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Predicting depression outcomes throughout inpatient treatment using the general and specific personality disorder factors

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Abstract

Background. Clinical intuition suggests that personality disorders hinder the treatment of depression, but research findings are mixed. One reason for this might be the way in which current assessment measures conflate general aspects of personality disorders, such as overall severity, with specific aspects, such as stylistic tendencies. The goal of this study was to clarify the unique contributions of the general and specific aspects of personality disorders to depression outcomes.

Methods. Patients admitted to the Menninger Clinic, Houston, between 2012 and 2015 ($N = 2352$) were followed over a 6–8-week course of multimodal inpatient treatment. Personality disorder symptoms were assessed with the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, 4th edition Axis II Personality Screening Questionnaire at admission, and depression severity was assessed using the Patient Health Questionnaire-9 every fortnight. General and specific personality disorder factors estimated with a confirmatory bifactor model were used to predict latent growth curves of depression scores in a structural equation model.

Results. The general factor predicted higher initial depression scores but not different rates of change. By contrast, the specific borderline factor predicted slower rates of decline in depression scores, while the specific antisocial factor predicted a *U* shaped pattern of change.

Conclusions. Personality disorder symptoms are best represented by a general factor that reflects overall personality disorder severity, and specific factors that reflect unique personality styles. The general factor predicts overall depression severity while specific factors predict poorer prognosis which may be masked in prior studies that do not separate the two.

With clinical depression ranked as the world's leading cause of disability (World Health Organization, 2017), there is a pressing need to understand predictors of prognosis. One important aspect of depression is that it frequently co-occurs with other disorders, including personality disorders (Friborg et al., 2014). Patients diagnosed with a comorbid personality disorder (PD) show a more severe and persistent course of depression when left untreated (Cyranowski et al., 2004; Grilo et al., 2010). Clinical intuition suggests that PDs hinder treatment for depression (Clarkin, Petrini, & Diamond, 2019). Yet, results from meta-analyses are mixed: some support the link between PDs and poorer depression outcomes (Newton-Howes et al., 2014; Newton-Howes, Tyrer, & Johnson, 2006; Reich, 2003) while others not (Kool et al., 2005; Mulder, 2002).

Studies vary widely in their choice of treatments, outcome measures, and sample characteristics, making any task of aggregating findings challenging and inconclusive (French, Turner, Dawson, & Moran, 2017). However, a consistent finding is that controlled studies tend to report a weaker relationship between PDs and depression outcomes (Mulder, 2002). For instance, controlling for baseline depression severity often negates the adverse effect of PDs on depression outcomes (De Bolle et al., 2011; Erkens et al., 2018; van Bronswijk et al., 2018). This implies that PDs are associated with higher depression scores throughout treatment, but the pattern of change is no different to patients without a PD diagnosis (Fowler et al., 2018; Moradveisi, Huibers, Renner, Arasteh, & Arntz, 2013). In short, a PD diagnosis does not alter general responses to treatment; it just predicts a poorer start.

Another issue concerns the measurement validity of PDs. PDs are assessed using categorical criteria that represent distinct entities, but comorbidity rates among PDs are too high to be considered truly distinct (Tyrer, Reed, & Crawford, 2015). Predicting depression outcomes from the presence of a specific personality disorder would conflate the unique aspects of that disorder with aspects shared with other disorders. As Mulder (2002) put it, 'Classification problems mean that it remains unclear whether personality disorder categories

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are a general measure of personality pathology affecting outcome or whether individual categories, or clusters, predict different outcomes.' (p. 366). Unless the general and specific aspects of PDs are separated out, it is uncertain how each contributes to depression outcomes.

A statistical method for separating out the general and specific aspects of a measure is the bifactor model (Markon, 2019). In this factor analytic model, the covariance among a set of items is attributed to a general latent variable or 'factor' that summarizes the common variance among items, as well as specific factors that summarize the covariance among specific clusters of items (Reise, 2012). Put differently, responses to each item are decomposed for the variance associated with a general underlying construct, as well as the variance associated with specific constructs. These sources of variance are considered orthogonal, that is, the specific factors reflect distinct constructs not explained by the general construct (Chen, West, & Sousa, 2006).

A growing number of studies have shown that the positive correlations among PD symptom ratings or diagnoses are best explained by a general PD factor, as well as specific factors that reflect individual PDs or PD clusters (Conway, Hammen, & Brennan, 2016; Jahng et al., 2011; Sharp et al., 2015; Williams, Scalco, & Simms, 2018; Wright, Hopwood, Skodol, & Morey, 2016). The general PD factor is thought to reflect the severity of individuals' personality dysfunction on a continuum (Sharp et al., 2015), and predicts social functioning, occupational functioning, treatment use, and suicidality (Conway et al., 2016). The meaning of the specific factors is less clear, but they are thought to reflect stylistic expressions of disturbance (Wright et al., 2016). While the bifactor model is primarily a statistical tool for estimating the general and specific variance within a measure, it also maps onto alternative nosologies of PD that separate out severity and style (Skodol et al., 2011b; Tyrer et al., 2011).

The mixed predictive value of PDs for depression outcomes might result from current PD measures conflating two sources of variance – general and specific personality pathology – that differ in their direction of influence. For instance, if general PD reflects overall illness severity, then it may predict higher depression scores overall, giving the impression that PDs predict poorer outcomes. However, if general PD or its sequelae are controlled for (e.g. via baseline randomization or covarying baseline depression severity), then depression scores might normalize, giving the impression that PDs do not predict poorer outcomes. In either case, general PD would predict the overall severity of depression, not the rate of change. Specific PD factors might reflect stylistic expressions that predict differential rates of change, but these effects are masked by general PD severity. We tested this hypothesis by estimating the unique contribution of general and specific PD factors to changes in depression severity over an inpatient treatment using the bifactor model and latent growth models.

Table 1. Clinical and demographic characteristics of the inpatient sample ($N = 2352$)

Sample Characteristic	*M* or *N*	S.D. or %
Clinical		
PHQ-9 (admission)	15	7
Minimal or none (0–4)	233	10%
Mild (5–9)	327	14%
Moderate (10–14)	455	18%
Moderate severe (15–19)	575	24%
Severe (20–27)	762	32%
Length of Stay (weeks)	6	3
Episode Number		
First admission	2055	87%
>1 admission	297	13%
Program		
Hope	641	27%
CPAS	379	16%
Compass	758	32%
PIC	574	24%
Demographic		
Age	35	15
Sex		
Female	1120	48%
Male	1232	52%
Racial Background		
White or Caucasian	2096	89%
Other[a]	255	11%
Highest Level of Education		
Some schooling	56	2%
High School Diploma or Equivalent	211	9%
Some College	814	35%
Bachelors, Technical, or Associates Degree	761	33%
Postgraduate (Masters, Doctoral, or Professional Degree)	481	21%
Marital Status		
Married	1760	75%
Never married/separated	592	25%

Compass, Compass Program for Young Adults (18–30); Hope, Hope Program for Adults; CPAS, Comprehensive Psychiatric Assessment Service; PIC, Professionals in Crisis program.
[a]Includes Asian, Black or African-American, Native American or Other Pacific Islander, and Multiracial.

