Journal of Abnormal Psychology
2012, Vol. 121, No. 3, 699–706

0021-843X/12/$12.00 DOI: 10.1037/a0029042

An Empirical Examination of Distributional Assumptions Underlying the Relationship Between Personality Disorder Symptoms and Personality Traits

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This article examines the relationship between personality disorder (PD) symptoms and personality traits using a variety of distributional assumptions. Prior work in this area relies almost exclusively on linear models that treat PD symptoms as normally distributed and continuous. However, these assumptions rarely hold, and thus the results of prior studies are potentially biased. Here we explore the effect of varying the distributions underlying regression models relating PD symptomatology to personality traits using the initial wave of the Longitudinal Study of Personality Disorders ($N = 250$; Lenzenweger, 1999), a university-based sample selected to include PD rates resembling epidemiological samples. PD symptoms were regressed on personality traits. The distributions underlying the dependent variable (i.e., PD symptoms) were variously modeled as normally distributed, as counts (Poisson, Negative-Binomial), and with two-part mixture distributions (zero-inflated, hurdle). We found that treating symptoms as normally distributed resulted in violations of model assumptions, that the negative-binomial and hurdle models were empirically equivalent, but that the coefficients achieving significance often differ depending on which part of the mixture distributions are being predicted (i.e., presence vs. severity of PD). Results have implications for how the relationship between normal and abnormal personality is understood.

Keywords: personality disorders, personality traits, count regression, zero-inflated distributions, hurdle models

Personality disorder (PD) researchers have called for an integration of normal and pathological personality functioning within comprehensive dimensional models of personality (e.g., Depue & Lenzenweger, 2005; Widiger, Livesley, & Clark, 2009). It has been argued that normal personality exists on a continuum of functioning with PDs (Widiger & Trull, 2007). A large empirical literature examining the relationship between traits and PDs contributed to the seminal decision to use a dimensional trait system to conceptualize phenotypic variation in PD in DSM-5 (Skodol et al., 2011). This research relies primarily on cross-sectional correlations and linear regression to model the relationship between personality traits and PD symptoms. However, these analytic tools suffer from limitations when the underlying distribution of the variables is severely non-normal, as is the case with PD symptoms in the population. Alternative approaches that more accurately model the observed PD symptom distribution may provide better estimates of the relationship between personality and its disorder, and may offer new insights into the nature of that relationship.

The extant nosology of PDs represents personality pathology as a collection of "distinct clinical syndromes" (p. 689; American Psychiatric Association, 2000) that differ categorically from normative functioning and each other. These distinctions have been criticized as arbitrary and lacking in robust scientific support (Widiger & Trull, 2007), and diagnostic criteria treated as dimensional markers for disorders perform better by empirical standards (Morey et al., 2007). However, measuring disorders *dimensionally* cannot confirm their *continuity* with normal functioning (Lenzenweger & Clarkin, 2005), and the accumulated research presents a mixed picture related to this issue. Meta-analyses (Samuel & Widiger, 2008; Saulsman & Page, 2004) demonstrate that basic personality traits exhibit significant and replicable relationships to PD, but the association between traits and PDs are generally only modest in size ($Mdn\ |r| = .15$; range = .02–.54; Samuel & Widiger, 2008). Furthermore, in regression models, the five-factor

This article was published Online First June 25, 2012.

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This research was supported by grants (MH45448, Lenzenweger; F31MH087053, Wright) from the National Institute of Mental Health, Washington, DC. The views and opinions contained within are solely those of the authors and do not reflect the official position of the funding source.

This manuscript reports on work that represents a portion of the first author's dissertation. We thank Jerry S. Wiggins for providing consultation on the initial use of the Revised Interpersonal Adjectives Scales–Big Five, and Lauren Korfine for project coordination in the early phase of the study. We are indebted to Michael N. Hallquist for generously sharing his time to instruct the first author on the intricacies of the R statistical package. Emily B. Ansell, Nicholas R. Eaton, Christopher J. Hopwood, and Kristian E. Markon each provided helpful comments on an early draft of this work.

