generally capable of reality testing in a way that psychotic patients are not capable of. The question of PPD’s relationship with psychosis was a theme that was later to preoccupy empirical work regarding the heritability and family history of PPD. Psychodynamic theory also anticipated interest in the role of trauma as a risk factor. The psychological process of splitting is theorized to result from disturbed attachments [78], with the cognitive style of the individual determining how such early life attachment trauma would lead to specific personality disorder symptoms [79].

Empirical work has provided mixed support for psychodynamic theories of PPD and paranoia. A systematized, self-psychological model emerged in the 1990s that posited that paranoid delusions arise when an individual is unable to tolerate the discrepancy between an implicit, negative view of the self and a conflicting, idealized positive self-concept. In such individuals, blame must be externalized to another person in the form of paranoid delusions [80]. However, later work failed to replicate support for the importance of the discrepancy between implicit and explicit experiences of the self [81]. Instead, it has supported a simple, direct relationship between paranoia and low self-esteem [82] and shame [83].

Cognitive

Cognitive theories of PPD have tended to emphasize dysfunctional beliefs about the self, cognitive style, and social cognition. Aaron Beck has theorized that individuals with PPD hold dysfunctional beliefs of themselves as lacking efficacy while others are malicious and deceptive: this leads to fears about vulnerability, a tendency towards guardedness, and discomfort with emotional closeness [84]. The role of projection and emotion is thus deemphasized, although there are some parallels with psychodynamic theories in its postulating that a concept of self-deficiency is at the core of PPD. There has been some limited empirical support of Beck’s theory of PPD. In a study of college students, negative beliefs about self and others predicted paranoia [85]. In clinical populations, hypersensitivity to criticism, a form of psychological vulnerability, is associated with paranoia [86].

A tendency towards a reasoning bias that jumps to conclusions has been a consistent and robustly replicated finding of empirical research in paranoia [87] [88]. This work must be interpreted in light of the severe thought disorder encountered in psychotic populations. Research in samples with non-psychotic, PPD individuals has confirmed that the same reasoning bias applies in PPD [89]. Similarly, in community samples on non-psychotic, non-patients, “jumping to conclusions” is predictive of paranoia [90]. Although findings of a reasoning bias in PPD are not surprising, it is not yet known why this reasoning bias occurs, and to what degree it reflects a vulnerability to psychotic disorders. Given the weight of

evidence that PPD does not represent a schizophrenia-spectrum psychiatric disorder, it seems likely that reasoning bias alone is not a sufficient explanation of paranoia.

Social Cognition

The demographics of PPD reviewed previously suggest that social factors are important risk factors. The importance of childhood trauma as a predictor of PPD symptoms indicates that social learning and relationship history may in fact play a causal role in the development of the disorder. Lower social rank is correlated with paranoia [48]. Lower social rank may lead to paranoia due to a change in how the individual experiences social interactions, termed "dysphoric self-consciousness" (Kramer et al., 1998). A study in graduate business school students found that people with short tenure (1st and 2nd year students), compared to those with seniority, are more likely to personalize antagonistic experiences. This state can be described as *hypervigilant* [91]. In order to probe the direction of causality, an intriguing study using virtual reality found that lowering the apparent height of an individual in a simulated social interaction increased paranoid, suspicious interpretations of interactions [92]. The results of this experimental study confirm a causal role of self-consciousness in social interactions in the generation of paranoia. It is also possible that deficits in social cognition may promote suspicion. Lower perspective taking ability in a role playing task has been found to predict the development of Cluster A personality disorders and delusional disorder [93]. Poor theory of mind skills are related to traits of hostility [54]. In an experimental study, the presence of theory of mind deficits was predictive of paranoid attribution [94]. In total, the role of social context and innate "social skills" in the form of cognitive empathy appears to play in important role in the formation of paranoid thoughts. As of yet, there has been no empirical research examining social cognition in PPD.