Method

Participants

The sample consisted of 2352 inpatients admitted to the Menninger Clinic, Houston, between June 2012 and June 2015. Full demographics are presented in Table 1. Patients were mostly White/Caucasian American (89%), middle-aged ($M = 35$, S.D. = 15), and a mix of sexes (48% female). Most participants underwent some form of higher education, including some college (35%), completing a Bachelor's, Technical or Associates Degree (33%), or attaining a postgraduate degree or doctorate (21%). There were no exclusion criteria; participants of all diagnoses and severity levels were recruited and included in the analysis. Over half (56%) of patients reported moderately severe or severe depression on the Patient Health Questionnaire-9 (PHQ-9). Data were collected as part of the hospital's ongoing Adult Outcomes Project, which aims to integrate research and routine clinical practice (Allen et al., 2009). Collection and use of all data were

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approved by the Baylor College of Medicine's Institutional Review Board (IRB).

Rates of Diagnostic and statistical manual of mental disorders, 4th edition (DSM-IV) PDs were as follows: borderline personality disorder (19%), avoidant personality disorder (16%), obsessive-compulsive personality disorder (9%), antisocial personality disorder (3%), narcissistic personality disorder (2%), and schizotypal personality disorder (0.4%). Note that histrionic, schizoid, dependent and paranoid PDs showed prevalence rates of <0.01% in our pilot samples ($N = 1200$), so we limited their assessment to ensure a complete assessment of the remaining PDs. This is also consistent with the main PD types included in the DSM-5 Section III (American Psychiatric Association, 2013). Of the 31% of patients meeting the criteria for any PD, 34% met the criteria for at least one other PD.

Measures

Personality disorder symptoms were assessed within 72 h of admission using the Structured Clinical Interview II for DSM-IV Personality Disorders Screening Questionnaire (SCID-II-PSQ; First, Spitzer, Gibbon, Williams, and Benjamin, 1994). Seven-to-nine symptoms for antisocial, avoidant, borderline, narcissistic, obsessive-compulsive, and schizotypal personality disorders were rated by patients with a 'yes' (threshold or true) or 'no' (subthreshold, false or absent). Internal consistency was acceptable or near acceptable for most disorders ($\alpha_{narcissistic} = 0.66$, $\alpha_{avoidant} = 0.74$, $\alpha_{borderline} = 0.75$, $\alpha_{antisocial} = 0.86$), except for two ($\alpha_{obsessive} = 0.56$, $\alpha_{schizotypal} = 0.51$). We analysed the antisocial behaviour items after the age of 15; a diagnosis of conduct disorder was not required.

Depression symptoms were assessed at admission and every fortnight until discharge with the PHQ-9 (Kroenke, Spitzer, & Williams, 2001). The PHQ-9 is a screening questionnaire based on the DSM-IV criteria for major depressive disorder. Patients rated the frequency of depressive symptoms over the past fortnight on a Likert scale ranging from 0 (not at all) to 3 (nearly every day). Responses were then summed to form total depression scores. The PHQ-9 shows excellent criterion validity, with sensitivity and specificity rates for detecting depression of 88% or more (Kroenke, Spitzer, Williams, & Lowe, 2010; Manea, Gilbody, & McMillan, 2015). The internal consistency in our sample averaged across each assessment period was excellent ($\alpha = 0.90$; range = 0.89–0.91).

Intervention

Patients were admitted to one of four inpatient programs: Compass (31%) for young adults (18–24); Comprehensive Psychiatric Assessment Service (CPAS; 18%) for adults in crisis; Hope (27%) for adults with more chronic difficulties; and Professionals in Crisis (PIC; 24%) for professionals with long-standing disorders. All programs were multimodal and equally intensive, consisting of individual and group psychotherapy, psychoeducation, social and recreational activities, family work, psychopharmacology and medication management, general psychiatric and medical care, and continuous nursing care. Patients were treated by multidisciplinary teams composed of psychiatrists, psychologists, social workers, psychiatric nurses, and rehabilitation specialists. Patients stayed for 6 weeks on average (S.D. = 3 weeks).

Data analysis

Confirmatory factor analysis

Our bifactor model included a general factor with loadings from all PD items, as well as six orthogonal specific factors each with loadings from a single PD. The general and specific factors were uncorrelated. We compared the bifactor model to a single-factor model, which included a single factor with loadings from all PD items, and correlated factors model, which included six correlated factors each representing a PD diagnosis with no cross-loadings.

Models were estimated using the robust maximum-likelihood estimator and compared using information criteria that penalize for model complexity based on the number of freely estimated parameters. Models were also estimated with robust weighted least squares to assess their global fit, and factor reliabilities were evaluated with model-based reliability indices (Dueber, 2017). Further details can be found in online Supplementary S1. Confirmatory factor analyses were run in Mplus 8.0 (Muthén & Muthén, 2017).

Latent growth model

We used latent growth curve models to estimate the PHQ-9 symptom trajectories over the 2–8-week course of inpatient treatment. We compared three models: an unconditional model with growth factors only, a part conditional model with growth factors, bifactor PD factors, and clinical covariates, and a full conditional model with growth factors, bifactor PD factors, clinical covariates, and demographic covariates. We also re-ran these models using the correlated PD factors (see online Supplementary S2). In all models, PHQ-9 scores at admission were included as a covariate to control for baseline differences in severity other than those attributed to general PD, as well as the spurious effects of repeated measures e.g. regression to the mean (Chou, Chi, Weisner, Pentz, & Hser, 2010).

In the unconditional growth model, we estimated an intercept factor with loadings from observed PHQ-9 scores at weeks 2–8 fixed to one, and a linear slope factor with loadings from PHQ-9 scores at weeks 2–8 reflecting a linear increase in time (week 2 scores = 0, week 4 scores = 1, week 6 scores = 2, week 8 scores = 3). We then tested whether adding a quadratic slope factor, whose loadings reflected non-linear increments in time (e.g. week 2 = 0, week 4 = 1, week 6 = 4, week 8 = 9), improved the model fit using information criteria. The model included growth factor variances that reflect heterogeneity in the intercept and slopes. The intercept and slope growth factors were freely correlated.