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domains and facets generally only explain a minority of the variance in PDs (e.g., Bagby, Costa, Widiger, Ryder, & Marshall, 2005). Thus, normal personality traits and PD are not interchangeable representations of functioning, despite clearly recognizable shared content (Krueger et al., 2011). Other researchers and theorists have highlighted the general impairment associated with PDs (Kernberg, 1984; Livesley & Jang, 2005; Pincus, 2005). Relatedly, a number of investigators have found that PD is primarily characterized by higher neuroticism, lower conscientiousness, and lower agreeableness, with less in the way of distinction beyond this core trait profile (Hopwood et al., 2011; Morey et al., 2002; Saulsman & Page, 2004). Thus, it may be that a particular combination of traits reflects personality pathology generally, with further differentiation occurring in the presence of this profile.

The question of *continuity* in personality and its pathology has taken center stage in the development of the DSM-5, which will shift to a dimensional model based on the robust empirical findings suggesting that PD is fundamentally dimensional in nature (Skodol et al., 2011). However, in part because of issues raised here, DSM-5 will distinguish between personality dysfunction and the description of that dysfunction using pathological traits (Hopwood et al., 2011; Krueger et al., 2011). Indeed, the sum of the empirical literature leaves an unclear picture of how PD and personality traits are related to each other. It may be that PD is dimensionally continuous with basic personality traits (e.g., Depue & Lenzenweger, 2005). Alternatively, continuities and discontinuities may exist in these relationships, with some driven by the *presence* of PD and others, perhaps more subtle, driven by the *severity* of PD beyond its presence (see Lenzenweger & Clarkin, 2005). What is clear is that the theoretical goal of integrating normative personality traits and PD remains elusive.

The key theoretical questions of how personality and PD relate to one another are also inherently questions of methodology. Dimensional approaches can make varied distributional assumptions that may have relevance for advancing the understanding of the relationship between normal and abnormal personality. Research examining this question relies almost exclusively on standard correlation and linear regression. These approaches make several important assumptions (i.e., normality of residuals, homoscedasticity, linearity of relationship, independence), that, when violated, can bias estimation (Cohen, Cohen, West, & Aiken, 2003). Less serious are biased standard errors, which can produce incorrect significance tests for parameters. More serious violations occur when the actual effect of a relationship is misestimated. A major contributing source to the violation of these assumptions is the distribution of the variables being modeled.

In the population, the actual distribution of PD symptoms is highly positively skewed with a majority of individuals suffering from no symptoms. Figure 1 provides an example of such a distribution using the narcissistic personality disorder (NPD) features in the first wave of the *Longitudinal Study of Personality Disorders* (LSPD; Lenzenweger, 1999), the dataset used here. This histogram is characteristic of a count distribution. Modeling techniques for counts are primarily based on the Poisson and Negative-Binomial (NB) distributions and can be used for regression when appropriate (Cameron & Trivedi, 1998; Long, 1997). Of note is the large number of zeros in the distribution, which has important implications for modeling the relationship between the symptoms and other variables of interest. These zeros carry important information about who does and does not possess symptoms of PD. With large numbers of zeros in the data, two potential modifications to basic count models are recommended: zero-inflated and hurdle models (Atkins & Gallop, 2007; Cameron & Trivedi, 1998; Long, 1997). Zero-inflated models estimate a group of individuals based on the excess of zeros beyond a standard Poisson or NB model, which are treated as individuals who can only take on a zero value. Hurdle models make a binary distinction between those who have a zero value versus those who have a nonzero value. Despite this distinction in the treatment of zero-values, both models estimate separate regression coefficients for the zero versus nonzero (e.g., absence vs. presence of PD) and the count (e.g., severity of PD) portion of the models. These models are ideal for evaluating whether the traits that give rise to any symptoms of PDs are the same as those that predict the number of symptoms once present.