Computational and Biological Models of Paranoia

One of the first computerized "chatbots" was programmed by a psychiatrist to simulate a paranoid psychological process. PARRY was programmed to interact by text with a human in conversation [95]. PARRY was prone to experiencing shame in the form of thoughts of himself as stupid or crazy, triggered easily by social interaction. As negative affect rises, PARRY searches for whom to blame. The negative affect has a decay function: if the conversation continues without triggering shame, negative affect returns to baseline after a time, and PARRY's paranoia remains at bay. PARRY is also capable of instigating projective identification, as his increasingly hostile responses have the potential to elicit negative responses from the human interacting with him. This positive feedback loop between PARRY and a human user can lead to escalating paranoia. One fascinating implication of PARRY provides an important insight for psychotherapists engaging with PPD individuals: if they can delay triggering shame until the completion of the decay function of negative affect, they can shape the behavior of the paranoid personality.

An interesting cognitive and computational model has been described based on the finding that antipsychotic drugs, which block the D2 receptor, suppress the conditioned avoidance response [96]. In the conditioned avoidance response, the subject learns to eventually avoid an unconditioned, noxious heralded by a conditioned, neutral stimulus, by escaping it. In the typical experiment, avoidance and escape are afforded by two chambers, one of which is the

avoidance and escape chamber. In the CAR model of paranoia, paranoid thoughts are created by the psychological escape behavior of externalizing blame, and maintained by avoidance behaviors such as isolation [97]. One of the key insights afforded by the model is that conditioned avoidance is extremely resistant to extinction, a property that perfectly characterizes on of the most vexing aspects of paranoia. Again, this model provides an important clue for psychotherapy and rehabilitation regarding the role of social isolation in perpetuating paranoid ideation.

Neurobiology

The biological literature on PPD is sparse, but some interesting clues have emerged regarding a biological mechanism. In an experiment involving brain sensory processing as measured by EEG event related potentials (ERPs) to auditory stimuli, PPD was found to have a faster latency of the N100 Event Related Potential (ERP) to auditory stimuli, suggesting hypervigilance [98]. PPD cases had normal mismatch negativity (MMN) in this study. MMN is the increase in the amplitude of the N100 ERP to a second tone that does not match the preceding tone, and is reduced in schizophrenia. The findings from this study suggest that PPD has important neurophysiological differences from schizophrenia, and may be characterized by hypervigilance to the environment.

A study of cerebrospinal fluid levels of stress neuropeptide corticotropin-releasing hormone (CRH) in a sample of personality disordered and normal adults found that CRH levels were inversely related to childhood history of parental care [99]. In this sample, PPD, but not BPD, was associated with elevated CRH concentration (39.8 pg/ml vs 27.1 pg/ml; see Figure 1). Although CRH is best known for its role in the stress response and anxiety, in primates direct brain injection of exogenous CRH caused radically altered emotional expression and social behavior behavior in rhesus monkeys. Administration of CRH induced “wall facing” behavior, in which the normally social monkeys appeared withdrawn and non-interactive [100]. One must wonder if the wall facing behavior observed after CRH administration is not a form of paranoid social anxiety like that found in PPD.

Social stress, as reviewed previously, appears to be a risk factor for paranoia. Given convincing evidence of the role of the dopamine D2 receptor type in the pathophysiology of schizophrenia [101], dopamine may also play a role in the pathophysiology of PPD. In animal models, social defeat stress increases dopamine release as measured by microdialysis of the nucleus accumbens (NAC) and prefrontal cortex [102], suggesting that the paranoia of PPD may be driven by central dopamine elevations. Although there have been no biological investigations of dopamine signaling in PPD, research has examined the familial relationship between PPD and psychotic disorders.