In the part conditional growth model with PD factors and clinical covariates, the best-fitting growth factors from the unconditional model were regressed onto the general and specific PD factors (antisocial, avoidant, borderline, narcissistic, obsessive, and schizotypal). Growth curves and PD factors were estimated within the same structural equation model. Growth factors were also regressed onto the clinical covariates, including PHQ-9 scores at admission, length of inpatient stay, number of prior admissions (first admission *v.* one or more prior admissions), and inpatient program (HOPE *v.* Compass; CPAS *v.* Compass; PIC *v.* Compass). All covariates were centred.

In the full conditional model with PD factors, clinical covariates, and demographic covariates, the growth factors were regressed onto the general and specific PD factors, clinical covariates, and demographic variables, including age at admission, sex, ethnicity (White/Caucasian *v.* all other ethnic groups), the highest level of education obtained (up to some college *v.* bachelor's

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degree or beyond), and marital status (married *v.* not married/separated). All covariates were centred.

Growth models were run in Mplus 8.0 using the MLR estimator (Muthén & Muthén, 2017). Missing data were mainly a function of the length of inpatient stay (e.g. those who were discharged before the 8-week period showed missing responses up to that point). Given that we could explain the cause of missingness, we assumed that missing responses were Missing at Random and were handled with full-information maximum likelihood. Length of inpatient stay was included as a covariate in all models.

Results

Confirmatory bifactor analysis

Full details of the bifactor analysis, including model fit indices, factor loadings, and model comparisons, can be found in online Supplementary S1 and Tables S1–S2. Briefly, the bifactor model showed a good fit that outperformed the correlated factor and single-factor models. The general factor showed healthy loadings and was well represented by its indicators. There was some variation in specific factor reliability: avoidant and borderline PD items loaded more strongly onto the general PD factor than the specific avoidant and borderline factors, respectively, reducing their reliability, whereas antisocial and narcissistic PD items overlapped least with the general variance and hence represented the antisocial and narcissistic factors well.

Latent growth models

An unconditional growth model with an intercept and linear slope factor showed a good-to-excellent fit (CFI = 0.96, TLI = 0.95, RMSEA = 0.07, SRMR = 0.02). Adding a quadratic slope factor improved the information explained (ΔAIC = 68; ΔBIC = 45; ΔaBIC = 58; model fit: CFI = 1, TLI = 1, RMSEA = 0, SRMR = 0). We report unstandardized coefficients for the intercept factor (η_0), slope factor (linear = η_1, quadratic = η_2), latent variance components (ζ_0, ζ_1, ζ_2), and regression weights (b_0, b_1, b_2). The intercept (e.g. the estimated mean PHQ-9 score at week 2) in the unconditional model with both linear and quadratic slope factors fell just under the PHQ-9's clinical threshold for major depression [$\eta_0 = 9.56$, $z = 66.39$, $p < 0.001$, 95% CI (9.27–9.84)], but patients varied substantially around the mean [$\zeta_0 = 37.56$, $z = 13.13$, $p < 0.001$, 95% CI (31.94–43.16)]. On average, patients showed a linear decline in PHQ-9 scores over the treatment period [$\eta_1 = -2.40$, $z = -16.87$, $p < 0.001$, 95% CI (−2.68 to −2.12)], but varied in the steepness of their individual slopes [$\zeta_1 = 13.76$, $z = 3.81$, $p < 0.001$, 95% CI (6.67–20.83)]. The rate of decline in PHQ-9 scores slowed with time [$\eta_2 = 0.34$, $z = 6.81$, $p < 0.001$, 95% CI (0.24–0.43)], but patients varied in the extent of this quadratic pattern of change [$\zeta_2 = 1.09$, $z = 3.28$, $p < 0.001$, 95% CI (0.44–1.74)].

In the part conditional growth model with the general and uncorrelated specific PD factors and clinical covariates, the intercept [$\eta_0 = 9.31$, $z = 82.29$, $p < 0.001$, 95% CI (9.09–9.53)], linear slope [$\eta_1 = -2.41$, $z = -11.90$, $p < 0.001$, 95% CI (−2.81 to −2.02)], and quadratic slope [$\eta_2 = 0.30$, $z = 3.60$, $p < 0.001$, 95% CI (0.14–0.46)] were similar to the unconditional model. Higher general PD factor scores predicted higher intercept values [$b_0 = 1.16$, $z = 6.49$, $p < 0.001$, 95% CI (0.81–1.51)], while lower intercept values were predicted by marginally higher borderline scores [$b_0 = -0.49$, $z = -1.86$, $p = 0.062$, 95% CI (−1.00 to 0.03)], higher antisocial scores [$b_0 = -0.55$, $z = -2.14$, $p = 0.032$, 95% CI (−1.05 to −0.05)], and higher narcissistic scores [$b_0 = -0.38$, $z = -1.96$, $p = 0.050$, 95% CI (−0.77 to 0)]. The general PD factor did not predict significant differences in the steepness of the linear slopes [$b_1 = -0.09$, $z = -0.42$, $p = 0.678$, 95% CI (−0.53 to 0.35)]. By contrast, higher borderline scores predicted flatter linear slopes [$b_1 = 0.58$, $z = 1.97$, $p = 0.049$, 95% CI (0.01–1.16)], while higher antisocial scores predicted a stronger quadratic (i.e. *U* shaped) pattern of growth [$b_2 = 0.25$, $z = 2.26$, $p = 0.024$, 95% CI (0.03–0.46)]. Regression coefficients for the clinical covariates were similar to those in the full conditional growth model (see below).

In the full conditional growth model with bifactor PD factors, clinical covariates, and demographic covariates, the growth factors were almost identical to the part conditional growth model (see online Supplementary Table S1). Higher intercept values were again predicted by higher general PD scores [$b_0 = 1.14$, $z = 6.36$, $p < 0.001$, 95% CI (0.79–1.49)], lower borderline scores [$b_0 = -0.64$, $z = -2.47$, $p = 0.013$, 95% CI (−1.14 to −0.13)), and lower antisocial scores [$b_0 = -0.51$, $z = -1.99$, $p = 0.047$, 95% CI (−1.02 to −0.01)]. The association between general PD and the linear slope strengthened but did not reach significance [$b_1 = -0.22$, $z = -0.96$, $p = 0.340$, 95% CI (−0.66 to 0.23)]. Moreover, the association between borderline scores and linear slopes decreased slightly but was now marginal [$b_1 = 0.52$, $z = 1.75$, $p = 0.08$, 95% CI (−0.06 to 1.11)], while the association between antisocial scores and quadratic slopes increased slightly and remained significant [$b_2 = 0.26$, $z = 2.36$, $p = 0.018$, 95% CI (0.04–0.47)]. Figure 1 shows the growth curves predicted by the general, borderline, and antisocial factors, and online Supplementary Table S3 shows the regression coefficients for the remaining PD factors, clinical covariates, and demographic covariates.