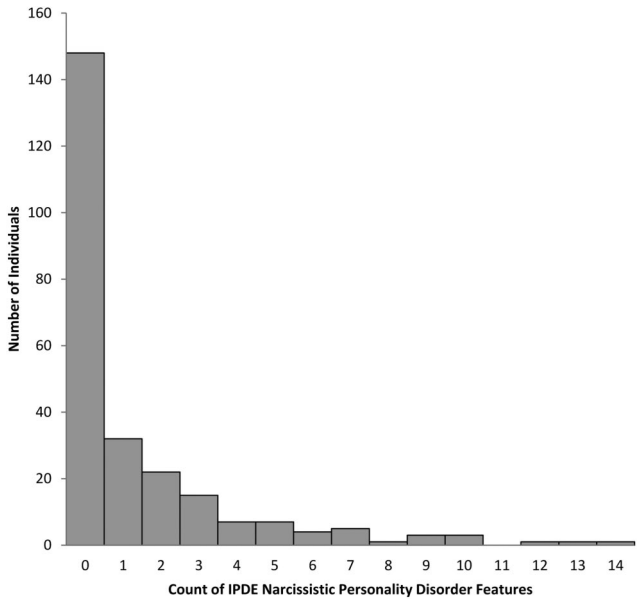

Figure 1. Observed Longitudinal Study of Personality Disorders (LSPD) narcissistic personality disorder features.

The current study explores the relationship of personality traits to PD symptoms using regression models capable of appropriately modeling the distribution of symptoms in the population. We use the LSPD sample, which is made up of participants recruited both with and without significant pathology, unlike samples selected based on shared diagnostic status or for high levels of pathology. As a result, the distributions of PD symptoms closely matches those found in epidemiological samples (Lenzenweger, 2008). The LSPD dataset is ideal for the types of investigations pursued here because it captures the boundary between those individuals whose personalities function well and those who evidence dysfunction.

Our first aim was to evaluate whether the assumptions of linear regression are violated when predicting PD symptoms and to compare Poisson, zero-inflated Poisson (ZIP), Poisson hurdle (PH), negative-binomial (NB), zero-inflated negative-binomial (ZINB), and negative-binomial hurdle (NBH) regression models that predict PD symptoms from personality trait scores. Our second and more substantive aim involves comparing the patterns of

significant regression coefficients to determine the effect of varying distributional assumptions on the relationship between traits and PDs. Attention is also given to differences in patterns in the prediction of presence versus severity of PD features in the zero-inflated/hurdle models.

Method

Participants

Detail concerning the participant selection procedure in the LSPD is given elsewhere (Lenzenweger, 1999). The 250 participants were drawn from a nonclinical university population, are balanced on gender (53% women), and the mean age at entry into the study was 18.88 years ($SD = 0.51$). Approximately half of the participants were selected based on putative positive PD status as assessed by a self-report PD screener. This ensured an adequate sampling of PD pathology in a nonclinical population. Based on diagnostic assessments conducted by experienced clinicians, 11% of the participants qualified for a PD diagnosis of some sort, and 45.2% met the criteria for an Axis I diagnosis (Lenzenweger, 1999). Rates of diagnosed PDs in the sample were as follows: paranoid = 1.2%, schizoid = 1.2%, schizotypal = 1.6%, antisocial = 0.8%, borderline = 1.6%, histrionic = 3.5%, narcissistic = 3.1%, obsessive–compulsive = 1.6%, passive-aggressive = 0.8%, avoidant = 1.2%, dependent = 0.8%, and not otherwise specified = 4.3%. It is important to note that these rates closely mirror the rates of PD found in large epidemiological samples (Lenzenweger, 2008).

Measures

At baseline, participants completed a diagnostic clinical interview and self-report personality measures. Only data from these initial assessments are used here.

The *International Personality Disorder Examination* (IPDE; Loranger, 1999) was used to assess PD symptomatology. The *DSM–III–R* criteria were assessed in this study because these were in effect at the time the LSPD was undertaken. The interrater reliability for IPDE assessments (based on intraclass correlation coefficients) was excellent, ranging between .84 and .92 for all PD dimensions used for this study. For each symptom, an individual may receive a score of 0 (*absent or normal*), 1 (*exaggerated/accentuated*), or 2 (*criterion/pathological*). These values are summed within each disorder to create a count of disorder related features.

The *Revised Interpersonal Adjective Scales–Big Five* (IASR-B5; Trapnell & Wiggins, 1990) contains 124 trait descriptive adjectives rated on a 0 to 8 scale that provides scores for the personality trait dimensions of Dominance, Affiliation, Conscientiousness, Neuroticism, and Openness. Coefficient alphas ranged from .82 to .96.