The Cluster A personality disorders have long been hypothesized to be related to schizophrenia. Family studies have presented mixed evidence, with 3 of 4 blinded family association studies reporting increased familial risk for PPD in schizophrenic probands (reviewed in Webb & Levinson, 1993). However, the data are sparse and the strength of association between PPD and schizophrenia is weaker than that between Schizotypal Personality Disorder and schizophrenia [104]. In one blind family study, PPD was in fact

more common in relatives of unipolar depressives than schizophrenics [105]. The genetic relationship between PPD and delusional disorder has more supportive evidence. 30% of family members of delusional disorder have paranoid personality disorder traits, compared to 3% of family members of controls [106]. In contrast , schizoid and schizotypal personality disorder are more common in families of schizophrenics and less common in families of delusional disorder patients [24] [107]. Offspring of parents with schizophrenia have been found to be at higher risk for avoidant and schizotypal personality disorder, but not PPD [108]. These results were echoed in an adoption study, which showed that adopted away offspring of mothers with schizophrenia had higher rates of schizotypal but not PPD. Furthermore, in adopted away offspring of mothers with schizophrenia spectrum disorders, no signal was found for PPD [109]. In summary, studies examining the genetic relatedness of PPD to schizophrenia have found some evidence of a relationship, but the relationship between schizophrenia and schizotypal personality disorder is stronger. PPD appears instead to have a genetic relationship to affective disorder and delusional disorder. This work would suggest that PPD does not represent a dopaminergic psychosis. Biological research is needed to further test the connection between dopaminergic function and PPD.

Treatment of Paranoid Personality Disorder

Relatively little is known about the treatment of PPD. Partly due to a mistrust of and reluctance to participate in research by persons with PPD [86], the lack of knowledge is also the consequence of PPD's clinical significance being underappreciated. There are no FDA approved medications for PPD, nor for its frequently comorbid condition of BPD. There have been no clinical trials specific to PPD.

Given the frequent comorbidity of PPD and BPD, indirect evidence may be gleaned from treatment trials for BPD. These have measured the effects of psychopharmacological treatment on aggression, which is highly correlated with suspicious and hostile traits. Meta-analysis of clinical trials in BPD find evidence for positive effects on aggression by antipsychotic medications (Standardized Mean Difference (SMD) = −0.31 [110]) antidepressants (SMD = −0.55 [111]), and mood stabilizers (SMD = −1.83 [112]). Given the relatively small size of the trials, the computed effect sizes are not reliable and difficult to compare. Nonetheless, it is interesting to note that antipsychotics as a class do not have a large effect on aggression, and refute the assumption that PPD is treatable with the same tools as the treatment of psychosis.

Little is known about effective psychotherapeutic approaches to PPD. Some cases of PPD seek psychoanalysis. These are usually not identified in the clinical assessment, but nonetheless are accepted for analysis less than 1/3rd of the time. In cases suitable for psychoanalysis, the symptoms are less severe, the case is comorbid with BPD, and the diagnosis is often missed by the clinician [113]. In theory, many of the approaches in transference focused psychotherapy, found to be effective for BPD [114], should work in PPD. However, published trials do not comment on comorbidity with PPD. Cognitive Behavioral Therapy (CBT) of PPD has been advocated for, based on a model of externalized shame, which shares a common language with psychodynamic models (Beck et al., 2004). Systematic data is lacking regarding CBT, although case studies support its potential

effectiveness [115] [116]. Experimental data provide intriguing clues about potential approaches. In a study of social exclusion using the cyber ball game, cognitive reappraisal was surprisingly found to increase, rather than decrease paranoia in paranoia prone individuals [117]. It is tempting to explain this finding based on resistance to extinction of the Conditioned Avoidant Response (CAR); the paranoid patient may be negatively motivated to reappraise their feelings and beliefs. The validating pose of Dialectical Behavioral Therapy provides a potential solution to this problem. Because the paranoid anxiety of PPD is neither delusional nor bizarre, clinicians may be able to see some truth in the suspicious of PPD. Pointing this out could help to exit the interaction from a positive feedback loop of suspicious hostility by diffusing tension and mistrust. Computational models suggest slowing down the pace of therapy may be advantageous, to “wait out” labile emotional reactions [118]. This would suggest that clinicians should titrate the intensity of psychotherapy sessions by the emotional and physiological state of the client.

