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BRIEF REPORT

Borderline Personality Disorder Traits Associate With Midlife Cardiometabolic Risk

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There is growing interest in relationships between borderline personality disorder (BPD) pathology and physical health outcomes. Diagnostic BPD and BPD-related traits, for instance, have been shown to associate with self-reported cardiovascular disease and various cardiometabolic risk factors. However, potential confounding of these associations by comorbid depression, which itself contributes to risk for heart disease, remains unresolved, and previous research is limited by nearly uniform reliance on self-reported health status. In the present study, we examine the association of BPD traits and contemporaneously assessed depressive mood with instrumented measures of cardiometabolic risk in a midlife community sample ($N = 1,295$). BPD pathology was measured using dimensional, multi-informant trait measures; depressive symptomology was self-reported; and cardiometabolic risk was indexed via multiple indicators of insulin resistance, adiposity, dyslipidemia, and blood pressure. Structural equation modeling was used to estimate the effects of BPD traits and depressive symptoms on aggregated cardiometabolic risk, adjusting for their shared variance. Results showed both BPD features and depressive symptomatology related to the extent of cardiometabolic risk; when examined simultaneously, only BPD associated independently with risk indicators. In further supporting a link between BPD pathology and cardiovascular disease risk, these findings warrant future work to elucidate intervening behavioral and biological mechanisms.

Keywords: borderline personality disorder, personality pathology, cardiometabolic risk, cardiovascular disease, depression

Although borderline personality disorder (BPD) is well-studied for its relation to psychosocial impairments, recent research suggests this same pathology may also contribute to physical health risks (Dixon-Gordon, Whalen, Layden, & Chapman, 2015; Quirk et al., 2016; Tomko, Trull, Wood, & Sher, 2014). For instance, diagnostic BPD or BPD-related traits have been found associated with both self-reported cardiovascular disease (El-Gabalawy, Katz, & Sareen, 2010; Lee et al., 2010; Moran et al., 2007; Powers & Oltmanns, 2013) and various heart disease risk factors, such as obesity (Frankenburg & Zanarini, 2006; Greggersen, Rudolf, Brandt, et al., 2011; Greggersen, Rudolf, Fassbinder, et al., 2011; Powers & Oltmanns, 2013; Sansone, Wiederman, & Monteith, 2001), hypertension (El-Gabalawy et al., 2010; Frankenburg & Zanarini, 2006), fasting insulin or diabetes (El-Gabalawy et al., 2010; Greggersen, Rudolf, Brandt, et al., 2011), and preclinical atherosclerosis (carotid artery thickening; Greggersen, Rudolf, Brandt, et al., 2011). In addition to studying risk factors individually, the metabolic syndrome—a composite measure of abnormalities in lipid metabolism, glycemic control, central adiposity, and blood pressure—is commonly used to represent cardiovascular risk and predict incident disease. Using this index, one additional study found that prevalence of the metabolic syndrome among BPD patients from an inpatient psychiatric sample exceeded that of the general population (Kahl et al., 2013). These investigators also reported that BPD patients with the metabolic syndrome were more likely than those without to have co-occurring depression and dysthymia, but did not address whether this comorbidity fully confounded the association between BPD and metabolic syndrome.

The confound of comorbid depression is particularly relevant because depressive symptoms are known to confer risk for cardiovascular disease and metabolic syndrome (Pan, Sun, Okereke, Rexrode, & Hu, 2011; Rugulies, 2002; Vancampfort et al., 2014).