Discussion

Research findings are mixed as to whether PDs predict poorer outcomes following treatment for depression. One problem is that current assessment measures conflate what is shared among PDs (i.e. severity) with what is specific to particular PDs (i.e. style; Hopwood et al., 2011). These two sources of variance might predict depression outcomes in opposite directions, which could contribute to the mixed findings. We investigated the unique contributions of the general and specific components of personality pathology to depression prognosis by first separating out these two sources of variance with the bifactor model, and then using the resultant general and specific PD factors to predict changes in depression severity over an inpatient treatment using latent growth models.

Consistent with past studies, we found that the covariation in PD symptom reports was best summarized by a general PD factor, as well as specific factors reflecting each PD assessed (Conway et al., 2016; Jahng et al., 2011; Sharp et al., 2015[1]; Williams et al., 2018; Wright et al., 2016). Furthermore, borderline items (e.g. 'identity disturbance', 'empty'), avoidant items (e.g. 'preoccupied with rejection', 'views self as inept'), and schizotypal items ('ideas of reference', 'social anxiety') loaded most strongly onto the general PD factor[2] (Conway et al., 2016; Sharp et al., 2015; Williams et al., 2018; Wright et al., 2016), supporting the idea that general PD reflects overall dysfunction in self-functioning (e.g. an incoherent or inadequate sense of identity) and interpersonal functioning (e.g. a general insecurity or mistrust of others). The general PD factor predicted higher initial depression scores,

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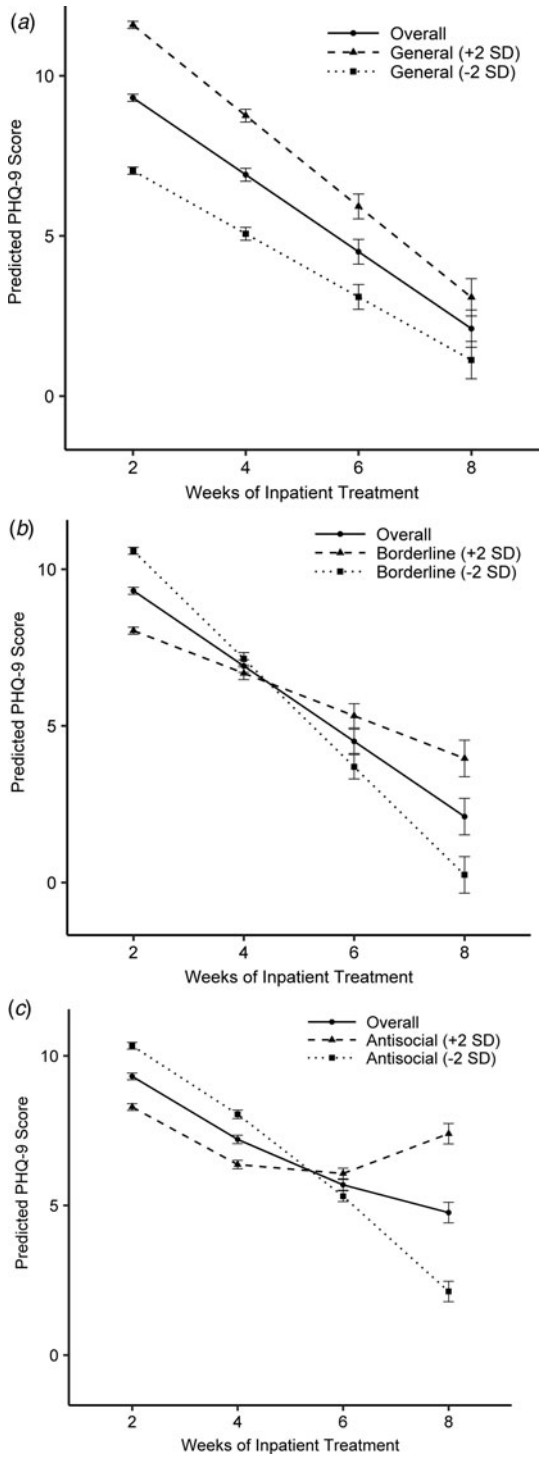


Fig. 1. (*a*) The linear slope factor for general personality disorder scores ± 2 standard deviations (s.d.) from the mean; (*b*) The linear slope factor for specific borderline factor scores ± 2 s.d. from the mean; (*c*) The quadratic slope factor for specific antisocial factor scores ± 2 s.d. from the mean. The 'Overall' slope in each sub-figure reflects the linear or quadratic slope holding the general and specific factors constant. All growth factors are conditional on centred clinical and demographic covariates. Error bars reflect standard errors of the predicted means.

but not differential rates of change. By contrast, the specific borderline factor predicted slower rates of decline over the treatment period, while the antisocial factor predicted a *U* shaped pattern of change.

Prognostic value of the general and specific PD factors

Higher general PD scores predicted higher initial depression scores 2 weeks into an 8-week inpatient treatment but did not predict significant differences in the growth curves. In other words, the common variance among PD symptoms predicted more severe depression but similar rates of change. This suggests that the general PD factor captures overall illness severity, which is not in itself a strong prognostic predictor. Prior studies have also reported higher depression scores in the presence of a PD (Fowler et al., 2018; Moradveisi et al., 2013), which normalizes after baseline depression severity – a marker of overall illness severity – is controlled for (De Bolle et al., 2011; Erkens et al., 2018; van Bronswijk et al., 2018). If general severity is not controlled for, the associated rise in depression scores may be misinterpreted as the negative effect of PDs on depression outcomes. But if it is, PDs will be said to have no prognostic value for depression outcomes. The mixed findings regarding the prognostic value of PDs on depression outcomes might be explained largely by the extent to which the effect of general severity is controlled for (Mulder, 2002).

Higher specific borderline factor scores were associated with lower initial depression scores and flatter linear slopes. That is, once the effect of general PD and the other specific PD factors was controlled for, borderline features (i.e. Negative Affectivity; see online Supplementary S1) predicted slower changes throughout an inpatient treatment. This is particularly interesting given that another study using an overlapping dataset reported that a BPD diagnosis, while associated with higher initial depression scores, was not associated with different rates of change (Fowler et al., 2018). If anything, patients with a BPD diagnosis showed better absolute outcomes, in that their depression scores dropped a larger amount to reach a similar endpoint to those without a BPD diagnosis. Our study suggests that the increased baseline severity associated with BPD was in fact a function of general PD severity. Only once the common variance in PD ratings is separated from the specific variance do we find that stylistic borderline features are associated with poorer depression outcomes.