Mentalization based treatment (MBT) is a validated approach to BPD that combines approaches from psychodynamic therapy, CBT, and interpersonal psychotherapy [119]. MBT emphasizes building the capacity to mentalize, a psychological skill related to cognitive empathy and Theory of Mind. Although no MBT trials have specifically targeted PPD, when PPD was comorbid with BPD, PPD did not appear to predict treatment nonresponse (Bateman, personal communication).

Although these psychotherapeutic approaches have promise, there is reason for caution. Data from a large number of patients in intensive psychotherapeutic day treatment programs reveal that PPD is an important predictor of treatment failure and dropout (Karterud et al., 2003) [120]. This appeared to be true whether or not PPD occurred by itself, or comorbid with BPD [120].

Descriptive Data

We present descriptive data from the research program of the Clinical Neuroscience and Psychopharmacology Research Unit at The University of Chicago, Department of Psychiatry. Over the last seventeen years, a total of 115 adults with PPD have been studied, along with 208 individuals with BPD and 417 normal controls. 71 of the PPD cases have comorbid BPD, making BPD an interesting and important comparison group. All subjects were recruited either through clinical referrals or media advertisements seeking volunteers for research regarding problems with anger, mood, suicide, and aggression. All subjects provided written, informed consent with consent forms approved by the IRB of The University of Chicago.

Demographic data are displayed in Table 2, broken down into four groups: PPD+BPD, PPD Alone, BPD Alone, and Normal Control. A higher percentage of PPD patients (61%) are African American compared to 20% of the normal control group. These race differences confirm previous findings of higher rates of African Americans in PPD [121] [122] and are likely due to differential exposure to stress and trauma [26].

The clinical data in Table 3 reveals that individuals in the PPD-only group meet, on average more than 2 BPD criteria. On the other hand, BPD individuals meet on average only 1.5 PPD criteria. Data were analyzed using ANCOVA, covarying for age and gender; all results are two-tailed. Although both PPD and BPD have a higher rate of suicide attempt and self-injurious behavior relative to normal controls, BPD has a significantly higher rate of suicide and self-injurious behavior than PPD. Interestingly, the comorbid PPD+BPD group has a higher rate of suicide attempt and self-injurious behavior than the PPD only group but the comorbid PPD+BPD group did not have a higher rate of suicide attempt and self-injurious behavior than the BPD group. This suggests that having comorbid PPD does not increase the risk of suicide or self-injury in individuals with BPD, while having comorbid BPD does increase the risk of suicide in individuals with PPD. This is the first data that we are aware of addressing the risk of suicide and self-injury in PPD.

Replicating the association of PPD with childhood trauma, PPD is associated with higher levels of emotional abuse, emotional neglect, physical abuse, physical neglect, and sexual abuse relative to normal control subjects, as measured by the Childhood Trauma Questionnaire (CTQ) [123] (Table 4). There were no significant differences between PPD +BPD, PPD-only, and BPD groups for any of the CTQ subscale scores. The results confirm previous reports of strong relationships between PPD and childhood trauma. Interestingly, PPD, unlike BPD, was *not* correlated with the CTQ Lie scale, a measure of positive response bias. This would suggest that retrospective reports of childhood trauma by PPD individuals are not contaminated by response bias.

Relationships between PPD and impulsivity and aggression are depicted in Table 5. Overall, both BPD and PPD are characterized by higher levels of impulsivity and aggression than normal controls. However, BPD is more impulsive and more self-injurious than PPD, the latter finding mirroring the higher rate of suicide attempt in BPD. However, PPD is significantly more aggressive than BPD. Effects of comorbidity are also seen. PPD comorbidity with BPD increases aggression relative to BPD alone. BPD comorbidity with PPD increases impulsivity and self-aggression. These results highlight the importance of recognizing PPD when it is comorbid with other more widely acknowledged personality disorders.