Results

A series of regression models were estimated in R Package PSCL (Zeileis, Kleiber, & Jackman, 2008). Each PD's count and the Total PD count were regressed on each personality trait score separately. A model was estimated for each personality trait separately in keeping with past literature, and because the traits are orthogonal in theory, but in practice often exhibit relationships that attenuate regression coefficients when entered simultaneously in a model. Trait predictors were standardized. For each trait-PD pairing, a set of models was estimated with Normal, Poisson, ZIP, PH, NB, ZINB, and NBH distributions specified for the PD counts.

The linear regression models were evaluated by testing linearity, normality of the residuals, and homoscedasticity. A minority (22%) of the models violated the assumption of linearity. However, all model residuals exhibited significant skewness ($M = 2.75$; range = 1.83–4.41) and excess kurtosis ($M = 10.50$; range = 3.59–25.89). Visual analyses suggested that the assumption of homoscedasticity was untenable for all models. In addition, 36% of the models predicted negative symptom counts. Thus, the assumptions of linear regression models do not hold and a considerable proportion result in the prediction of impossible values, all of which emphasizes the need for count based models.

Poisson regression models make strict assumptions about the variance of the observations that are frequently violated in applied research (Coxe, West, & Aiken, 2009). The NB differs from the Poisson in that it estimates an additional parameter for the variance of counts, and therefore these two models are nested and can be compared using likelihood ratio tests (LTRs; Long, 1997). In each case, the NB model was a significant improvement over its Poisson counterpart. Models which account for the large stack of zeros in the data using either zero-inflation or a hurdle are non-nested relative to the basic count models and each other, and therefore require an alternative test. To compare non-nested models, we employed a Vuong (1989) non-nested LRT. The Vuong LRT compares two models under the null hypothesis "that the competing models are equally close to the true data generating process" (p. 307) such that a significant statistic favors one model over the other, and a nonsignificant statistic suggests that the models are equivalent. When models are quantitatively equivalent additional criteria are required to select between models. One common approach is to select the model with the fewest estimated parameters, placing a premium on parsimony. Yet there may be a conceptual/theoretical preference for considering more complex models under these conditions. As Atkins and Gallop (2007) argue, a histogram like that depicted in Figure 1 would appear to suggest two separate processes: There are those individuals without any personality pathology, and among those that have it, a range of severity.[1] They go on to note that the two-step count models are well-suited for investigating "psychological models in which there are two processes and where the determinants of those processes differ" (p. 733). Our results of a comprehensive comparison of models suggest that with few exceptions, the NB, ZINB, and NBH models are equivalent.[2] When a model was favored, it was a two-step model, and between those the hurdle models. In addition, a number of ZINB models were nonidentified. Therefore, we retain for consideration the NB and NBH models.

Table 1 reports the regression coefficients for the Normal, NB, and NBH and their significance. No formula exists for transforming the different results to the same effect size for direct compar-

[1] The distribution in Figure 1 is highly representative of each of the PDs in the LSPD.

[2] Full results available upon request.

Table 1
Summary of Coefficients From Models Regressing Personality Disorder Symptoms on Personality Traits