How should we interpret the specific effect of borderline features on depression outcomes, if the general PD factor also reflects characteristics associated with borderline difficulties (Clark, Nuzum, & Ro, 2018)? One idea is that the general PD factor represents the non-specific ways in which disturbances in self and interpersonal functioning manifest across PDs, while the specific borderline factor reflects personality tendencies that explicitly feature these themes, such as a fragile (or malleable) identity and interpersonal sensitivity. When these personality tendencies interfere with one's life, they may cause a difficulty in trusting the personal relevance of socially communicated information that challenges their rigid and impairing beliefs about the self, other, and world – like the information presented in treatment (Fonagy, Luyten, Allison, & Campbell, 2017).

Alternatively, the association between the specific borderline factor and poorer prognosis may be a by-product of controlling for general PD, which reduced the initial depression scores and hence steepness of the slope. However, those with higher borderline factor scores were predicted to have higher end-point depression scores than those with low borderline factor scores, suggesting that the flatter slope is not purely a function of removing the baseline severity effect. Still, we caution any definitive interpretation of these findings given that the specific borderline factor's reliability was relatively weak, and the significance of its

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prediction did not survive correction for demographic variables at a 5% alpha level.

Higher specific antisocial factor scores in the bifactor growth model were associated with lower initial depression scores and stronger quadratic (i.e. *U* shaped) slopes. That is, once the effect of general PD and the other specific PD factors was controlled for, antisocial features (i.e. Disinhibition; see online Supplementary S1) predicted an initial decline followed by an upward inflection in depression scores. Few have documented the prognostic value of ASPD on depression outcomes, but an early prospective study reported higher depression recurrence rates associated with ASPD (and BPD) compared to bipolar disorder (Perry, 1988). More generally, ASPD is associated with high rates of recidivism (Bonta, Blais, & Wilson, 2014). The specific mechanisms that predict recurrence in offending and depression severity are unlikely to be the same, but the broader mechanisms associated with antisocial features may contribute to both, such as disinhibition (Remster, 2013). Future studies that include measures of hypothesized treatments mechanisms are necessary to test these hypotheses.

Limitations

Using dichotomous criterion-counts to assess underlying PD dimensions lacks genuine dimensionality and may have artificially inflated the correlations among items, as they are designed to detect threshold levels of pathology at the cost of specificity. While this may question the substantive validity of the general PD factor, the specific borderline and antisocial factors are free from the general variance and hence common method effects. Still, our PD measure is limited to self-report ratings that do not capture the full nature of personality difficulties relative to a multi-informant approach (Carlson, Vazire, & Oltmanns, 2013). Furthermore, the PHQ-9 may be subject to self-report biases, insofar as patients diagnosed with a PD often rate their depression as more severe than do clinicians (Unger, Hoffmann, Kohler, Mackert, & Fydrich, 2013). Therefore, the slower rate of decline associated with borderline features and the *U* shaped pattern of change associated with antisocial features may be a function of stylistic patterns in *reporting* rather than behaving. Nonetheless, the two are unlikely to be distinct: negative response styles may in themselves reflect behavioural tendencies that confer risk to psychopathology (Lahey et al., 2012).

We did not sample the full range of personality disorders, particularly histrionic, schizoid, dependent, and paranoid PDs, due to their low occurrence. Therefore, we must be cautious in generalizing our findings as they might be limited to personality configurations found within a depression-seeking sample. However, the construct validity of these lower frequency PDs has been questioned due to their low rates of prevalence in the population and low symptom specificity (Skodol et al., 2011a). This is not to say that these PDs lack clinical utility; rather, they might be better thought of as capturing broader-level traits than specific PDs, as was proposed in the DSM-5 alternative model (American Psychiatric Association, 2013). Nonetheless, even the PDs sampled demonstrate a pattern of loadings consistent with the trait domains featured across the ICD-11 and DSM 5 alternative model of PD[3] (Bach et al., 2020; see online Supplementary S1). This highlights the densely hierarchical nature of PDs and we encourage researchers to assess multiple levels of functioning for a more comprehensive picture of personality disorder.

We have assumed that PDs are a primary feature of the clinical profile that shapes the course of depression (Tyrer, 2015). There is good evidence supporting this: PDs in adolescence significantly increase the risk of depression in adulthood (Johnson et al., 1999), and improvements in PD precede improvements in depression, but not the reverse (Gunderson et al., 2004). Nonetheless, the presence of both a PD and depression in adolescence often outweighs the predictive strength of either one alone (Crawford et al., 2008; Kasen, Cohen, Skodol, Johnson, & Brook, 1999). Hence, the relationship between PDs and depression may not be a simple, unidirectional one (Livesley, 2015).

Furthermore, while we assume that borderline and antisocial features are static predictors of depression prognosis, personality traits are context- and mood-dependent (Hopwood, Zimmermann, Pincus, & Krueger, 2015; Wright, Hopwood, & Simms, 2015). Our baseline assessment of PD is limited to a certain context (e.g. an acute illness state) and does not capture personality dynamics in terms of variation in how people interact with their environments. We advise future researchers to take repeated measurements of PDs to investigate their reciprocal relationships with other problems, to ultimately inform on the mechanisms of change.

Implications

Our findings suggest that personality disorder assessment should include both shared and specific aspects of PDs. There is clear overlap in PD symptoms that in part reflects semantic redundancy, but also the overall degree of life impairment that patients experience (Livesley, 2011). The specific characteristics associated with PDs should not, however, be dismissed (or focused on exclusively, as is currently the case). Rather, a patient's overall level of severity as well as their stylistic expressions should be assessed (Hopwood et al., 2011). This 'binomial nomenclature' of PDs is already featured in the 11th revision of the International Classification of Diseases, where PD diagnosis is based on a single dimension that reflects the severity of personality impairment (ranging from mild to severe dysfunction), as well as five trait-domains (e.g. negative affectivity, detachment, disinhibition, dissociality, and anankastia) that specify the ways in which this impairment is expressed (Tyrer et al., 2015). There is also a movement towards a binomial taxonomy in the DSM-5's alternative model of PDs (Skodol et al., 2011b). These two systems differ in several ways but share a common ground in representing the general (severity) and specific (stylistic) components of personality disorders (Bach, Sellbom, Skjernov, & Simonsen, 2018).

We note that while borderline (and antisocial) factors uniquely predicted depression outcomes, this does not support the inclusion of a borderline PD qualifier in the diagnostic system – which is a topic of much debate (Reed, 2018) – because these factors reflect features or perhaps trait dimensions (i.e. Negative Affectivity and Disinhibition; see online Supplementary S1) that vary across the sample, rather than a 'borderline' subgroup of patients. In fact, while a borderline qualifier is consistent with current practice, the current findings suggest that it may be somewhat redundant, given that (i) borderline items loaded preferentially onto the general PD factor, (ii) the remaining items that loaded onto the specific borderline factor reflect Negative Affectivity (see online Supplementary S1), and (iii) a 'borderline pattern' might be best captured by combinations of trait domains, such as high levels of Negative Affectivity and Disinhibition (Bach et al., 2020), the analogues of which (i.e. borderline and antisocial factors) predicted poorer outcomes in this study.

