Optimizing Prediction of Psychosocial and Clinical Outcomes with a Transdiagnostic Model of Personality Disorder

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Abstract

Transdiagnostic models hold promise for transforming research and treatment practices for personality disorders (PDs), but widespread acceptance and implementation of such approaches will require persuasive evidence of construct validity and clinical utility. Toward that end, we examined the criterion validity of a hierarchical transdiagnostic PD model in relation to psychosocial and clinical outcomes in a high-risk community sample of 700 young adults. Participants and their mothers completed semi-structured interviews to assess young adults’ PD symptomatology, psychosocial functioning, suicidality, and mental health treatment use. Bifactor modeling revealed an overarching dimension of PD severity—capturing symptoms across all PD categories—that strongly predicted all functional and clinical outcomes in multivariate analyses. Effect sizes for lower-order, specific PD processes were comparatively modest for functional outcomes; however, they provided clinically significant information about suicide risk and treatment use. We discuss implications of a transdiagnostic perspective for research on PD etiology, classification, and treatment.

Keywords

borderline personality disorder; DSM-5; hierarchy; psychosocial functioning; suicide; transdiagnostic

Mounting evidence supports the use of transdiagnostic models to predict, explain, and treat mental illness more efficiently. These models center on the psychopathological processes that transcend traditional disorder boundaries (Barlow, Allen, & Choate, 2004; Krueger & Markon, 2011; Nolen-Hoeksema & Watkins, 2011). Transdiagnostic processes can pertain to an entire set of psychopathologies or characterize a limited range of conditions. Thus, transdiagnostic frameworks can be structured hierarchically (e.g., Mineka, Watson, & Clark, 1998). For instance, we know now that negative affectivity is the substrate of all anxiety and depressive disorders, whereas low positive affectivity represents a more specific, but still
transdiagnostic, diathesis for unipolar depression and social phobia (Watson & Naragon-Gainey, 2014).

A substantial body of research has examined the transdiagnostic personality traits that serve as the scaffolding for the PD domain. One line of research has examined connections between the latent structure undergirding the various PDs maps and Five Factor Model personality trait constructs (e.g., Markon, Krueger, & Watson, 2005; O’Connor, 2005; Samuel & Widiger, 2008). Recent work has also explored the possibility that the various PDs observed in clinical practice represent the expression of (a) a core trait representing general personality pathology, deficits in “personality functioning,” or negative affectivity; and (b) more specific abnormal personality processes (e.g., dependency, mistrust) that determine the exact manifestation, or outward appearance, of PD (e.g., Hopwood et al., 2011; see also Morey et al., 2011).

Such a hierarchical model—involving a broadband propensity to all PD as well as specific personality processes controlling the surface features of PD—represents a potentially useful framework for PD research and treatment. Transdiagnostic dimensions, as compared to discrete PD diagnoses, arguably provide superior targets for etiological and clinical investigations. For instance, treatments that concentrate on transdiagnostic dimensions of PD, rather than the manifest pathology of a given diagnostic category, stand to remediate multiple forms of PD. Likewise, instead of separately designing empirical studies to uncover the origins of various PD diagnoses, the etiology of PD might be more efficiently established by examining factors controlling variation on transdiagnostic PD dimensions.

As compared to the steady stream of research on the latent structure of PD, very little empirical work has addressed the external correlates (i.e., nomological network) of transdiagnostic dimensions emerging from factor analytic studies. The present study addressed that omission in the literature. We began by using bifactor modeling to distinguish a general PD severity dimension from several specific PD dimensions that capture the “style” of PD expression. We then exploited the fact that PD is intertwined closely with psychosocial dysfunction to evaluate the construct validity of the general and specific dimensions of the hierarchical transdiagnostic model.

**Transdiagnostic versus Specific Elements of Personality Pathology in Relation to Psychosocial Dysfunction**

Several research groups have gone beyond categorical PD diagnoses to examine the predictive utility of a general dimension of personality pathology in relation to clinical and/or functional outcomes. In a prospective clinical study, Seivewright, Tyrer, and Johnson (2004) showed that patients’ general PD severity, which was determined by the frequency and diversity of PD comorbidity, reliably predicted social dysfunction 12 years later. Morey and colleagues demonstrated that clinician ratings of global personality functioning—a construct thought to underlie risk for all PDs (Morey et al., 2011)—were associated cross-sectionally with clinical judgments regarding patient functioning and prognosis (Morey, Bender, & Skodol, 2013). These findings prompted the inclusion of a transdiagnostic dimension of personality functioning—measuring disturbances in self and/or interpersonal
capacities (see Bender, Morey, & Skodol, 2011)—as part of the PD diagnostic algorithm in Section III of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association [APA], 2013).

Although the above studies provide some support for the validity of a general dimension of PD severity, they do not consider the possibility of lower-order transdiagnostic dimensions. In the only study to compare the effects of general versus specific PD features on functional outcomes, Hopwood et al. (2011) parsed a general dimension of personality pathology from five “stylistic” elements of PD in the Collaborative Longitudinal Personality Disorders Study (CLPS). They found that greater overall PD severity was strongly associated with social and occupational dysfunction, whereas the empirically derived PD styles (i.e., Peculiarity, Withdrawal, Submissiveness, Instability, and Deliberateness) had relatively weak effects. Along these same lines, Jahng et al. (2011) showed in the National Epidemiological Survey on Alcohol and Related Conditions (NESARC) that PD comorbidity with substance misuse was explained by an overarching factor representing PD severity and a more narrow-bandwidth, specific factor representing cluster B personality pathology. Thus, this latter study suggests that, in some research contexts, it is important to consider the contributions of both general PD severity and also PD manifestation in forecasting clinical outcomes.

### The Contours of Psychosocial Functioning

Psychosocial functioning generally refers to the capacity to navigate the physical and social environment, although many different definitions of functioning have been advanced in the literature (Ro & Clark, 2009; see also Livesley, 1998). The variety of conceptualizations of psychosocial functioning has led to blurry lines of demarcation between personality, psychopathology, and functioning. Indeed, there appears to be substantial overlap between psychosocial functioning and maladaptive personality traits. A recent study found that psychosocial functioning and PD traits showed poor discriminant validity in prospectively predicting daily reports of psychosocial dysfunction in an undergraduate sample (Calabrese & Simms, 2014). Research into the latent structure of psychosocial functioning offers a method for delineating the contours of functioning constructs. Recent quantitative modeling studies have supported a hierarchical configuration of multiple functioning facets (e.g., subjective well-being, social capabilities, basic day-to-day task performance) that can be analyzed separately or combined into a single index of functional impairment (Ro & Clark, 2009, 2013).