	Normal		Negative-binomial		Negative-binomial hurdle			
Disorder	β	*p*	RR	*p*	OR(h)	*p*(h)	RR(NB)	*p*(NB)
Paranoid								
DOM	−0.021	0.741	0.964	0.789	0.881	0.358	1.058	0.756
LOV	**−0.244**	**0.000**	**0.544**	**0.000**	**0.572**	**0.000**	0.587	0.074
CONS	−0.121	0.054	0.772	0.053	**0.667**	**0.004**	1.027	0.907
NEUR	**0.357**	**0.000**	**2.296**	**0.000**	**2.045**	**0.000**	**2.275**	**0.001**
OPEN	0.114	0.072	**1.374**	**0.021**	1.182	0.229	1.717	0.078
Schizoid								
DOM	**−0.252**	**0.000**	**0.628**	**0.001**	**0.437**	**0.000**	0.950	0.753
LOV	**−0.380**	**0.000**	**0.502**	**0.000**	**0.561**	**0.000**	**0.579**	**0.000**
CONS	−0.090	0.155	0.780	0.098	0.885	0.400	0.740	0.182
NEUR	0.003	0.967	1.006	0.971	1.165	0.293	0.836	0.334
OPEN	−0.007	0.913	0.984	0.914	0.839	0.233	1.265	0.269
Schizotypal								
DOM	**−0.308**	**0.000**	**0.680**	**0.000**	**0.650**	**0.001**	**0.714**	**0.005**
LOV	**−0.308**	**0.000**	**0.600**	**0.000**	**0.542**	**0.000**	**0.683**	**0.022**
CONS	−0.068	0.280	0.890	0.279	0.878	0.310	0.918	0.615
NEUR	**0.237**	**0.000**	**1.447**	**0.000**	**1.417**	**0.008**	**1.429**	**0.023**
OPEN	0.065	0.309	1.118	0.306	1.037	0.777	1.198	0.294
Antisocial								
DOM	0.120	0.055	1.259	0.101	1.217	0.152	1.209	0.218
LOV	**−0.248**	**0.000**	**0.562**	**0.000**	**0.579**	**0.000**	**0.696**	**0.037**
CONS	−0.092	0.144	0.791	0.091	0.866	0.278	0.796	0.248
NEUR	**0.130**	**0.039**	**1.480**	**0.004**	1.167	0.247	**1.601**	**0.027**
OPEN	0.025	0.688	1.056	0.699	1.246	0.106	0.876	0.452
Borderline								
DOM	0.052	0.415	1.096	0.480	1.091	0.508	1.070	0.675
LOV	**−0.269**	**0.000**	**0.638**	**0.000**	**0.648**	**0.002**	**0.727**	**0.022**
CONS	**−0.161**	**0.010**	**0.741**	**0.017**	**0.638**	**0.001**	0.960	0.804
NEUR	**0.367**	**0.000**	**2.143**	**0.000**	**1.692**	**0.000**	**2.034**	**0.000**
OPEN	0.064	0.312	1.135	0.327	1.062	0.647	1.163	0.372
Histrionic								
DOM	**0.189**	**0.002**	**1.350**	**0.012**	**1.456**	**0.006**	1.158	0.186
LOV	0.027	0.668	1.038	0.757	1.017	0.894	1.041	0.691
CONS	**−0.156**	**0.013**	0.815	0.081	0.795	0.077	0.875	0.165
NEUR	**0.308**	**0.000**	**1.644**	**0.000**	**1.808**	**0.000**	**1.292**	**0.018**
OPEN	0.093	0.141	1.158	0.218	1.267	0.071	1.026	0.814
Narcissistic								
DOM	**0.201**	**0.001**	**1.343**	**0.017**	1.266	0.075	**1.295**	**0.017**
LOV	**−0.289**	**0.000**	**0.616**	**0.000**	**0.665**	**0.003**	**0.699**	**0.002**
CONS	−0.088	0.161	0.866	0.243	**0.775**	**0.049**	0.989	0.923
NEUR	**0.313**	**0.000**	**1.705**	**0.000**	**2.273**	**0.000**	1.193	0.150
OPEN	**0.135**	**0.033**	**1.292**	**0.039**	**1.382**	**0.015**	1.094	0.504
Avoidant								
DOM	**−0.409**	**0.000**	**0.552**	**0.000**	**0.499**	**0.000**	**0.659**	**0.000**
LOV	**−0.279**	**0.000**	**0.583**	**0.000**	**0.543**	**0.000**	0.759	0.087
CONS	**−0.167**	**0.007**	**0.763**	**0.023**	0.795	0.082	0.793	0.076
NEUR	**0.377**	**0.000**	**1.870**	**0.000**	**2.596**	**0.000**	1.276	0.066
OPEN	**−0.126**	**0.047**	**0.787**	**0.049**	0.804	0.103	0.837	0.248
Dependent								
DOM	**−0.138**	**0.027**	**0.791**	**0.044**	**0.736**	**0.022**	0.880	0.489
LOV	0.037	0.558	1.082	0.513	1.105	0.451	1.023	0.919
CONS	**−0.159**	**0.011**	**0.752**	**0.013**	**0.693**	**0.006**	0.856	0.444
NEUR	**0.425**	**0.000**	**2.221**	**0.000**	**2.818**	**0.000**	**1.803**	**0.005**
OPEN	0.004	0.947	1.008	0.944	0.984	0.901	1.045	0.839
Obsessive-compulsive								
DOM	−0.044	0.483	0.953	0.628	**0.757**	**0.033**	1.078	0.415
LOV	**−0.340**	**0.000**	**0.632**	**0.000**	**0.628**	**0.001**	**0.686**	**0.000**
CONS	−0.065	0.300	0.916	0.372	0.911	0.464	0.932	0.508
NEUR	**0.284**	**0.000**	**1.476**	**0.000**	**1.785**	**0.000**	**1.239**	**0.041**
OPEN	0.063	0.318	1.083	0.421	1.017	0.891	1.116	0.280