A subset of subjects completed a multi-dimensional questionnaire assessment of cognitive and emotional empathy, the Interpersonal Reactivity Index (IRI; [124]) (Table 6). So, for reasons of statistical power, two separate ANOVAs were computed to control PPD and BPD to normal controls. PPD and BPD shared a pattern of diminished cognitive empathy (decreased Perspective Taking), and some aspects of enhanced emotional empathy (increased Personal Distress). These data replicate previous work finding decreased cognitive empathy and intact or increased emotional empathy in BPD [125], and suggest that PPD shares a similar profile with respect to empathy. To our knowledge, this is the first characterization of social cognition in PPD.

In summary, descriptive data from this sample of PPD and BPD cases confirm that PPD, like BPD, is associated with childhood trauma. Aggression is encountered in both disorders, but is more outwardly directed in PPD and more inwardly directed in BPD. BPD is more closely

associated with impulsivity, suicide risk, and self-injury. Given the relationship of paranoia with social cognition [93] [94], it is interesting to note that our preliminary data suggest that PPD, like BPD, is deficient in cognitive empathy.

Summary

Since its inception by Kraepelin, who presciently distinguished PPD from dementia praecox, PPD has continued to be a relevant description of a group of humans with a severe, debilitating mental disorder. For a disorder that has attracted scant attention, there has been surprisingly coherence between the theoretical and empirical science regarding it. Social and developmental factors point to stress, trauma and neglect as likely being causative. Although among the personality disorders, BPD is the most automatically associated with childhood trauma, individuals with PPD are likely to endorse neglect, physical abuse, and sexual abuse in their past. A psychological theory of paranoia and PPD has been built around the observation that PPD individuals are characterized by negative emotionality, hypervigilance, cognitive rigidity, and an aggressive, hostile disposition. The dominant theme in psychodynamic and contemporary psychological approach is externalized hostility, triggered by a vulnerable, fragile sense of self in the context of stressful social interactions. Biological data are scarce, but reinforce the phenotypic characteristics of hypervigilance and stress reactivity. Knowledge about treatment approaches remains general, but available data paint a picture of a disorder that is often comorbid with BPD and perhaps even more challenging to treat. Descriptive data are presented which reinforce this portrait of PPD as related to trauma, social adversity, risk of aggressive behavior, and impaired social cognition.

There are several critical questions for future research. The question of the dimensional versus syndromic nature of PPD is not yet fully answered. Available data on the whole support the dimensional approach, but assessment approaches need to be validated and standardized to be useful in the clinic. The position of PPD relative delusional disorders and schizophrenia has been clarified, but the boundary between non-psychotic paranoia and paranoid delusions must be more clearly defined by empirical research. Although biological data regarding the mechanism of PPD are scarce, what is known so far supports the potentially enormous value of the NIMH rDOC approach, which organizes PPD symptoms under the negatively valenced emotion and social processes categories. Anchoring the clinical and psychological approach to PPD in brain-based systems of negative affect and social processes could substantially accelerate the progress of research. There is an enormous body of neuroscience regarding the neural circuits mediating normal emotional and social behavior that can be applied to PPD. We think it is likely that these milestones must be reached to enable achieving the ultimate goal of treating, or even curing, PPD.

While acknowledging the current limitations of the science of PPD, it would be a mistake to discount the value of expertise regarding PPD in the clinic. In our experience, being able to identify PPD in difficult clinical scenarios is needed to perceive and understand the underlying psychopathological process. This has powerful predictive value for treatment planning and avoiding the kind of misunderstandings that can lead to negative outcomes. Most clinicians encounter PPD cases in the clinic, hospital, or forensic setting, and it is probable that these cases are among the most challenging they will encounter.