Perhaps because of the complexity and variable definitions of functioning, it is rarely assessed as thoroughly as the psychopathology domain. Due to time constraints, larger studies typically employ brief self-report instruments to characterize dysfunction (e.g., Grant et al., 2004), precluding inquiry into the context and severity of reported problems in functioning. Other studies use gold-standard interview measures, but rely on only one informant (typically the proband; e.g., Hopwood et al., 2011), increasing the chances of reporting bias (Oltmanns & Turkheimer, 2009).

In the present study, multiple informants and multiple interview measures—covering social life, family, romantic, and work functioning—were used to characterize psychosocial
functioning. By focusing on these social and occupational domains—and by querying multiple informants—we aimed to minimize overlap with personality trait and personality “functioning” constructs (e.g., Bender et al., 2011; Calabrese & Simms, 2014), thereby limiting criterion contamination in our analyses of PD-functioning associations. Expanding coverage of functioning domains even further, we investigated the effects of PD on suicidality and mental health treatment use. These clinical outcomes are considered facets of psychosocial dysfunction (see Bender et al., 2001; Yen et al., 2003; Zimmerman et al., 2012) but seldom have been examined in community studies of personality pathology.

**Present Study**

Our main objective was to evaluate the construct validity of a two-level transdiagnostic conceptualization of personality pathology by modeling the independent effects of an overarching dimension of pathology and specific PD features. Our models statistically adjusted for the effects of comorbid clinical disorder syndromes (Axis I disorders in previous editions of *DSM*) given previous evidence that they outcompete PDs in predicting psychosocial dysfunction (Grant et al., 2004; but cf. Lenzenweger, Lane, Loranger, and Kessler, 2007). As mentioned above, we applied exploratory bifactor modeling to PD interview responses to differentiate an overarching dimension of PD severity from specific features of personality pathology that were statistically orthogonal to the general dimension. Synthesizing extensive interview data, we constructed a higher-order latent variable model of psychosocial functioning to more precisely determine the nature of connections between PD dimensions and functioning domains. To our knowledge, this was the first study to evaluate a “tripartite” model of general personality pathology, specific PD features, and clinical disorders in a community setting.

Consistent with prior results (Hopwood et al., 2011; Morey et al., 2013), we hypothesized that the general personality pathology dimension would be strongly associated with dysfunction. We predicted that effect sizes would be comparatively weak—although still perhaps theoretically and clinically meaningful (e.g., Jahng et al., 2011)—for the specific dimensions of pathology. We made no directional hypotheses regarding whether PD-functioning associations would withstand adjustment for clinical disorder comorbidity given mixed findings in previous research (Grant et al., 2004; Lenzenweger et al., 2007).

**Methods**

**Participants**

The present sample of 700 was a subset of over 5,000 mother-offspring pairs participating in the Mater-University Study of Pregnancy (MUSP) in Brisbane, Australia (Keeping et al., 1989). The MUSP was designed to evaluate the health and development of children born between 1981 and 1984 at the Mater Misericordiae Mother’s Hospital. Mothers were assessed for depression using the Delusions-Symptoms-States Inventory (DSSI; Bedford & Foulds, 1978) during pregnancy, post-partum, 6 months after birth, and 5 years after birth. The present study selected and followed up 815 of the original families at offspring age 15. This subsample was overselected for mothers with elevated peripartum responses on the DSSI in an attempt to capture a sample of women with varying histories of depression.
Diagnoses of maternal depression were subsequently confirmed via semistructured interview at offspring age 15. Specifically, 354 (43.4%) of the 815 participating mothers were qualified for a diagnosis of major depression or dysthymia by offspring age 15 (Keenan-Miller, Hammen, & Brennan, 2007). Thus, nearly half of the offspring sample was at high risk for psychopathology due to maternal depression. Full details of the sampling procedure are provided in Hammen, Shih, Altman, & Brennan (2003).

The mother-child pairs were recontacted when offspring reached age 20 and invited to participate in a follow-up assessment. Of the 815 offspring in the original sample, 700 (362 females; 85.9% retention) completed personality and psychosocial impairment assessments at age 20. The final sample was 92% Caucasian and 8% minority (Asian, Pacific Islander, and Aboriginal). The median family income fell in the lower middle class and mothers’ median education level was grade 10. Offspring completing the age 20 assessments did not differ from those who participated at age 15 but not 20 in terms of family income (t(782) = −1.49, p = .14), maternal depression history (χ²(1, 815) = 0.18, p = .67), or history of any depressive, anxiety, or externalizing disorder by age 15 (χ² s < 1, ps > .10). Offspring participating at age 20 were more likely to be female (χ²(1, 815) = 11.08, p < .01).

**Procedures**

Interviews to assess diagnostic status were administered to offspring in their homes at age 15. At age 20, offspring completed questionnaire and interview measures to assess personality pathology and two interviews related to psychosocial impairment. Interviewers were advanced graduate students in psychology and were blind to maternal and offspring psychiatric history. All participants gave their written informed consent (or assent), and offspring were compensated AU$15 at the age 15 timepoint and AU$50 at the age 20 timepoint. All procedures were approved by the UCLA Institutional Review Board, Emory University Investigations Committee, and the University of Queensland Ethics Review Committee.

**Measures**

**Clinical Disorder Diagnoses**—Current clinical disorder diagnoses were determined with the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1995), which was administered to offspring at age 20. Offspring were classified as having a depressive diagnosis (unipolar depression and dysthymia), anxiety diagnosis, or externalizing diagnosis (substance use and disruptive behavior disorder). To determine interrater reliabilities for diagnoses, a sample of 55 interviews were selected and reviewed by a second trained clinician blind to the original diagnoses. Weighted kappas for depressive, anxiety, substance use, and disruptive behavior disorders were all above 0.79.