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It is possible that associations with depression and BPD are due to a more general, shared pathway, such as trait neuroticism or emotion dysregulation, which have established associations with cardiovascular risk factors (Bleil, Gianaros, Jennings, Flory, & Manuck et al., 2008; Gianaros et al., 2014; Kinnunen, Kokkonen, Kaprio, & Pulkkinen, 2005). Behaviorally, both forms of psychopathology involve negative affectivity and problematic health behaviors (e.g., poor diet, reduced physical activity, and substance use; Bonnet et al., 2005; Strine et al., 2008; Trull et al., 2018) that contribute to risk for incident cardiovascular disease (Patnode, Evans, Senger, Redmond, & Lin, 2017). Biological explanations for the association between depression and cardiovascular disease risk may also extend to BPD, including the potential role of systemic inflammation (Marsland, McCaffery, Muldoon, & Manuck, 2010; Schiepers, Wichers, & Maes, 2005), perturbed adrenocortical functioning (Anagnostis, Athyros, Tziomalos, Karagiannis, & Mikhailidis, 2009), and altered cardiac autonomic control (Carnethon et al., 2006; Kop, Synowski, Gottlieb, 2011). However, unlike depression, BPD is further defined by hostility, impulsivity, and interpersonal problems (American Psychiatric Association, 2013) that are themselves associated with cardiovascular outcomes (Bogg & Roberts, 2004; Valtorta, Kanaan, Gilbody, Ronzi, & Hanratty, 2016) and could confer risk independent of covarying depression or common vulnerability factor.

At present, the unique effects of BPD features are unclear. Among the few studies that controlled for mood disorders, only one measured concurrent symptomatology (Sansone et al., 2001) and the remainder relied on self-reported lifetime occurrence. Of these, four reported that associations between BPD measures and participant-reported history of cardiovascular disease or individual risk factors persisted after adjusting for lifetime depression (El-Gabalawy et al., 2010; Lee et al., 2010; Powers & Oltmanns, 2013; Sansone et al., 2001), whereas two studies failed to find a unique effect of BPD (Greggersen, Rudolf, Brandt, et al., 2011; Greggersen, Rudolf, Fassbinder, et al., 2011). Given this inconclusive literature and its nearly uniform reliance on self-reported health status, as opposed to measured cardiovascular risk, here we further examine BPD features in relation to aggregated cardiometabolic risk and contemporaneously assessed depressive mood. The study uses data from a large community sample of midlife adults on whom instrumented measures of component risk factors were available, along with a multi-informant, dimensional measure of BPD traits and reported depressive symptomatology.

Method

Data for this study were acquired from the University of Pittsburgh Adult Health and Behavior project, a registry of behavioral and biological measurements on non-Hispanic Caucasian and African American individuals (30–54 years old) recruited from 2001 to 2005 via mass-mail solicitation from communities of southwestern Pennsylvania in the United States (Manuck, Phillips, Gianaros, Flory, & Muldoon, 2010; Marsland et al., 2010). Exclusion criteria included history of atherosclerotic, cardiovascular, chronic kidney, or liver disease, past-year cancer treatment, neurologic disorders, and psychotic illness. Further exclusions included pregnancy and using insulin, nitrates, glucocorticoid, antiarrhythmic, psychotropic, or prescription weight-loss medications. Informed consent was obtained in accordance with approved protocol guidelines of the University of Pittsburgh Institutional Review Board. Data used for the present analyses were available for 1,295 participants, which is the final sample size for this study. The demographic breakdown is presented in Table 1.

BPD Assessment

A composite measure of scores on items of the NEO Five-Factor Inventory (Costa & McCrae, 1992) was used to measure BPD. These items selected to be prototypical of BPD have demonstrated sufficient convergent and discriminative validity to support their use for approximating its symptomology (Few et al., 2016). Participants and up to two informants (88% had two informants) rated the relevant personality items from the NEO Five-Factor Inventory. Dimensional BPD pathology scores were calculated from the relevant items as described in Few and colleagues (2016) for the self- and informant-reported items.

Depression Assessment

Depressive symptoms were self-reported using the Beck Depression Inventory (Beck, Steer, & Garbin, 1988). Subscale scores for Anhedonia, Depressive Cognitions, and Somatic Symptoms (Shafer, 2006) were used.