Table 1 (*continued*)

	Normal		Negative-binomial		Negative-binomial hurdle			
Disorder	β	*p*	RR	*p*	OR(h)	*p*(h)	RR(NB)	*p*(NB)
Total PD								
DOM	−0.051	0.423	0.958	0.587	0.858	0.393	0.971	0.673
LOV	**−0.355**	**0.000**	**0.686**	**0.000**	0.797	0.227	**0.684**	**0.000**
CONS	**−0.179**	**0.004**	**0.820**	**0.011**	1.055	0.758	**0.794**	**0.004**
NEUR	**0.437**	**0.000**	**1.646**	**0.000**	**2.002**	**0.001**	**1.572**	**0.000**
OPEN	0.078	0.221	1.095	0.249	1.105	0.573	1.090	0.293

Note. DOM = dominance; LOV = affiliation; CONS = conscientiousness; NEUR = neuroticism; OPEN = openness; OR = odds ratio; RR = rate ratio; NB = negative-binomial; h = hurdle class; PD = personality disorder. Boldface indicates $p < .05$.

ison across models. Therefore, what is most informative is the valence and significance level of each coefficient. Odds/rate ratios of 1.0 indicate no effect, and those below 1.0 indicate a negative association between predictor and outcome. Given the number of coefficients we highlight notable findings, and refer readers to Table 1 for a more detail. Importantly, similar patterns emerge across all models. Specifically, radical differences, such as a valence change, do not occur. However, pattern differences in significant coefficients across steps of the NBH models suggest different processes associated with the presence versus severity of PD symptoms of different types. We briefly summarize these findings organized around the trait dimensions.

Dominance most often predicted PD presence (schizoid, histrionic, dependent, obsessive-compulsive) and less commonly PD severity (narcissistic) or both (schizotypal, avoidant). *Affiliation,* in contrast, tended to predict both presence and severity (schizoid, schizotypal, antisocial, borderline, narcissistic, and obsessive-compulsive) and otherwise just presence (paranoid, avoidant) when considering individual PDs. *Conscientiousness* only predicted PD presence (paranoid, borderline, narcissistic, dependent). *Neuroticism* was a strong predictor, most commonly of both presence and severity (paranoid, schizotypal, borderline, histrionic, dependent, obsessive-compulsive). Interesting deviations from this include the fact that neuroticism only predicted severity in antisocial, but not presence, and presence but not severity in narcissistic features. *Openness* only predicted presence of Narcissistic PD, a result that is not easily interpreted. Finally, the Total PD symptom model is considered separately as the hurdle step predicting presence reflects a more stringent step between those with any PD and those with none at all. Here only neuroticism is predictive of both PD presence and severity, although conscientiousness and affiliation also predict severity.

Discussion

The current study addresses an implicit assumption and likely limitation in much of the prior work linking personality traits and PD—specifically, although PD is not a normally distributed phenomenon in the population, it has consistently been modeled as such. First, we demonstrated that the basic assumptions of linear regression are violated and frequently result in the prediction of impossible values (i.e., negative counts). Second, we found that NB and NBH models do a comparable job of fitting the count distributions of PD symptoms and they cannot be distinguished quantitatively. Nevertheless, despite the flexibility of the NB distribution to account for a large proportion of individuals with zero values, this feature of the data is suggestive of distinct processes that are worth examining via two-step count models (Atkins & Gallop, 2007; Long, 1997). With these models interesting differences emerge across the two steps. When predicting individual diagnostic constructs, results suggest that neuroticism and affiliation are predictive of both PD presence and severity, whereas dominance is more often, and conscientiousness is exclusively, predictive of the presence of PD symptoms.