**Personality Pathology**—Offspring PD symptomatology at age 20 was assessed using the Structured Clinical Interview for DSM-IV Axis II Personality Disorders, Version 2.0 (SCID-II, First, Spitzer, Gibbon, Williams, & Benjamin, 1994). A large literature has supported the reliability and validity of the SCID-II, including its convergent validity with other semistructured interview measures (Crawford et al., 2005; Schotte et al., 1998; Skodol, Oldham, Rosnick, Kellman, & Hyler, 1991). In line with prior work and administration
guidelines, participants first completed the self-report version of the SCID-II, and interviewers then queried all positively endorsed symptoms to confirm their presence. Interviewers rated each symptom that participants endorsed on the SCID-II self-report as either absent, subthreshold, or present. The interview had a lifetime timeframe, in the sense that participants reported on how they generally felt, acted, and thought. The SCID-II interview yields both dimensional (i.e., sum of symptom ratings) and categorical scoring of the 10 PD diagnoses. In the present analyses, interviewer-rated symptom counts were the primary explanatory factors, although categorical PDs were still tabulated for descriptive purposes. Participants qualifying for PD-NOS, meaning that the participant fell one criterion short of a PD diagnosis in at least two of the 10 DSM-IV diagnostic categories, were included in the PD category along with the 10 DSM-IV PD categories. Cronbach's alpha estimates for the 10 PD scales were generally good (median: 0.71; range = 0.52 to 0.89). The histrionic (alpha = .52) and schizoid (alpha = .60) PD dimensions demonstrated the lowest reliability estimates, likely due to the relatively small number of items used to assess each (six and seven items, respectively). The kappa coefficients indexing the interrater reliability for each symptom across a randomly-selected sample of 34 respondents ranged from 0.76 to 1.0 (median = 0.96).

**Psychosocial Functioning**—Impairment in young adults’ psychosocial functioning in the past six months was assessed by two instruments that were administered to both mothers and offspring. The Longitudinal Interval Follow-Up Examination (LIFE; Keller et al., 1987) is a semistructured interview used extensively in prior research on psychosocial functioning. The reliability and predictive validity of the LIFE functioning scales have been supported in many previous studies (e.g., Hopwood et al., 2009). The LIFE consists of separate dimensions for family of origin, friendship, recreation, work, academic, and marital functioning. For the purposes of this study, marital functioning was rated for spouses but also any participant who reported having a stable (i.e., at least three months of exclusive dating) romantic partner (N = 379), with missing data accommodated by full information maximum likelihood estimation. Thus, the marital domain refers to functioning in stable romantic relationships.

Each dimension is rated by interviewers on a six- to eight-point ordinal scale to represent typical functioning in that domain over the past six months. Detailed descriptions of each scale point are used to anchor interviewer ratings, and standard queries are supplied to probe the quality of functioning in each role. In the present study, interrater reliability estimates for LIFE dimensions were generally good for the offspring interviews (median intraclass correlation [ICC]: 0.76; range = .71 to .88). Interviews with mothers about offspring function were also adequately reliable overall (median ICC: 0.75; range = .40 to .91), although ICCs in the work (0.40) and friendship (0.58) domains were substandard. As explained in more detail below, we took a latent variable modeling approach—which isolated the common, reliable variance across manifest indicators—to defining functioning here, and this type of analysis mitigated concerns about lower reliabilities on several individual scales.

Offspring and mothers also completed an interview version of the Social Adjustment Scale (SAS; Weissman, 1990). The SAS queries role functioning in work, social life, family of
origin, marital, and parental arenas, although parenting was not assessed due to low frequency in the 20-year-old offspring. Again, the marital domain covered functioning in all exclusive romantic relationships of at least three months’ duration. Each of the domains is rated on a seven-point scale on the basis of functioning over the past six months. The SAS is one of the most widely used instruments to assess functioning in research across clinical psychology and psychiatry, and a multitude of studies support its interrater reliability, concurrent validity, and sensitivity to clinical improvement in patient populations (e.g., Bagge et al., 2004; Weissman, Olfson, Gameroff, Feder, & Fuentes, 2001). The median interrater reliability estimates were 0.79 (ICC range = .44 to .91) for offspring and 0.71 (ICC range = 0.47 to 0.91) for mothers. Relatively low reliabilities in the friendship domain pulled down the median ICC for both offspring and mothers; all other domains demonstrated acceptable levels of reliability. Again, our factor analytic method for determining individual differences in functioning allowed us to isolate the systematic variance in all scales, mitigating to some extent concerns about low reliability for several dimensions.

Treatment use and suicidality in the past five years were assessed as part of the LIFE interview with offspring. Regarding treatment, participants were asked to report the highest level of treatment for psychological difficulties they had received over the past five years. Interviewers recorded whether any of the following treatments had been accessed: over-the-counter medication; general practitioner; psychiatric medication; self-help group or telephone counseling; outpatient psychiatric or psychological services; or hospitalization. Regarding suicidality, participants were asked if they had experienced any of the following in the past five years: suicidal thoughts, but no attempt; one non-serious suicide attempt; one serious suicide attempt; or multiple suicide attempts.\(^1\) Prior research using the LIFE has shown these items to be valid predictors of clinical status (e.g., Bender et al., 2001).

**Internalizing and Externalizing Symptoms**—To understand the nature of the general PD severity factor, we examined its correlations with the Anxiety/Depression, Withdrawal, Intrusion (i.e., entitlement, attention-seeking), Aggression, and Delinquency subscales of the Young Adult Self Report (YASR), which was completed at the age 20 assessment point. The YASR is part of the Achenbach System of Empirically Based Assessments (Achenbach, 2009). Achenbach (2009) has demonstrated the reliability and validity of the YASR scales in multiple samples, and in the present study reliability was acceptable for all dimensions (Cronbach’s alpha range = .73 to .91).

**Data Analytic Plan**

We used latent variable modeling to examine psychosocial functioning, taking advantage of the multiple instruments and multiple informants used to assess functioning in this study. We specified a higher-order configuration for the model, such that intermediate Social Life, Family, Marital, and Work factors were facets of an overarching General Functioning factor. In turn, lower-level factors representing mother versus offspring ratings within a particular domain were indicators of the intermediate functioning factors to account for the high

\(^1\)The statistical significance and interpretation of results were unchanged if the four-level ordinal treatment use and suicidality variables were dichotomized (i.e., no treatment versus any treatment; no suicidality versus any suicidality) and binary logistic regressions were performed.
correlations among ratings from the same respondent (see Figure 1). With this higher-order framework, we were able to examine generalized functioning deficits as well as specialized areas of impairment. The higher-order framework was preferred to bifactor modeling due to minimal prior theoretical and empirical support for the latter approach. Indeed, previous latent variable modeling work has found relatively little support for distinct “styles” of social functioning that are separable from general social impairment (Ro & Clark, 2009, 2013).

Both treatment use and suicidality were considered ordinal categorical variables and regressed on the diagnostic constructs in ordinal logistic regressions. We coded our treatment use variable so that the outcome had four levels: none; over-the-counter medication, general practitioner visit, self-help program, or telephone counseling; outpatient psychiatric or psychological services; and hospitalization. The suicidality outcome also had four levels: none; suicidal ideation; one suicide attempt; and multiple suicide attempts.