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Our findings also highlight the importance of studying the unique contributions of the general and specific aspects of PDs to depression outcomes. If these components are not separated out, then their potentially conflicting relationships may obscure prognostic predictions. We used the bifactor model to achieve this, which is not without controversy (Sellbom & Tellegen, 2019; van Bork, Epskamp, Rhemtulla, Borsboom, & van der Maas, 2017; Watts, Poore, & Waldman, 2019). Nonetheless, we hope to have demonstrated that with proper theoretical justification (e.g. conceptualizing personality dysfunction in terms of shared and stylistic features), model evaluation beyond standard fit indices (e.g. information criteria and model-based reliability indices), and external validation (e.g. predicting future depression outcomes and comparing predictions with alternative models), the bifactor model can be meaningfully applied to assessment research. Future studies should investigate how general and specific PD factors interact with different treatments in randomized controlled studies to better understand 'what works for whom' (for an example in the developmental psychopathology field, see Aitken et al., 2020).

We have shown that personality disorder symptoms are best described by a general factor that reflects the severity of individuals' personality dysfunction, as well as specific factors that reflect stylistic expressions associated with different disorders. Borderline and antisocial features are associated with poorer prognosis throughout inpatient treatment for depression, once the variance associated with a general personality disorder is controlled.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S003329172000361X

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Conflict of interest. The authors declare no conflicts of interest.

Notes

† The notes appear after the main text.

1 There is partial overlap between Sharp et al.'s (2015) sample and our own, but our analytic approaches differ. We used a confirmatory model to actively test the bifactor structure that Sharp et al. and others have reported using exploratory methods. Moreover, confirmatory models are more restrictive and less likely to overfit sample-specific variances than exploratory solutions (unless the model is markedly mis-specified).

2 While borderline PD items formed a specific factor in our study, others have shown that borderline items load to unity with the general factor (Sharp et al., 2015; Williams et al., 2018; Wright et al., 2016). The reason for this disparity is unclear and may be a product of different methodological features (e.g. exploratory *v.* confirmatory models).

3 We thank the reviewer who pointed this out.

References

Aitken, M., Haltigan, J. D., Szatmari, P., Dubicka, B., Fonagy, P., Kelvin, R., … Goodyer, I. M. (2020). Toward precision therapeutics: General and specific factors differentiate symptom change in depressed adolescents. *Journal of Child Psychology and Psychiatry*, *61*(9), 998–1008. doi: 10.1111/jcpp.13194.

Allen, J. G., Frueh, B. C., Ellis, T. E., Latini, D. M., Mahoney, J. S., Oldham, J. M., … Wallin, L. (2009). Integrating outcomes assessment and research into clinical care in inpatient adult psychiatric treatment. *Bulletin of the Menninger Clinic*, *73*(4), 259–295. doi: 10.1521/bumc.2009.73.4.259

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Bach, B., Kerber, A., Aluja, A., Bastiaens, T., Keeley, J. W., Claes, L., … Zimmermann, J. (2020). International assessment of DSM-5 and ICD-11 personality disorder traits: Toward a common nosology in DSM-5.1. *Psychopathology*, *53*(3-4), 179–188. doi: 10.1159/000507589.

Bach, B., Sellbom, M., Skjernov, M., & Simonsen, E. (2018). ICD-11 and DSM-5 personality trait domains capture categorical personality disorders: Finding a common ground. *Australian & New Zealand Journal of Psychiatry*, *52*(5), 425–434. doi: 10.1177/0004867417727867

Bonta, J., Blais, J., & Wilson, H. A. (2014). A theoretically informed meta-analysis of the risk for general and violent recidivism for mentally disordered offenders. *Aggression and Violent Behavior*, *19*(3), 278–287. doi: 10.1016/j.avb.2014.04.014

Carlson, E. N., Vazire, S., & Oltmanns, T. F. (2013). Self-other knowledge asymmetries in personality pathology. *Journal of Personality*, *81*(2), 155–170. doi: 10.1111/j.1467-6494.2012.00794.x

Chen, F. F., West, S. G., & Sousa, K. H. (2006). A comparison of bifactor and second-order models of quality of life. *Multivariate Behavioral Research*, *41*(2), 189–225. doi: 10.1207/s15327906mbr4102_5

Chou, C.-P., Chi, F., Weisner, C., Pentz, M., & Hser, Y.-I. (2010). Initial Status in growth curve modeling for randomized trials. *Journal of Drug Issues*, *40*(1), 155–172. doi: 10.1177/002204261004000109

Clark, L. A., Nuzum, H., & Ro, E. (2018). Manifestations of personality impairment severity: Comorbidity, course/prognosis, psychosocial dysfunction, and 'borderline' personality features. *Current Opinion in Psychology*, *21*, 117–121. doi: 10.1016/j.copsyc.2017.12.004

Clarkin, J. F., Petrini, M., & Diamond, D. (2019). Complex depression: The treatment of major depression and severe personality pathology. *Journal of Clinical Psychology*, *75*(5), 824–833. doi: 10.1002/jclp.22759

Conway, C. C., Hammen, C., & Brennan, P. A. (2016). Optimizing prediction of psychosocial and clinical outcomes with a transdiagnostic model of personality disorder. *Journal of Personality Disorders*, *30*(4), 545–566. doi: 10.1521/pedi_2015_29_218

Crawford, T. N., Cohen, P., First, M. B., Skodol, A. E., Johnson, J. G., & Kasen, S. (2008). Comorbid axis I and axis II disorders in early adolescence: Outcomes 20 years later. *Archives of General Psychiatry*, *65*(6), 641–648. doi: 10.1001/archpsyc.65.6.641

Cyranowski, J. M., Frank, E., Winter, E., Rucci, P., Novick, D., Pilkonis, P., … Kupfer, D. J. (2004). Personality pathology and outcome in recurrently depressed women over 2 years of maintenance interpersonal psychotherapy. *Psychological Medicine*, *34*(4), 659–669. doi: 10.1017/S0033291703001661

De Bolle, M., De Fruyt, F., Quilty, L. C., Rolland, J. P., Decuyper, M., & Bagby, R. M. (2011). Does personality disorder co-morbidity impact treatment outcome for patients with major depression? A multi-level analysis. *Journal of Personality Disorders*, *25*(1), 1–15. doi: 10.1521/pedi.2011.25.1.1

Dueber, D. M. (2017). Bifactor Indices Calculator: A Microsoft Excel-based tool to calculate various indices relevant to bifactor CFA models. https://doi.org/10.13023/edp.tool.01

Erkens, N., Schramm, E., Kriston, L., Hautzinger, M., Harter, M., Schweiger, U., & Klein, J. P. (2018). Association of comorbid personality disorders with clinical characteristics and outcome in a randomized controlled trial comparing two psychotherapies for early-onset persistent depressive disorder. *Journal of Affective Disorders*, *229*, 262–268. doi: 10.1016/j.jad.2017.12.091

First, M., Spitzer, R., Gibbon, M., Williams, J., & Benjamin, L. (1994). *Structured clinical interview for DSM–IV axis II personality disorders* (*SCID II*). New York, NY: Biometric Research Department.