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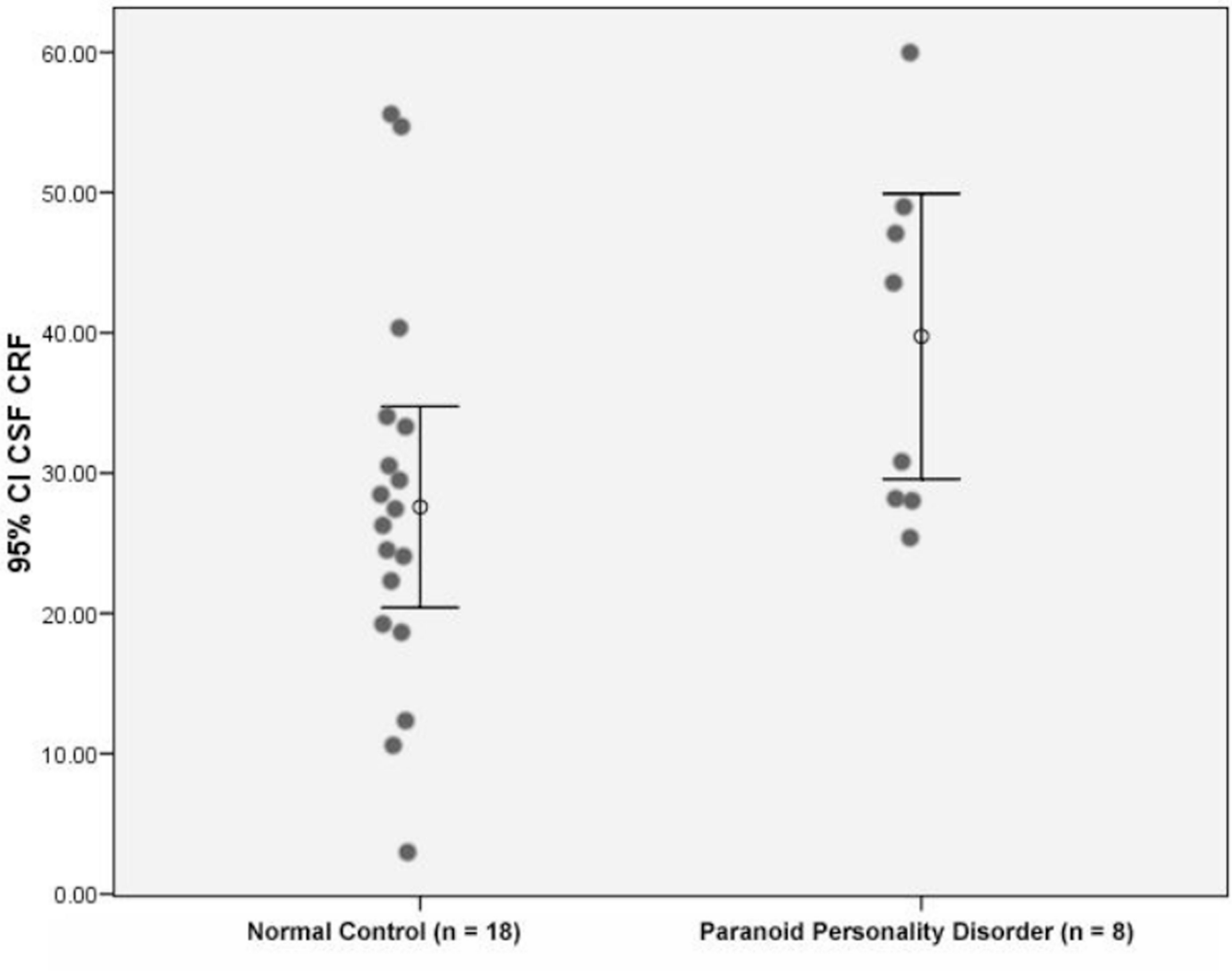

Figure 1. Levels of CSF CRH in PPD

PPD subjects (n = 8) had significantly higher levels of CSF CRH than normal control subjects (n=18).