We submitted the 10 PD dimensions to a bifactor exploratory factor analysis, a form of exploratory structural equation modeling, with Geomin rotation to separate an overarching dimension of PD severity from a parsimonious set of lower-order, specific PD dimensions (see Jennrich & Benter, 2011, 2012). A bifactor approach was selected to maximize consistency with the one prior latent variable modeling study to examine external correlates of general versus specific PD processes (Jahng et al., 2011). Exploratory bifactor analysis estimates indicator loadings on one general factor and various specific factors that are orthogonal to the general factor, meaning we could determine whether the specific dimensions had an effect on functioning outcomes for people with equal levels of overall personality pathology. This modeling feature aligned well with our aim of uncovering specific factors that were uncorrelated with the general severity of PD (see Hopwood et al., 2011). In the present analyses, intercorrelations among specific factors were freely estimated to allow for the possibility that these abnormal personality processes are partially overlapping. We began by estimating a bifactor model with one specific factor, and we then estimated models with increasing numbers of specific factors until the solution was not substantially improved—as judged by traditional model fit indices and the interpretability of parameter estimates—by additional specific factors. Factor scores from the final solution were used in subsequent predictive analyses.2

In the main analyses, each functional and clinical outcome was regressed on the general PD dimension, the specific PD dimensions, and clinical disorder diagnoses simultaneously to determine their relative contributions. In the psychosocial functioning analyses, effects of PD on the lower-order factors (i.e., Social Life, Family, Marital, Work) were estimated in a model that omitted the superordinate General Functioning factor so that the lower-order factors would not be residualized on General Functioning. Effect sizes were computed for all analyses to permit comparisons of the strength of associations across explanatory factors.

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2 A model combining the hierarchical functioning model and PD bifactor model (i.e., functioning factors regressed on PD factors) failed to converge. Thus, factor scores were saved from the PD bifactor model and were used as predictors in subsequent criterion-related validity analyses. Analyses involving psychosocial functioning were carried out in a latent variable modeling framework (i.e., functioning factors, and not factor scores, were regressed on PD factor scores), and those involving treatment use and suicidality were not carried out in a latent variable modeling framework (i.e., observed single-indicator clinical outcomes regressed on PD factor scores).
Analyses were conducted in Mplus 7.0 using a robust maximum likelihood estimator (Muthén & Muthén, 1998–2012).

## Results

### Descriptive Statistics

Table 1 presents rates of categorical PDs and clinical disorders, along with mean levels and dispersion of the dimensional PD ratings. The most prevalent PDs were PD-NOS ($n = 10$), borderline ($n = 16$), paranoid ($n = 16$), antisocial ($n = 18$), obsessive-compulsive ($n = 25$), and avoidant ($n = 26$). There were three or fewer cases of all other PD diagnoses. Table 1 also shows that the clinical disorder diagnoses were more common than PD. In total, 44.6% of the sample qualified for at least one current depressive, anxiety, or externalizing diagnosis, compared to 13.6% for current PD.

Correlations within psychosocial impairment domains across interview measures and raters were generally moderate to high, supporting the common factor analysis approach to the impairment data. The median correlation of respondents’ ratings in the same functioning domain across interview measures was .82. The corresponding median correlation for different respondents in the same functioning domain and with the same interview measure was .46, whereas the median correlation of different respondents on different measures within functioning domains was .43. Complete descriptive statistics for LIFE and SAS interviews are available upon request. The majority of the sample reported no suicidality (75.1%), whereas the remaining quarter endorsed suicidal ideation (18.0%), one suicide attempt (4.6%), or multiple suicide attempts (2.3%). Approximately one in four participants (25.1%) accessed mental health treatment in the five years prior to the age 20 interview. The modal treatment was psychological or psychiatric intervention on an outpatient basis (13.9%); over-the-counter medications, general practitioner visits, self-help programs, and telephone counseling were less common (8.1%); and a small subset of participants reported a psychiatric hospitalization (3.1%).

### Latent Structure of Psychosocial Functioning

The higher-order functioning model provided a good fit to the data ($\chi^2(156) = 481.07$, $p < .001$; CFI = .95; RMSEA = .05; Standardized Root Mean Square Residual [SRMR] = .06). The median factor loading of the intermediate functioning subdomain factors on the overarching General Functioning factor was .82 (range = .60 to .87), indicating that the subdomains are empirically distinct yet related facets of functioning (see Figure 1). All factor loadings in the model were statistically significant at an alpha level of .001.

### Delineating Specific Personality Disorder Dimensions

A bifactor model with one specific factor was did not fit the data well ($\chi^2(26) = 156.26$, $p < .001$; CFI = .92; RMSEA = .09; SRMR = .04) and produced a negative error variance estimate. With two specific factors, fit was improved but not optimal ($\chi^2(18) = 67.03$, $p < .001$; CFI = .97; RMSEA = .06; SRMR = .03). The solution with three specific factors provided superior fit to the data according to all fit indices ($\chi^2(11) = 28.85$, $p < .01$; CFI = .99; RMSEA = .05; SRMR = .02), and it was associated with a highly statistically significant
chi-square difference test in comparison with the two-specific-factor solution ($\Delta \chi^2(7) = 38.18$, $p < .001$). A model with four specific factors failed to converge, and models with additional specific factors either failed to converge or yielded uninterpretable parameter estimates (i.e., Heywood cases). Thus, the bifactor model with one general factor and three specific factors was judged to provide the most reasonable fit to the data. Table 2 presents the factor loadings of the 10 PD dimensions on all four latent dimensions. The overarching general severity dimension was most strongly related to the borderline and paranoid PD dimensions, whereas the schizoid PD dimension was the least saturated with the general factor. The first specific dimension was marked by moderately high loadings by avoidant and dependent PD dimensions, and we refer to this dimension as Submissiveness. Our second specific dimension (Instability vs. Rigidity) was defined on opposite poles by borderline PD and obsessive-compulsive PD. The third specific dimension (Attention Seeking) was most strongly linked with histrionic and narcissistic PD dimensions. The factor correlation between Submissiveness and Attention Seeking was in the small-to-moderate range ($r = −.29$), and there were small interrelations between Submissiveness and Instability vs. Rigidity ($r = .02$) and Instability vs. Rigidity and Attention Seeking ($r = −.09$).