Table 1
Descriptive Statistics of Psychopathology and Biomedical Characteristics

Variable	*M* (*SD*) or percentage
Demographics	
Age (years)	44.6 (6.7)
Sex (male)	47.3
Race (Caucasian)	83.5
Years of education	15.7 (2.8)
Annual family income	
Less than $14,999	10.7
$15,000–$24,999	9.5
$25,000–$49,999	26.8
$50,000–$64,999	16.8
Above $65,000	35.5
Psychopathology and biomedical measures	
Borderline personality disorder traits	
Self	1.40 (1.18)
Informant X	1.41 (0.51)
Informant Y	1.34 (0.48)
Depression	
Somatic symptoms	0.16 (0.25)
Anhedonia	0.25 (0.29)
Depressive cognitions	0.18 (0.31)
Metabolic syndrome	
Systolic blood pressure (mm Hg)	116.44 (13.6)
Diastolic blood pressure (mm Hg)	78.31 (9.4)
Insulin (μU/ml)	13.42 (7.7)
Glucose (mg/dl)	96.45 (18.2)
Body mass index (kg/m^2)	27.46 (5.8)
Waist circumference (inches)	91.95 (15.9)
High-density lipoprotein (mg/dl)	53.52 (14.6)
Triglycerides (mg/dl)	120.48 (81.3)

Note. Borderline personality disorder traits measured using the NEO Five-Factor Inventory. Depression measured using the Beck Depression Inventory.

Cardiometabolic Assessment

Presence of the metabolic syndrome is commonly defined by a consensus count of the number of syndrome components whose values exceed thresholds deemed indicative of elevated disease risk by various professional bodies (e.g., American Heart Association, National Cholesterol Education Program, and International Diabetes Federation). As a binary measure, the metabolic syndrome has at least two notable limitations: (a) dichotomizing distributions of continuous variables such as blood pressure and fasting glucose loses much predictive information and (b) in younger, midlife populations, true risk may be underestimated when many individuals exhibit subthreshold elevations on multiple indicators (Dermody et al., 2016). Also, a quantitative index of cardiometabolic risk created from continuous distributions of the same variables better predicts incident disease than categorically defined metabolic syndrome (Agarwal et al., 2012), and confirmatory factor analyses (CFAs) using multiple risk variables consistently show a single second-order factor underlying four first-order factors (blood pressure, insulin resistance, weight/adiposity, and dyslipidemia; Marsland et al., 2010; Shen et al., 2003). Here, we express relative cardiometabolic risk (CMR) by derivation of this second-order latent factor using the same hierarchical modeling techniques.

Components of CMR were assessed in the morning after a 12-hr overnight fast, as detailed by Manuck et al. (2010). Blood pressure was measured by sphygmomanometry as the mean of two consecutive readings obtained in a seated position. At this visit, a nurse completed a medical history interview, determined body mass index (kg/m2), and drew a 40-ml blood sample. Determination of fasting serum lipids, glucose, and insulin was performed by the Heinz Nutrition Laboratory, University of Pittsburgh Graduate School of Public Health, as described previously (Muldoon, Nazzaro, Sutton-Tyrrell, & Manuck, 2000).[1]

Results

All statistical models were estimated using Mplus 8.3 (Muthén & Muthén, 2019). The effect of sex, age, and race was adjusted for at the level of observed variables in every model. Descriptive statistics for study variables can be found in Table 1.

CMR was modeled following previous work in this sample (Marsland et al., 2010; McCaffery, Marsland, Strohacker, Muldoon, & Manuck, 2012). Four first-order latent factors with two indicators each were specified using CFA: elevated blood pressure (indicated by systolic and diastolic pressures), insulin resistance (fasting insulin and glucose concentrations), heightened adiposity (body mass index and waist circumference), and dyslipidemia (fasting high-density lipoprotein and triglycerides). Good overall fit for this CMR model using this these data has been previously reported (Dermody et al., 2016). A latent factor for BPD was estimated from self and informant ratings of five-factor model traits using a multi-informant modeling approach, which leverages shared variance between raters to improve measurement reliability (DeYoung, 2006). Informant and self-report ratings all showed significant factor loadings on the latent BPD factor. Beck Depression Inventory subscale scores were then used to estimate a latent depression variable. All factor loadings were significant for the BPD and depression factors, and both models were fully saturated.