Table 1

Reliability of PPD

Study	Instrument	N	Interrater	Test-Retes	Trait	Sum
Mellsop et al., 1982	DSM-III	8	0.35			
Zanarini et al., 2000	DIPD-IV	2	0.58	0.39		0.86
Pilkonis et al.,	SIDP-R III	?	0.47			
Loranger et al., 2007			0.47			
Jane et al., 2006	SIDP-IV	12	.57		0.75	0.84
Lobbestael et al., 2011	SCID-II	3			0.85	0.85

Table 2

Demographics

Demographic data summarized across the four subject groups.

	PPD+BPD	Paranoid PD	Borderline PD	Normal
Number	32 (6.8 %)	31 (6.6 %)	92 (19.6%)	313 (66.7%)
Age	37	34	35	34
Gender (female)	52 (73.2%)	20 (45.5%)	137 (66.5%)	207 (49.8%)
Caucasian	16 (50%)	10 (32%)	39 (42%)	204 (65.2%)
African American	11 (34%)	19 (61%)	36 (39.1%)	64 (20.4%)
Hispanic	3 (12.5%)	1 (3.2%)	13 (14.1%)	12 (3.8%)
Asian	-	1 (3.2%)	2 (2.2%)	17 (5%)
Hollingshead	34.9	33.8	38.01	47.12

Chi-Square analysis reveals significant race differences between PPD and NC subjects (χ^2 (5, N = 518) = 38.75, p < .001) but not between PPD and BPD subjects subjects (χ^2 (4, N = 248) = 5.11, p = .28)

Table 3

Clinical Characteristics

Summary of the clinical characteristics of the four subject groups.

	PPD + BPD N = 71	Paranoid PD N = 44	Borderline PD N = 205	Normal N = 417
#BPD Criteria	6.27 (1.32)	2.23 (1.12)	6.17 (1.254)	
#PPD Criteria	4.54 (.753)	4.41 (.948)	1.50 (1.06)	
Suicide Attempt	33 (46.5%)	7 (15.9%)	76 (36.9%)	0 *
Self Injury	21 (29.6%)	1 (2.3%)	63 (30.6%)	0 *
Legal (arrests)	2.91	3.77	2.12	0.15 *
MDD (current)	25 (35.2%)	5 (11.4%)	72 (35%)	0 *
MDD (past)	27 (38%)	14 (31.8%)	82 (39.8%)	0 *
IED	44 (62%)	25 (56.8%)	98 (47.6%)	0 *
PTSD (current)	24 (33.8%)	6 (13.6%)	46 (22.3%)	0 *

* Chi Square tests reveal that BPD is more likely to have a history of suicide attempt than PPD (Chi Square (1, 249) = 7.30, p = .008) and history of self-injurious behavior (Chi Square (1, 249) = 15.36, p < .001). BPD+PPD was more likely than PPD to have suicide attempts (Chi-Square = 11.19, p < .001) and self-injurious behavior (Chi-Square = 13.09, p < .001). There were no significant differences between the BPD+PPD and BPD groups.

Table 4

Childhood Trauma

CTQ subscale scores across the four subject groups: All three subject groups had significantly higher CTQ subscale scores relative to normal controls.

	PPD + BPD N = 32 Mean F-test p-value	Paranoid PD N = 31 Mean F-test p-value	Borderline PD N = 91 Mean F-test p-value	Normal N = 295 Mean F-test p-value
Emotional Abuse	15.53 (4.77)	11.97 (5.34)	14.37 (5.0)	6.79 (2.84)
	F = 14.450	F = 16.588	F = 102.7	
	p < .001	p < .001	p < .001	
Emotional Neglect	14.9 (5.09)	12.74 (5.24)	13.79 (5.28)	8.17 (3.82)
	F = 7.817	F = 10.468	F = 39.489	
	p = .005	p < .001	p < .001	
Physical Abuse	10.78 (4.7)	10.23 (4.88)	11.26 (5.21)	6.2 (2.1)
	F = 21.220	F = 7.593	F = 35.71	
	p < .001	p = .001	p < .001	
Physical Neglect	9.47 (2.98)	8.38 (3.84)	8.71 (3.65)	5.86 (1.78)
	F = 6.399	F = 10.435	F = 3.741	
	p = .012	p < .001	p < .001	
Sexual Abuse	9.16 (6.1)	8.35 (5.67)	9.43 (6.56)	5.28 (1.49)
	F = 12.185	F = 3.017	F = 12.185	
	p = .001	p = .05	p < .001	
Minimization	.06 (.25)	.26 (.58)	.11 (.40)	.67 (1.0)
	F = 2.904	F = 1.872	F = 11.195	
	p = .09	p = .16	p < .001	

No significant differences were found between BPD+PPD and BPD, BPD+BPD and PPD, PPD and BPD.