Table 2 presents the correlations of the general and specific factors (factor scores) with clinical symptoms and disorders, as assessed by the YASR and SCID, respectively. The general dimension exhibited the strongest associations with Aggression ($r = .57$) and Anxiety/Depression ($r = .54$), although moderate correlations were observed with the other YASR dimensions as well. Submissiveness was related predominantly to Anxiety/Depression and Withdrawal, whereas Attention Seeking was inversely related to Withdrawal and positively associated with Intrusion. Instability vs. Rigidity was linked with a variety of YASR scales, paralleling its statistically significant associations with all three classes of clinical disorder.

**Independent Effects of Personality Disorder Dimensions and Clinical Disorders on Psychosocial Functioning**

We performed a preliminary analysis to determine the incremental contributions, relative to the PD general dimension, of PD specific dimensions and the three types of clinical disorder to General Functioning deficits. The PD general dimension was strongly related to General Functioning ($\beta = .55$, $SE = .05$, $p < .001$, $R^2 = .30$). When PD specific dimensions were added to the model, their effect size was roughly one-fifth as large ($\Delta R^2 = .06$). Next, including clinical disorders in the model yielded another modest increment in General Functioning variance explained ($\Delta R^2 = .06$).

Table 3 presents the independent effects of general personality severity, specific personality dimensions, and clinical disorder diagnoses on the various functioning domains (i.e., all predictors are included simultaneously in the regressions). Focusing first on General Functioning, it is clear that general PD severity was a better predictor of dysfunction than the specific dimensions or clinical disorders. The Instability vs. Rigidity dimension demonstrated a statistically significant, but relatively small, association with General Functioning, and Submissiveness and Attention Seeking were largely unrelated to the broadband functioning factor.
Whereas the effect of general PD severity was fairly uniform across functioning facets, the specific dimensions were less consistently associated with the lower-order (i.e., Social Life, Family, Marital, and Work) functioning factors. The effect of Instability vs. Rigidity on General Functioning appeared to be driven largely by its highly statistically significant relation with Work functioning; it was not significantly linked to any other facet. Aside from a statistically significant, yet small, association between Submissiveness and Social Life impairment, the specific PD dimensions exhibited no other substantial relations with functioning facets.

**Independent Effects of Personality Disorder Dimensions and Clinical Disorders on Treatment Use and Suicidality**

PD, clinical disorders, and gender all influenced the likelihood of treatment use. The proportional odds ratio of 1.52 for the general severity dimension indicated that for every standard deviation increase in personality pathology severity the odds of seeking any mental health treatment were 52% greater. Likewise, the odds of outpatient or inpatient treatment were 52% greater than no treatment or consulting a general practitioner, self-help program, or telephone counseling (see Pedhazur, 1997, for more detail on the interpretation of proportional odds ratios). The Instability vs. Rigidity dimension was a key predictor (ORs = 1.69), independent of general PD severity and anxiety disorders (OR = 1.79). Finally, holding constant the level of PD and clinical disorder psychopathology, females were roughly twice as likely as males to seek out any mental health treatment over the past five years.

The Instability vs. Rigidity dimension was again influential in predicting rates of suicidality (OR = 2.76), and in this case it offered even better prediction than the general severity dimension (OR = 1.87). Additionally, after adjusting for other PD dimensions, a standard unit increment in Submissiveness increased the odds of suicidality by roughly 26%. Current depressive disorder diagnosis (OR = 2.88) was by far the best indicator of suicidality of all the clinical disorders; anxiety and externalizing disorders did not exert statistically significant effects after accounting for other predictors.3

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3In addition to exploratory bifactor analysis, we also performed a principal components analysis (PCA) to replicate the analytic strategy used by Hopwood et al. (2011) to separate PD “severity” and “style.” Specifically, PD severity was operationalized as the sum of scores on all 10 SCID-II PD dimensions, whereas specific PD dimensions were derived by individually regressing each of the 10 PD dimensions on the overall PD severity score and then submitting the 10 residuals from those regressions to a PCA with Varimax (orthogonal) rotation. Based on the Kaiser-Guttman rule and parallel analysis, we extracted five components (i.e., Withdrawal, Impulsivity, Instability, Submissiveness, and Peculiarity) in the PCA (eigenvalues: 1.75, 1.52, 1.26, 1.25, 1.09) that collectively explained 68.63% of the total variation across the residualized PD dimensions. The number and nature of the components is similar to the set of five extracted in CLPS, and we therefore followed Hopwood et al.’s (2011) naming conventions for the components. Fifteen out of the 35 specific PD dimension validity coefficients (i.e., associations of specific PD dimensions with functional and clinical outcomes) were statistically significant, and the effect sizes were generally in the small-to-moderate range ($\beta$ range = −.12 to .19; odds ratio range = 0.96 to 2.08). These relatively small effect sizes were consistent with results involving specific factors reported in the main text. Thus, the overall interpretation of results was not substantially different across PCA and bifactor analysis methods. We decided to use bifactor analysis for our primary analyses because it (a) is consistent with our theory of latent personality dimensions underlying PD symptom co-occurrence and (b) produces factors that are free from measurement error, enhancing the accuracy of structural associations (regressions) with functional and clinical outcomes. Full results from the PCA are available upon request.
Discussion

We exploited the well-established association between personality pathology and psychosocial dysfunction to evaluate the validity and potential clinical utility of a transdiagnostic model of PD. We parsed a general dimension of PD severity—underlying the full array of PD signs and symptoms—from a parsimonious set of subordinate (i.e., specific) dimensions of pathology and then examined the relative effect sizes of the general versus specific transdiagnostic dimensions in relation to psychosocial and clinical outcomes.

As hypothesized, overall PD severity was closely related to psychosocial dysfunction, mental health treatment use, and suicidality. Its involvement in diverse areas of dysfunction suggests that it operates as a nonspecific risk marker of impairment. These results are consistent with prominent theories of the structure of PD (e.g., Livesley et al., 1994; Parker et al., 2002) and prior data implicating transdiagnostic elements of PD in psychosocial dysfunction, clinical severity, and prognosis (Hopwood et al., 2011; Jahng et al., 2011; Morey et al., 2011, 2013).

Our empirically derived specific dimensions of personality pathology exerted a modest collective effect on the superordinate functioning factor (incremental $R^2 = .06$). More compelling was evidence that Instability vs. Rigidity outperformed the general PD dimension in predicting suicide risk and mental health treatment use. That is, even after adjusting for general PD severity, a standard unit increment in Instability vs. Rigidity predicted a 176% increase in suicide risk and a 69% increase in likelihood of treatment use. Overall, these specific effects indicate that information about the symptomatic expression of personality pathology (e.g., “unstable” versus “submissive” manifestation) might have predictive utility for prevention and/or intervention purposes, even among people with equal amounts of general personality pathology.