Once each of these models was estimated individually, all three latent factors were combined into a single CFA to determine overall model fit, along with the zero-order correlations between latent BPD, depression, and CMR. This combined model achieved good fit by all three alternative fit indices ($\chi^2 = 182.64$, $df = 70$; root mean square error of approximation = .04; comparative fit index = .98; standardized root mean square residual = .02). As expected, BPD and depression each had significant associations with CMR ($r = .15$, $p < .001$; $r = .11$, $p = .007$). Furthermore, BPD and depression were strongly correlated, $r = .60$, $p < .001$, supporting the need for the subsequent multivariate model to account for covariation.

For the final model, we regressed CMR on BPD and depression simultaneously to evaluate the effects of nonshared variance (depicted in Figure 1). This model had the same number of parameters and equivalent fit to the combined CFA. When CMR was regressed onto BPD adjusting for the effect of depression, the standardized beta only decreased by .03 and remained significant ($\beta = .12$, $p = .031$). In contrast, the effect of CMR on depression controlling for BPD decreased by .08 and was no longer significant ($\beta = .03$, $p = .568$).

On the request of a reviewer, we added socioeconomic status (measured using reported family income and years of education) as a covariate at the level of observed variables. This addition did not change results of the correlational model. In the regression model, the association between depression and CMR remained nonsignificant ($\beta = .02$, $p = .682$), whereas the effect of BPD on CMR also attenuated by .01 but was no longer significant ($p = .050$). Indicators of socioeconomic status associated modestly with CMR indicators ($\beta s \leq |.05|$), whereas income showed much stronger associations with BPD traits ($\beta s \geq -.18$).

Discussion

In a contribution to the growing literature establishing a link between BPD pathology and cardiovascular disease risk, we aimed to evaluate whether this association exists independently of comorbid depressive symptomology. Consistent with previous research, results indicate that both BPD traits and depressive pathology predicted CMR. However, only the effect of BPD traits remained significant and virtually unchanged after accounting for their shared variance, affirming its unique role over and above co-occurring depression.

Having more firmly established a broad link, this study provides impetus for further investigation of potential pathways that may account for the relationship between BPD pathology and CMR. The strong correlation between BPD traits and depression found in this study, within the context of extensive clinical comorbidity, reinforces the possibility of a shared source of pathology that contributes to risk in each. Our study points to the value in distinguishing general and specific risk factors and the need to account for overlapping features when designing studies of BPD

[1] A sensitivity analysis was performed to evaluate the effect of including participants taking medications that affect cardiovascular measurements used to estimate CMR. Exclusion of participants taking antihypertensives, oral hypoglycemics, and cholesterol-lowering medications resulted in 146 dropped cases ($n = 1{,}153$). Results from this model were virtually identical, so findings from the full sample are reported.