Fonagy, P., Luyten, P., Allison, E., & Campbell, C. (2017). What we have changed our minds about: Part 2. Borderline personality disorder, epistemic trust and the developmental significance of social communication. *Borderline Personal Disorder and Emotion Dysregulation*, *4*, 9. doi: 10.1186/s40479-017-0062-8

Fowler, J. C., Clapp, J. D., Madan, A., Allen, J. G., Frueh, B. C., Fonagy, P., & Oldham, J. M. (2018). A naturalistic longitudinal study of extended inpatient treatment for adults with borderline personality disorder: An examination of treatment response, remission and deterioration. *Journal of Affective Disorders*, *235*, 323–331. doi: 10.1016/j.jad.2017.12.054

https://doi.org/10.1017/S003329172000361X Published online by Cambridge University Press

French, L. R. M., Turner, K. M., Dawson, S., & Moran, P. (2017). Psychological treatment of depression and anxiety in patients with co-morbid personality disorder: A scoping study of trial evidence. *Personality and Mental Health*, *11*(2), 101–117. doi: 10.1002/pmh.1372

Friborg, O., Martinsen, E. W., Martinussen, M., Kaiser, S., Overgard, K. T., & Rosenvinge, J. H. (2014). Comorbidity of personality disorders in mood disorders: A meta-analytic review of 122 studies from 1988 to 2010. *Journal of Affective Disorders*, *152-154*, 1–11. doi: 10.1016/j.jad.2013.08.023

Grilo, C. M., Stout, R. L., Markowitz, J. C., Sanislow, C. A., Ansell, E. B., Skodol, A. E., … McGlashan, T. H. (2010). Personality disorders predict relapse after remission from an episode of major depressive disorder: A 6-year prospective study. *The Journal of Clinical Psychiatry*, *71*(12), 1629–1635. doi: 10.4088/JCP.08m04200gre

Gunderson, J. G., Morey, L. C., Stout, R. L., Skodol, A. E., Shea, M. T., McGlashan, T. H., … Bender, D. S. (2004). Major depressive disorder and borderline personality disorder revisited. *The Journal of Clinical Psychiatry*, *65*(8), 1049–1056. doi: 10.4088/JCP.v65n0804

Hopwood, C. J., Malone, J. C., Ansell, E. B., Sanislow, C. A., Grilo, C. M., McGlashan, T. H., … Morey, L. C. (2011). Personality assessment in DSM-5: Empirical support for rating severity, style, and traits. *Journal of Personality Disorders*, *25*(3), 305–320. doi: 10.1521/pedi.2011.25.3.305

Hopwood, C. J., Zimmermann, J., Pincus, A. L., & Krueger, R. F. (2015). Connecting personality structure and dynamics: Towards a more evidence-based and clinically useful diagnostic scheme. *Journal of Personality Disorders*, *29*(4), 431–448. doi: 10.1521/pedi.2015.29.4.431

Jahng, S., Trull, T. J., Wood, P. K., Tragesser, S. L., Tomko, R., Grant, J. D., … Sher, K. J. (2011). Distinguishing general and specific personality disorder features and implications for substance dependence comorbidity. *Journal of Abnormal Psychology*, *120*(3), 656–669. doi: 10.1037/a0023539

Johnson, J. G., Cohen, P., Skodol, A. E., Oldham, J. M., Kasen, S., & Brook, J. S. (1999). Personality disorders in adolescence and risk of major mental disorders and suicidality during adulthood. *Archives of General Psychiatry*, *56*(9), 805. doi: 10.1001/archpsyc.56.9.805

Kasen, S., Cohen, P., Skodol, A. E., Johnson, J. G., & Brook, J. S. (1999). Influence of child and adolescent psychiatric disorders on young adult personality disorder. *American Journal of Psychiatry*, *156*(10), 1529–1535. doi: 10.1176/ajp.156.10.1529

Kool, S., Schoevers, R., de Maat, S., Van, R., Molenaar, P., Vink, A., & Dekker, J. (2005). Efficacy of pharmacotherapy in depressed patients with and without personality disorders: A systematic review and meta-analysis. *Journal of Affective Disorders*, *88*(3), 269–278. doi: 10.1016/j.jad.2005.05.017

Kroenke, K., Spitzer, R. L., & Williams, J. B. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, *16*(9), 606–613. doi: 10.1046/j.1525-1497.2001.016009606.x

Kroenke, K., Spitzer, R. L., Williams, J. B., & Lowe, B. (2010). The patient health questionnaire somatic, anxiety, and depressive symptom scales: A systematic review. *General Hospital Psychiatry*, *32*(4), 345–359. doi: 10.1016/j.genhosppsych.2010.03.006

Lahey, B. B., Applegate, B., Hakes, J. K., Zald, D. H., Hariri, A. R., & Rathouz, P. J. (2012). Is there a general factor of prevalent psychopathology during adulthood? *Journal of Abnormal Psychology*, *121*(4), 971–977. doi: 10.1037/a0028355

Livesley, W. J. (2011). An empirically-based classification of personality disorder. *Journal of Personality Disorders*, *25*(3), 397–420. doi: 10.1521/pedi.2011.25.3.397

Livesley, W. J. (2015). A hypothesis too far? Commentary on personality dysfunction as the cause of recurrent non-cognitive mental disorder. *Personality and Mental Health*, *9*(1), 14–16. doi: 10.1002/pmh.1285

Manea, L., Gilbody, S., & McMillan, D. (2015). A diagnostic meta-analysis of the Patient Health Questionnaire-9 (PHQ-9) algorithm scoring method as a screen for depression. *General Hospital Psychiatry*, *37*(1), 67–75. doi: 10.1016/j.genhosppsych.2014.09.009

Markon, K. E. (2019). Bifactor and hierarchical models: Specification, inference, and interpretation. *Annual Reviews of Clinical Psychology*, *15*, 51–69. doi: 10.1146/annurev-clinpsy-050718-095522

Moradveisi, L., Huibers, M. J., Renner, F., Arasteh, M., & Arntz, A. (2013). The influence of comorbid personality disorder on the effects of behavioural activation vs. Antidepressant medication for major depressive disorder: Results from a randomized trial in Iran. *Behaviour Research and Therapy*, *51*(8), 499–506. doi: 10.1016/j.brat.2013.05.006

Mulder, R. T. (2002). Personality pathology and treatment outcome in major depression: A review. *American Journal of Psychiatry*, *159*(3), 359–371. doi: 10.1176/appi.ajp.159.3.359

Muthén, L. K., & Muthén, B. O. (2017). *Mplus user's guide* (8th ed.). Los Angeles, California: Muthén & Muthén.