It is notable that we did not recover as many specific PD dimensions as did Hopwood et al. (2011) in the CLPS using a similar diagnostic interview. Differences in data analytic methods may partially explain this discrepancy, given that our supplementary principal component analysis (see Footnotes) identified five specific components that resembled (but were not isomorphic with) those reported by Hopwood et al. (2011). Additionally, our sample featured lower rates of odd-eccentric PDs, limiting our ability to detect specific PD dimensions relevant to psychosis, mistrust, and social isolation. Regardless, results from the two studies converge to signal limited incremental validity—at least with respect to psychosocial functioning—of specific PD features. In the CLPS, the mean standardized effect ($\beta$) of specific components on future psychosocial outcomes was .04, and the corresponding mean $\beta$ in cross-sectional analyses was .06. Most observers would consider these very small effect sizes. In contrast, another study to parse general and specific PD dimensions showed that specific PD features—in the form of cluster B traits—outperformed a general factor representing PD severity in predicting NESARC alcohol and substance use disorders (Jahng et al., 2011). Thus, the practical significance of these specific domains of personality pathology for psychosocial functioning is debatable and may vary across research contexts.
The Nature of General Personality Pathology

What is the general severity factor cutting across all PD categories? Our analyses of its external correlates indicate that it reflects both internalizing and externalizing processes, particularly aggression, anxiety, and depression. Hopwood et al.’s (2011) analysis of the CLPS data also revealed moderate-to-large correlations of general PD severity with aggression and trait negative affectivity (both $r_s = .46$). Thus, while these two studies have uncovered important connections between transdiagnostic personality pathology and personality/clinical traits, it appears that PD severity is not reducible to elevated negative affectivity. Various maladaptive interpersonal processes are widely associated with PD and may form part of the core of PD (see Jahng et al., 2011). Indeed, interpersonal dysfunction (i.e., impoverished empathy and intimacy), alongside deficits in identity and self-direction, has been proposed as a primary sign of PD in new diagnostic systems (e.g., APA, 2013; Morey et al., 2011, 2013). Additional research clearly is needed to investigate the psychological and biological mechanisms driving individual differences in general personality pathology (cf. Lahey et al., 2012).

Likewise, the construct validity of specific PD factors identified in this and other studies awaits systematic study. Due to the unavoidable arbitrariness in how the specific factors were named (as is the case in all factor analytic studies of PD that base factor names on the pattern and strength of factor loadings) and the limited array of external correlates examined here, the nature of the specific PD processes remains uncertain. Future large-scale studies clearly are needed to (a) replicate the present bifactor structure and (b) elaborate the nomological network of the specific factors to foster the convergent and discriminant validity of specific PD processes. Such research will determine whether the structural model implied by the bifactor approach can be useful for etiological research and clinical purposes.

Clinical Implications

Although this was not a treatment-seeking sample, we believe several of our findings are relevant to applied settings. First, general PD severity—and not the presence of one form of PD or another—was the best predictor of General Functioning and therefore may be especially useful for clinical decision-making (e.g., prognosis, optimal treatment intensity). In a similar vein, our results favor a general assessment of personality pathology, as opposed to querying individual disorder categories, especially when assessment time is limited. Computerized adaptive testing models of the general dimension could accelerate assessment even further (see Simms & Clark, 2005). Second, the coherence and construct validity of the general dimension reported here raise the possibility that the efficiency of PD treatment could be improved by targeting the commonalities among PDs. Existing transdiagnostic psychological treatments for clinical disorders were motivated by similar empirical observations (e.g., Barlow et al., 2004; Mansell, Harvey, Watkins, & Shafran, 2009), and such treatments could be adapted to address personality pathology. We speculate that such a treatment would involve fostering emotion regulation capabilities, social skills training, and improving awareness and reappraisal of rigid, maladaptive belief systems. Consistent with that hypothesis, dialectical behavior therapy (Linehan, 1993), a well-validated treatment approaches for borderline PD, addresses several cross-cutting deficits common to PD and other psychopathologies (e.g., emotion regulation, distress tolerance), and has proven...
effective for treating several disparate mental disorders (e.g., Lynch, Morse, Mendelson, & Robins, 2003; Telch, Agras, & Linehan, 2001). After completion of such a transdiagnostic treatment, supplemental treatment modules might be pursued as needed to address areas of remaining dysfunction (e.g., promoting positive affect and social skills training in schizoid PD). The effectiveness of transdiagnostic psychotherapy for PD is an issue that merits continued attention in intervention studies (Sauer-Zavala, Bentley, & Wilner, in press).

Third, identifying an individual’s pathological trait profile appears to provide complementary, and potentially critical, clinical information in some cases. The Instability vs. Rigidity dimension, in particular, might be an important assessment target for cases in which self-harm is a concern (e.g., due to presence of other predictors of suicide; see Sharp et al., 2012). Moreover, as described above, Instability vs. Rigidity was as closely linked with service use as the general PD dimension. These results are not entirely unexpected, given that borderline PD—the primary marker of the Instability vs. Rigidity dimension—surpasses other PDs in rates of suicidal behavior and mental health treatment (Bender et al., 2001; Yen et al., 2003).

Finally, our results reinforce the position that PD and clinical disorders should be assessed jointly when predicting dysfunction and clinical outcomes such as suicidality and need for treatment (Shiner & Allen, 2013). Clinical disorders demonstrated incremental predictive utility for nearly all functioning domains, and in some cases their effects were substantial. For instance, even after accounting for PD dimensions, a depression diagnosis was associated with nearly a three-fold increase in risk for suicidality. The independent influences of PD and clinical disorders observed here replicate epidemiological findings from the NESARC (Grant et al., 2004) and suggest that clinical management of PD should include assessment of coexisting clinical disorder psychopathology and vice versa.