These results have implications for understanding the relationship between normal-range personality traits and PD. Issues related to both dimensionality and continuity in personality and PD have emerged from the proposed revisions for DSM-5. The proposed two-step diagnostic process for DSM-5 defines PD using a continuum of self/interpersonal impairment, with separate maladaptive personality traits provided to characterize phenotypic variation in the expression of an individual's core personality pathology. The models employed here are consistent with this approach, distinguishing between aspects of personality related to the presence of PD, and those related to its expression once present. Contrasting the results of the models predicting the count of all PD symptoms with the other model is also informative. The Total PD hurdle models are special in that they serve to represent general personality pathology, and the first step differentiates between those who have absolutely no pathology from those with any degree of pathology. The Total PD models suggest that only neuroticism differentiates those with any pathology from those without, which is consistent with research that shows it is an important predictor of myriad public health outcomes, psychological and otherwise (Lahey, 2009). Neuroticism also predicts severity along with low affiliation and low conscientiousness. This pattern of associations with severity was the same as that found by Hopwood and colleagues (2011) in a clinical sample. Findings here point to the fact that other variables besides normative traits (with the exception for a propensity to experience negative emotions) are responsible for the presence any PD, although other traits can characterize the variability in phenotypic expression of distinct PDs and overall PD severity. *The implication is that normative traits alone are not ideal to differentiate normal from abnormal.* The DSM-5 proposal to draw on self and interpersonal processes to define general personality pathology with traits used to clarify specific forms of PD is consistent with these results, although future research should incorporate the distinct predictors in these models to formally test the proposal.