Table 5

Aggression and Self-Aggression

Impulsivity and Life History of Aggression (LHA) and subscale scores for the four subject groups.

	PPD + BPD N = 57 Mean F-test p-value	Paranoid PD N = 43 Mean F-test p-value	Borderline PD N = 176 Mean F-test p-value	Normal N = 373 Mean F-test p-value
Barratt Total Impulsiveness	76.08 (11.67)	67.07 (10.09)	73.16 (11.14)	55.96 (9.28)
	F = 5.29	F = 11.06	F = 87.64	
	p = .02	p < .001	p < .001	
LHA Aggression	18.65 (4.70)	18.91 (4.74)	16.92 (5.28)	4.81 (3.52)
	F = 83.24	F = 86.81	F = 107.37	
	p < .001	p < .001	p < .001	
LHA Self-Aggression	1.73 (2.13)	.29 (.71)	1.70 (2.44)	.01 (.18)
	F = .204	F = 1.2	F = 67.43	
	p = .652	p = .302	p < .001	
LHA Antisocial Behavior	7.79 (5.08)	8.33 (4.63)	6.33 (4.98)	.62 (1.32)
	F = 34.203	F = 45.63	F = 62.00	
	p < .001	p < .001	p < .001	
LHA Total	28.21 (9.00)	27.5 (8.36)	24.86 (8.77)	5.45 (3.99)
	F = 87.45	F = 98.50	F = 12.185	
	p < .001	p < .001	p < .001	

BPD > PPD for BIS-11 Impulsivity (t (t, 134) = −2.56, p = .01) and Self-Aggression (t (1, 208) = −6.58, p = .02). PPD > BPD for Aggression (t (1, 217) = 2.26, p = .03), Antisocial Behavior (t (1, 65.84)=2.37, p = .02). PPD+BPD > BPD for LHA Total (t (1, 228) = 2.47, p = .01). PPD+BPD > PPD for Impulsivity (t (1, 61) = 3.21, p = .002 and Self-Aggression (t (1, 4. = 4.23, p < .001)).

Table 6

Empathy

Empathy measures from the Interpersonal Reactivity Index (IRI) for the four subject groups. Due to the limited sample size, power was lacking for a single model incorporating PPD and BPD.

	PPD + BPD N = 6 Mean F-test p-value	Paranoid PD N = 11 Mean F-test p-value	Borderline PD N = 26 Mean F-test p-value	Normal N = 81 Mean F-test p-value
Perspective Taking	16.00 (4.69)	16.80 (3.56)	16.9 (4.78)	19.10 (4.83)
		F = 3.84	F = 10.86	
		p = .05 *	p = .001 *	
Empathic Concern	19.67 (4.97)	19.60 (2.30)	19.2 (4.12)	19.3 (4.93)
		F = .08	F = .96	
		p = 77	p = .33	
Personal Distress	13.33 (5.64)	13.60 (3.58)	12.60 (5.22)	8.71 (4.65)
		F = 8.01	F = 10.203	
		p = .01 *	p = .002 *	
Fantasy	14.17 (5.31)	15.40 (5.59)	15.3 (4.76)	14.33 (5.81)
		F = .00	F = .14	
		p = 1.0	p = .71	

* For exploratory purposes, two separate Multivariate ANCOVAs were performed for PPD and BPD (shown in the table). Comparisons between the PPD+BPD, PPD, and BPD groups did not result in any significant differences.