**Limitations**

A number of limitations of the present study highlight directions for future research. First, all analyses were cross-sectional, prohibiting inferences about causal relations between the onset of psychopathology and changes in clinical and functional outcomes. Prospective analyses are needed to determine whether a hierarchical conceptualization of personality pathology can improve the prediction of psychosocial dysfunction over time (cf. Gunderson et al., 2011). Second, the specific dimensions of personality pathology were based on symptom counts from DSM-defined categories, as compared to transdiagnostic personality trait structures that provide more comprehensive coverage of the PD domain (e.g., Five Factor Model; Widiger, Livesley, & Clark, 2009). Also, future bifactor research may benefit from analyzing the full array of PD signs and symptoms to derive specific dimensions in light of PD diagnosis heterogeneity (see, e.g., Markon, 2010). Third, it is important to note that we did not evaluate the validity of the DSM-5 Section III construct of “personality functioning” that is Criterion A of the General Criteria for Personality Disorder. In DSM-5 Section III, personality functioning is defined by the Level of Personality Functioning Scale (LPFS; APA, 2013), which assesses pathology related to identity, self-direction, empathy, and intimacy. To be sure, many of these same features are captured in a general assessment of the PD domain during the SCID-II interview, but they are not completely overlapping.
Thus, while the conceptualization of general PD severity in the present study is roughly equivalent to the personality functioning construct described in *DSM-5* (Hopwood et al., 2011), our data do not specifically support or refute the use of LPFS as an index of PD severity. Other transdiagnostic dimensions—perhaps identified through item response modeling of PD signs and symptoms—may offer equally valid metrics of general personality pathology. Fourth, while the high-risk sample may be considered an advantage for the present analyses, it is unclear to what extent results might generalize to unselected populations. Additionally, exploration of transdiagnostic models among other age groups is needed; it is possible that the nature and/or importance of the specific dimensions—particularly Instability vs. Rigidity and Attention Seeking—change over developmental stages.

**Conclusion**

The present results support a hierarchical transdiagnostic conceptualization of personality pathology. A general transdiagnostic dimension of PD severity was—by a factor of five—a stronger predictor of psychosocial dysfunction than specific dimensions of personality pathology in this high-risk sample. Nevertheless, the specific dimensions provided complementary, and sometimes superior, information about suicide risk and mental health treatment use. Thus, the data appear to uphold the thesis that hierarchical transdiagnostic accounts of PD—emphasizing, in order of clinical importance, a determination of significant overall personality pathology and individual differences on a parsimonious set of specific abnormal personality processes—can enhance the efficiency of research on the psychopathology and clinical correlates of PD. Future empirical work is needed to investigate the hypothesis that etiological and treatment models of PD can be improved by targeting transdiagnostic features of personality pathology.

**Acknowledgments**

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**References**


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Figure 1.
A higher-order latent variable model of psychosocial functioning. Standardized factor loadings are presented and are all statistically significant at a .001 alpha level. The prefix “M” denotes maternal responses and “O” denotes offspring responses. LIFE = Longitudinal Interval Follow-Up Examination; SAS = Social Adjustment Scale; Rec = Recreation.
Table 1

Descriptive Statistics for Personality Disorder and Clinical Disorder Constructs

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>Mean (SD) / N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Depressive Disorder Diagnosis</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>51 (7.2%)</td>
</tr>
<tr>
<td>2. Anxiety Disorder Diagnosis</td>
<td>.35</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>203 (28.8%)</td>
</tr>
<tr>
<td>3. Externalizing Disorder Diagnosis</td>
<td>.23</td>
<td>.07</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>139 (19.7%)</td>
</tr>
<tr>
<td>4. Personality Disorder Diagnosis</td>
<td>.37</td>
<td>.41</td>
<td>.26</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>95 (13.6%)</td>
</tr>
<tr>
<td>5. Paranoid PD Symptoms</td>
<td>.34</td>
<td>.26</td>
<td>.24</td>
<td>.57</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.59 (2.42)</td>
</tr>
<tr>
<td>6. Schizotypal PD Symptoms</td>
<td>.28</td>
<td>.30</td>
<td>.16</td>
<td>.43</td>
<td>.41</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.22 (2.20)</td>
</tr>
<tr>
<td>7. Schizoid PD Symptoms</td>
<td>.14</td>
<td>.11</td>
<td>—</td>
<td>.03</td>
<td>.34</td>
<td>.26</td>
<td>.28</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.53 (1.22)</td>
</tr>
<tr>
<td>8. Histrionic PD Symptoms</td>
<td>.20</td>
<td>.03</td>
<td>.17</td>
<td>.21</td>
<td>.21</td>
<td>.20</td>
<td>.12</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.57 (1.15)</td>
</tr>
<tr>
<td>10. Antisocial PD Symptoms</td>
<td>.17</td>
<td>.09</td>
<td>.31</td>
<td>.35</td>
<td>.32</td>
<td>.22</td>
<td>.10</td>
<td>.16</td>
<td>.25</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
<td>1.89 (4.40)</td>
</tr>
<tr>
<td>11. Borderline PD Symptoms</td>
<td>.41</td>
<td>.32</td>
<td>.31</td>
<td>.48</td>
<td>.60</td>
<td>.49</td>
<td>.20</td>
<td>.30</td>
<td>.35</td>
<td>.36</td>
<td>—</td>
<td></td>
<td></td>
<td>2.39 (3.93)</td>
</tr>
<tr>
<td>12. Avoidant PD Symptoms</td>
<td>.34</td>
<td>.38</td>
<td>.11</td>
<td>.47</td>
<td>.41</td>
<td>.40</td>
<td>.28</td>
<td>—</td>
<td>.01</td>
<td>.17</td>
<td>.06</td>
<td>.34</td>
<td>—</td>
<td>1.83 (2.68)</td>
</tr>
<tr>
<td>13. Dependent PD Symptoms</td>
<td>.35</td>
<td>.25</td>
<td>.16</td>
<td>.31</td>
<td>.31</td>
<td>.32</td>
<td>.15</td>
<td>.21</td>
<td>.23</td>
<td>.17</td>
<td>.40</td>
<td>.43</td>
<td>—</td>
<td>0.67 (1.46)</td>
</tr>
<tr>
<td>14. Obsessive-Compulsive PD Symptoms</td>
<td>.16</td>
<td>.21</td>
<td>—</td>
<td>.03</td>
<td>.45</td>
<td>.42</td>
<td>.33</td>
<td>.18</td>
<td>.25</td>
<td>.40</td>
<td>.14</td>
<td>.30</td>
<td>.23</td>
<td>2.53 (2.78)</td>
</tr>
</tbody>
</table>

Correlations between categorical diagnoses are tetrachoric, and those between categorical diagnoses and continuous symptom scores are biserial.
Table 2
Final Exploratory Bifactor Analysis Solution and Factor Correlations with Personality Disorder Dimensions