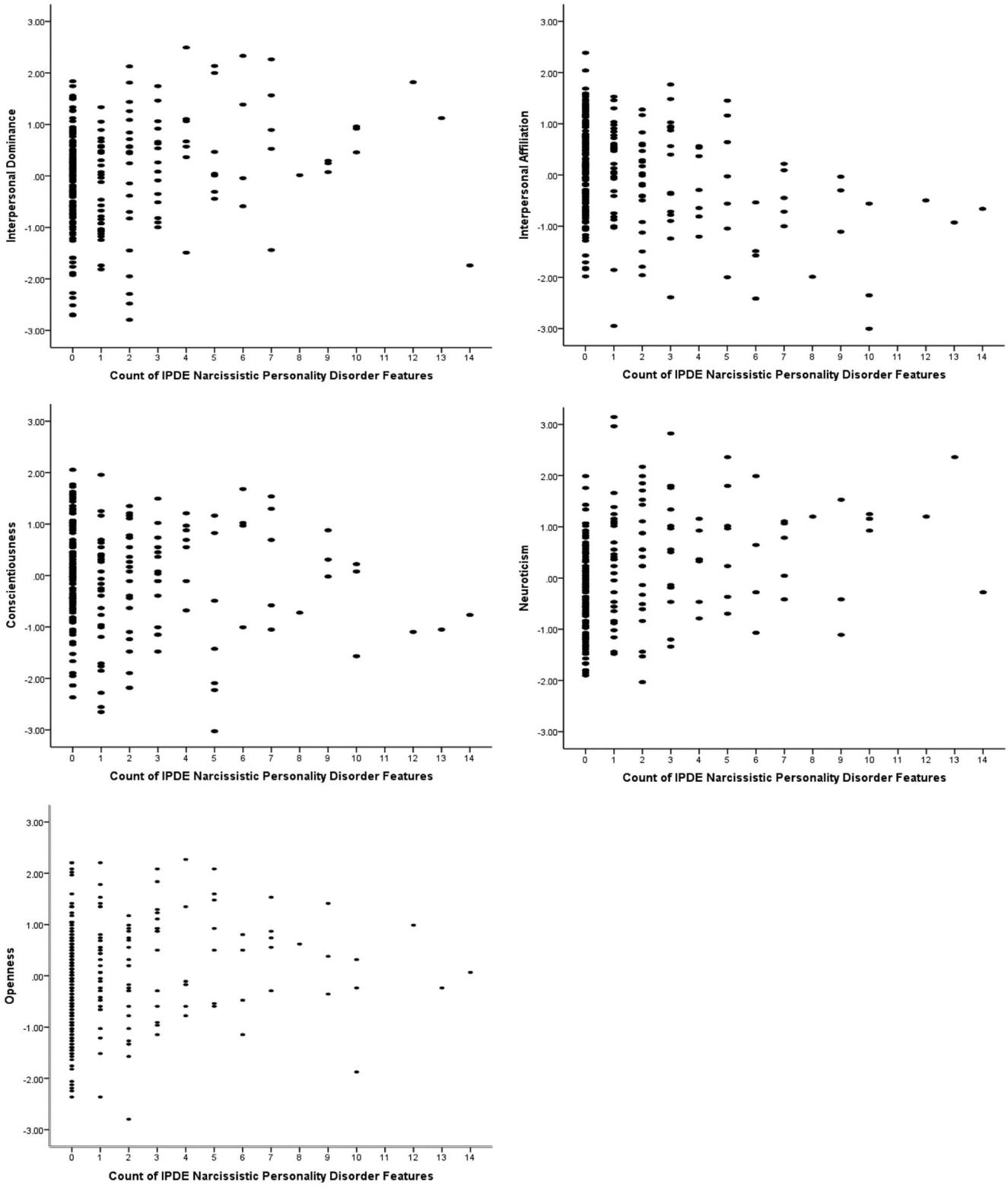

Figure 2. Scatter plots of personality trait scores and narcissistic personality disorder (NPD) features.

A complementary way of understanding these results is that basic traits exert themselves at different levels of pathology. Limited prior research suggests that the level of traits across the spectrum of PD is nonlinear (O'Connor, 2005), indicating that normative traits may be more or less informative for distinguishing individuals at different levels of PD dimensions. Figure 2 illustrates this, continuing with NPD as the exemplar. Trait scores for those individuals without symptoms occur across the range of values, indicating that knowing someone's trait level without knowing their pathology is often diagnostically uninformative. For example, there are individuals at all levels of Dominance, including high levels, without any NPD symptoms. Yet, once there is any narcissistic pathology, rising severity is associated with increases in dominance. The opposite is true with neuroticism. Those without narcissistic pathology are lower on average, but once pathology is present, neuroticism is unrelated to severity. These fine-grained relationships suggest that PDs are not reducible to sums of basic traits, but are more complex in their structure of associations.

Several caveats must be mentioned. First, our present sample was more homogenous in age, education, and social class than the U.S. population. Second, given that the participants were selected from a population of first-year university students, the sample may have been somewhat censored for individuals affected by very severe PDs. However, the results from the linear regression models are highly consistent with prior work (e.g., Samuel & Widiger, 2008), suggesting generalizability. Additionally, the distributions of all psychiatric disorders in the LSPD sample are consistent with the U.S. population distribution (Kessler, Chiu, Demler, & Walters, 2005; Lenzenweger, 2008). Third, although some may feel that running individual models for each trait does not provide the most accurate picture of these relationships, the overwhelming majority of prior research adopts this approach, and therefore we employed it to provide a clear comparison for readers. Fourth, we should highlight that the linear regression models are still quite robust, and exhibit patterns of associations consistent with the NB models. Thus, we are confident that prior studies have accurately identified general relationships between traits and PD. However, a necessary next step in the empirical study of PD is to move beyond this level of analysis to elucidate the exact structure of these relationships in addition to identifying etiologic and mechanistic processes (e.g., underlying neurobehavioral systems, see Depue & Lenzenweger, 2005).

In summary, we examined the effect of varying the distribution of PD symptoms in regression models with personality traits. To appropriately model them requires the use of count distributions, and two-step count models provide opportunities to examine discontinuities in these relationships. In the past, modeling the distributions implemented here might have been more challenging, but a number of user-friendly statistical packages now include these as standard features. We used R, but Mplus, Stata, SAS, and SPSS can employ some or all of these distributions. When these approaches are adopted, a more refined picture emerges suggesting that PD is not merely the tail end of a continuous distribution of normal traits, and the traits associated with the presence of PD are not always those associated with increasing severity. Although we do not argue that these results are definitive and recognize that they should be replicated, we suggest that these analytic approaches are more appropriate, have the potential to elucidate some of the issues associated with continuity and discontinuity in personality and its pathology, and inform ongoing efforts to refine the diagnosis of PD.

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Received October 2, 2011
Revision received April 27, 2012
Accepted May 22, 2012 ■