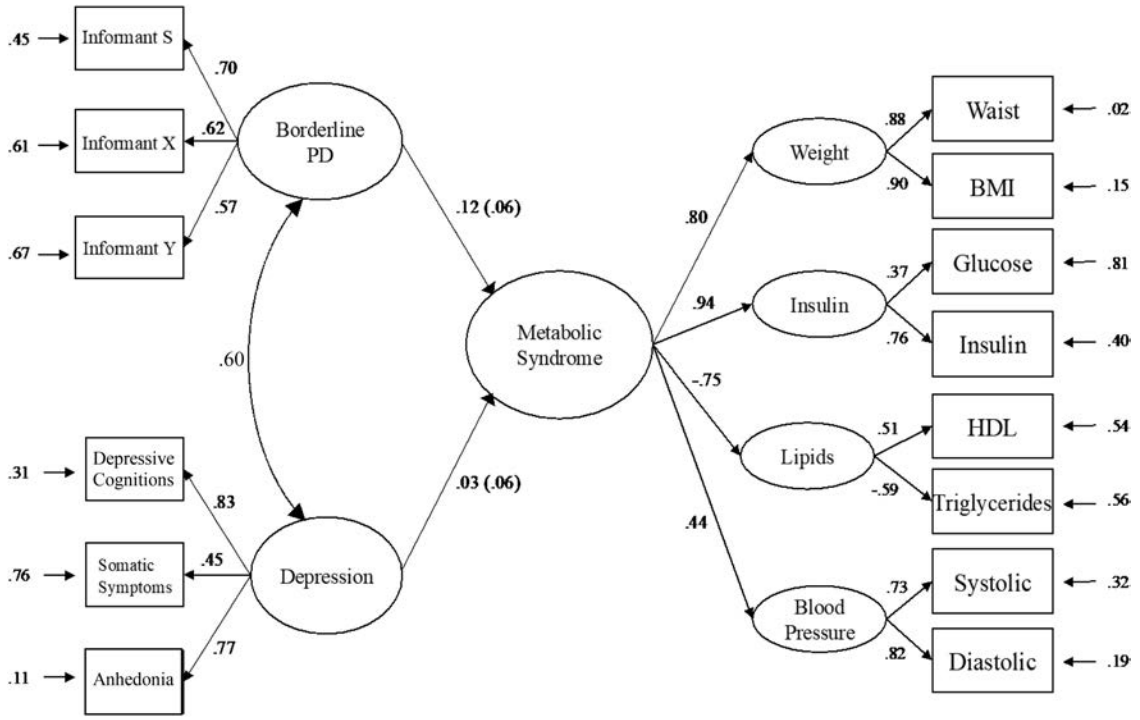


Figure 1. Structural model of metabolic syndrome regressed on borderline personality disorder traits and depression. All parameter estimates presented in standardized form. BMI = body mass index; HDL = high-density lipoprotein; BP = blood pressure; Informant S = self-report; Informants X and Y: other informant reports.

and CMR to clarify processes underlying their association. In addition to shared vulnerability factors, the role of BPD features distinct from depression should be evaluated in future work. Precision gained by examining relationships between specific components of CMR and BPD symptoms involving impulsivity, anger, and interpersonal dysfunction could inform mechanistic models. Another intriguing result was the minor impact on the association between CMR and BPD traits after adjusting for socioeconomic status. This was not due to large direct effects of socioeconomic status on CMR, but rather to relatively large associations with BPD traits. Exploring the relationship between BPD and socioeconomic status could be informative for understanding their joint role in CMR. The additional variance in cardiovascular risk associated with BPD traits independent of shared depressive symptoms could also be a function of *severity* rather than kind of psychopathology. Thus, it is possible that BPD pathology involves a greater breadth of pathological features that have an additive effect on disease risk. Indeed, it is consistently found that the presence of personality pathology negatively affects psychotherapeutic and pharmacological treatment outcomes of depression (Levenson, Wallace, Fournier, Rucci, & Frank, 2012), so it is reasonable to consider this effect in the context of physical health outcomes.

Regardless of how BPD traits relate to CMR, the effect found in this study suggests a clinically meaningful relationship. To compare with other intervening variables a medical professional commonly considers, each standard deviation increase in BPD traits has the same effect on CMR as approximately 9.2 years of aging in the sample used for this study. The effect of BPD traits on CMR is large enough that clinicians treating patients with these features should recommend monitoring of cardiovascular health.

Some limitations to the current study must be noted. Given the relationship between cardiovascular risk and age (North & Sinclair, 2012), it is possible that findings in this sample of midlife adults do not generalize to other periods of development. However, our results are consistent with previous studies of BPD using samples representing a range of ages (El-Gabalawy et al., 2010). Replication of our results with different racial and ethnic groups and using different measures of BPD and depression are also needed to assess their generalizability. In addition, though our use of a multi-informant trait measure of BPD is a methodological strength, some reservation is warranted in direct comparison with the single-informant measure of depression. Unlike previous studies, we did not limit analyses to a particular threshold of clinical severity for either BPD pathology or CMR. It is possible that full syndrome manifestation of BPD or recruitment of a sample enriched for the presence of individuals at high risk for cardiovascular disease would yield even stronger associations. By capturing a continuum of severity in BPD traits, the relationship found with CMR, even at subclinical levels, arguably provides more compelling evidence for this association and could be useful for conceptualizing disease prevention.

Findings from this study underscore the importance of widening the scope of BPD research to include physical health processes and outcomes in addition to psychosocial dysfunction. Understanding the relationship between BPD pathology and cardiovascular disease risk has implications for direct clinical application, as well as

the potential to enrich theories of the interface between physical and psychological functioning.

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