Newton-Howes, G., Tyrer, P., & Johnson, T. (2006). Personality disorder and the outcome of depression: Meta-analysis of published studies. *British Journal of Psychiatry*, *188*, 13–20. doi: 10.1192/bjp.188.1.13

Newton-Howes, G., Tyrer, P., Johnson, T., Mulder, R., Kool, S., Dekker, J., & Schoevers, R. (2014). Influence of personality on the outcome of treatment in depression: Systematic review and meta-analysis. *Journal of Personality Disorders*, *28*(4), 577–593. doi: 10.1521/pedi_2013_27_070

Perry, J. C. (1988). A prospective study of life stress, defenses, psychotic symptoms, and depression in borderline and antisocial personality disorders and bipolar type II affective disorder. *Journal of Personality Disorders*, *2*(1), 49–59. doi: 10.1521/pedi.1988.2.1.49

Reed, G. M. (2018). Progress in developing a classification of personality disorders for ICD-11. *World Psychiatry*, *17*(2), 227–229. doi: 10.1002/wps.20533

Reich, J. (2003). The effect of axis II disorders on the outcome of treatment of anxiety and unipolar depressive disorders: A review. *Journal of Personality Disorders*, *17*(5), 387–405. doi: 10.1521/pedi.17.5.387.22972

Reise, S. P. (2012). Invited paper: The rediscovery of bifactor measurement models. *Multivariate Behavioral Research*, *47*(5), 667–696. doi: 10.1080/00273171.2012.715555

Remster, B. (2013). Self-Control and the depression–delinquency link. *Deviant Behavior*, *35*(1), 66–84. doi: 10.1080/01639625.2013.822226

Sellbom, M., & Tellegen, A. (2019). Factor analysis in psychological assessment research: Common pitfalls and recommendations. *Psychological Assessment*, *31*(12), 1428–1441. doi: 10.1037/pas0000623

Sharp, C., Wright, A. G., Fowler, J. C., Frueh, B. C., Allen, J. G., Oldham, J., & Clark, L. A. (2015). The structure of personality pathology: Both general ('g') and specific ('s') factors? *Journal of Abnormal Psychology*, *124*(2), 387–398. doi: 10.1037/abn0000033

Skodol, A. E., Bender, D. S., Morey, L. C., Clark, L. A., Oldham, J. M., Alarcon, R. D., … Siever, L. J. (2011a). Personality Disorder Types Proposed for DSM-5. *Journal of Personality Disorders*, *25*(2), 136–169. doi: 10.1521/pedi.2011.25.2.136

Skodol, A. E., Clark, L. A., Bender, D. S., Krueger, R. F., Morey, L. C., Verheul, R., … Oldham, J. M. (2011b). Proposed changes in personality and personality disorder assessment and diagnosis for DSM-5 part I: Description and rationale. *Personality Disorders*, *2*(1), 4–22. doi: 10.1037/a0021891

Tyrer, P. (2015). Personality dysfunction is the cause of recurrent non-cognitive mental disorder: A testable hypothesis. *Personality and Mental Health*, *9*(1), 1–7. doi: 10.1002/pmh.1255

Tyrer, P., Crawford, M., Mulder, R., Blashfield, R., Farnam, A., Fossati, A., … Reed, G. M. (2011). The rationale for the reclassification of personality disorder in the 11th revision of the International Classification of Diseases (ICD-11). *Personality and Mental Health*, *5*(4), 246–259. doi: 10.1002/pmh.190

Tyrer, P., Reed, G. M., & Crawford, M. J. (2015). Classification, assessment, prevalence, and effect of personality disorder. *The Lancet*, *385*(9969), 717–726. doi: 10.1016/s0140-6736(14)61995-4

Unger, T., Hoffmann, S., Kohler, S., Mackert, A., & Fydrich, T. (2013). Personality disorders and outcome of inpatient treatment for depression: A 1-year prospective follow-up study. *Journal of Personality Disorders*, *27*(5), 636–651. doi: 10.1521/pedi_2012_26_052

van Bork, R., Epskamp, S., Rhemtulla, M., Borsboom, D., & van der Maas, H. L. J. (2017). What is the *p*-factor of psychopathology? Some risks of general factor modeling. *Theory & Psychology*, *27*(6), 759–773. doi: 10.1177/0959354317737185

van Bronswijk, S. C., Lemmens, L., Viechtbauer, W., Huibers, M. J. H., Arntz, A., & Peeters, F. (2018). The impact of personality disorder pathology on

https://doi.org/10.1017/S003329172000361X Published online by Cambridge University Press

the effectiveness of cognitive therapy and interpersonal psychotherapy for major depressive disorder. *Journal of Affective Disorders*, *225*, 530–538. doi: 10.1016/j.jad.2017.08.043

Watts, A. L., Poore, H. E., & Waldman, I. D. (2019). Riskier tests of the validity of the bifactor model of psychopathology. *Clinical Psychological Science*, *7* (6), 1285–1303. doi: 10.1177/2167702619855035.

Williams, T. F., Scalco, M. D., & Simms, L. J. (2018). The construct validity of general and specific dimensions of personality pathology. *Psychological Medicine*, *48*(5), 834–848. doi: 10.1017/S0033291717002227

World Health Organization. (2017). *Depression and other common mental disorders: Global health estimates* (*WHO/MSD/MER/2017.2*). Geneva: World Health Organization.

Wright, A. G. C., Hopwood, C. J., & Simms, L. J. (2015). Daily interpersonal and affective dynamics in personality disorder. *Journal of Personality Disorders*, *29*(4), 503–525. doi: 10.1521/pedi.2015.29.4.503

Wright, A. G., Hopwood, C. J., Skodol, A. E., & Morey, L. C. (2016). Longitudinal validation of general and specific structural features of personality pathology. *Journal of Abnormal Psychology*, *125*(8), 1120–1134. doi: 10.1037/abn0000165

https://doi.org/10.1017/S003329172000361X Published online by Cambridge University Press