<table>
<thead>
<tr>
<th>Symptom Dimension</th>
<th>General&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Submissiveness</th>
<th>Instability vs. Rigidity</th>
<th>Attention Seeking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paranoid</td>
<td>.75</td>
<td>.01</td>
<td>−.02</td>
<td>−.24</td>
</tr>
<tr>
<td>Schizotypal</td>
<td>.58</td>
<td>.16&lt;sup&gt;**&lt;/sup&gt;</td>
<td>.04</td>
<td>.02</td>
</tr>
<tr>
<td>Schizoid</td>
<td>.31</td>
<td>.15&lt;sup&gt;**&lt;/sup&gt;</td>
<td>−.08</td>
<td>−.03</td>
</tr>
<tr>
<td>Histrionic</td>
<td>.42</td>
<td>−.07</td>
<td>−.02</td>
<td>.45&lt;sup&gt;**&lt;/sup&gt;</td>
</tr>
<tr>
<td>Narcissistic</td>
<td>.57</td>
<td>.00</td>
<td>−.21&lt;sup&gt;**&lt;/sup&gt;</td>
<td>.29&lt;sup&gt;***&lt;/sup&gt;</td>
</tr>
<tr>
<td>Antisocial</td>
<td>.40</td>
<td>−.14&lt;sup&gt;**&lt;/sup&gt;</td>
<td>.10</td>
<td>−.04</td>
</tr>
<tr>
<td>Borderline</td>
<td>.80</td>
<td>−.02</td>
<td>.41&lt;sup&gt;***&lt;/sup&gt;</td>
<td>−.03</td>
</tr>
<tr>
<td>Avoidant</td>
<td>.44</td>
<td>.88&lt;sup&gt;***&lt;/sup&gt;</td>
<td>−.02</td>
<td>−.03</td>
</tr>
<tr>
<td>Dependent</td>
<td>.44</td>
<td>.32&lt;sup&gt;***&lt;/sup&gt;</td>
<td>.16&lt;sup&gt;**&lt;/sup&gt;</td>
<td>.18</td>
</tr>
<tr>
<td>Obsessive-Compulsive</td>
<td>.55</td>
<td>−.02</td>
<td>−.34&lt;sup&gt;***&lt;/sup&gt;</td>
<td>.00</td>
</tr>
</tbody>
</table>

Diagnostic Correlations

<table>
<thead>
<tr>
<th></th>
<th>Depressive Disorder</th>
<th>Anxiety Disorder</th>
<th>Externalizing Disorder</th>
<th>YASR Correlations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>.35&lt;sup&gt;***&lt;/sup&gt;</td>
<td>.29&lt;sup&gt;***&lt;/sup&gt;</td>
<td>.24&lt;sup&gt;***&lt;/sup&gt;</td>
<td>Anxiety/Depression</td>
</tr>
<tr>
<td></td>
<td>.13&lt;sup&gt;***&lt;/sup&gt;</td>
<td>.24&lt;sup&gt;***&lt;/sup&gt;</td>
<td>.22&lt;sup&gt;***&lt;/sup&gt;</td>
<td>.54&lt;sup&gt;***&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>.19&lt;sup&gt;***&lt;/sup&gt;</td>
<td>.09&lt;sup&gt;*&lt;/sup&gt;</td>
<td>.18&lt;sup&gt;***&lt;/sup&gt;</td>
<td>.30&lt;sup&gt;***&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>−.05</td>
<td>−.15&lt;sup&gt;***&lt;/sup&gt;</td>
<td>.00</td>
<td>.22&lt;sup&gt;***&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

YASR, Young Adult Self Report.

<sup>a</sup> All loadings on the general factor were statistically significant at a .001 alpha level.
Table 3
Regression of Functioning Outcomes on General Personality Pathology Severity, Specific Personality Pathology Dimensions, and Clinical Disorder Diagnoses

<table>
<thead>
<tr>
<th>Predictors</th>
<th>General Functioning</th>
<th>Social Life Functioning</th>
<th>Family Functioning</th>
<th>Marital Functioning</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b</td>
<td>SE</td>
<td>β</td>
<td>b</td>
</tr>
<tr>
<td><strong>Clinical Disorders</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive</td>
<td>0.29</td>
<td>0.09</td>
<td>.17***</td>
<td>0.27</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0.07</td>
<td>0.04</td>
<td>.07</td>
<td>0.10</td>
</tr>
<tr>
<td>Externalizing</td>
<td>0.21</td>
<td>0.06</td>
<td>.18***</td>
<td>0.05</td>
</tr>
<tr>
<td><strong>Personality Pathology</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall Severity</td>
<td>0.20</td>
<td>0.03</td>
<td>.41***</td>
<td>0.20</td>
</tr>
<tr>
<td>Submissiveness</td>
<td>0.04</td>
<td>0.02</td>
<td>.09</td>
<td>0.08</td>
</tr>
<tr>
<td>Instability vs. Rigidity</td>
<td>0.09</td>
<td>0.03</td>
<td>.14***</td>
<td>0.04</td>
</tr>
<tr>
<td>Attention Seeking</td>
<td>−0.03</td>
<td>0.04</td>
<td>−0.04</td>
<td>−0.06</td>
</tr>
<tr>
<td>Gender</td>
<td>−0.02</td>
<td>0.04</td>
<td>−0.02</td>
<td>−0.15</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Work Functioning</th>
<th>Treatment Use</th>
<th>Suicidality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b</td>
<td>SE</td>
<td>β</td>
</tr>
<tr>
<td><strong>Clinical Disorders</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive</td>
<td>0.41</td>
<td>0.14</td>
<td>.17**</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0.07</td>
<td>0.06</td>
<td>.05</td>
</tr>
<tr>
<td>Externalizing</td>
<td>0.27</td>
<td>0.07</td>
<td>.17***</td>
</tr>
<tr>
<td><strong>Personality Pathology</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall Severity</td>
<td>0.22</td>
<td>0.04</td>
<td>.33***</td>
</tr>
<tr>
<td>Submissiveness</td>
<td>0.06</td>
<td>0.04</td>
<td>.10</td>
</tr>
<tr>
<td>Instability vs. Rigidity</td>
<td>0.20</td>
<td>0.04</td>
<td>.23***</td>
</tr>
<tr>
<td>Attention Seeking</td>
<td>0.02</td>
<td>0.05</td>
<td>.03</td>
</tr>
<tr>
<td>Gender</td>
<td>0.13</td>
<td>0.05</td>
<td>.10**</td>
</tr>
</tbody>
</table>

The regressions involving Social Life, Family, Marital, and Work factors were performed in a model that omitted the superordinate General Functioning factor. All continuous predictors were standardized in the regression models. For gender, 0 = female; 1 = male. OR = proportional odds ratio.