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Dynamic Associations between Borderline Personality Disorder and Stressful Life Events over Five Years in Older Adults

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Abstract

The time course of borderline personality disorder (PD) is far more variable than traditionally assumed. Shifting environmental conditions are theorized to account, at least in part, for fluctuations in symptom presentation over time. In the present study, we evaluated the reciprocal influences of stressful life events and borderline pathology in a representative community sample of 1,630 older adults assessed 3 times over 5 years. An autoregressive cross-lagged model revealed strong, but imperfect, stability in symptoms of borderline PD over the study timeframe. After adjusting for this continuity in borderline PD, the prospective effect of life stress on borderline pathology was virtually nil, contrary to expectations. On the other hand, borderline pathology was prospectively related to subsequent dependent event (i.e., stressors to which individuals may have contributed), but not independent event (i.e., fateful stressors), exposure. This pattern of associations was consistent with a *stress generation* effect. We conclude that stressful life events do not substantially redirect the trajectory of borderline PD in older adults, possibly owing to inertia of borderline pathology at this developmental stage. Instead, symptoms of borderline PD seem to promote stress exposure, thereby setting the stage for continued social impairment and comorbid psychiatric problems.

Keywords

autoregressive cross-lagged; borderline personality disorder; life stress; longitudinal; stress generation

Borderline personality disorder (PD) historically has been considered a relatively intractable condition. Undercutting this received wisdom, recent longitudinal data indicate that appreciable improvement in borderline PD over time is not only possible but exceedingly common. Across 2 large clinical studies—the Collaborative Longitudinal Personality Disorders Study and the McLean Study of Adult Development—close to 90% of patients achieved diagnostic remission over 10 years of follow-up (Gunderson et al., 2011; Zanarini, Frankenburg, Reich, & Fitzmaurice, 2010). Further, a sizable minority of patients who recovered subsequently relapsed (Zanarini et al., 2010). These observations do not imply, however, that borderline PD is completely unstable in its presentation. Indeed, when

assessed dimensionally, borderline pathology demonstrates moderate-to-strong continuity, even over long intervals (e.g., Morey & Hopwood, 2013). Further, functional impairment tends to persist even in the context of patients' symptomatic remission (e.g., Gunderson et al., 2011; Paris, 2003; Skodol et al., 2005). Based on these new lines of evidence, investigators have reconceptualized borderline PD as a hybrid of stable and fluid processes (e.g., Paris et al., 2009; Widiger, 2009).

Models of change in features of borderline PD are at an embryonic stage, presumably owing to the prevailing view of borderline PD as a lifelong condition. By comparison, there is a robust research literature on the temporal course of quintessentially episodic disorders, such as major depression (e.g., Monroe & Harkness, 2011). We argue that empirical work on the exacerbation (and alleviation) of borderline PD is required to keep pace with the emerging (re)formulation of borderline pathology as a time-varying condition, akin to other emotional disorders. Such investigation promises to improve risk forecasting, prognostic decisions, and identification of targets for clinical intervention (e.g., Chanen & McCutcheon, 2013).

Stressful environmental conditions are a strong candidate to shape the temporal course of borderline PD. Stress exposure is intimately intertwined with various psychiatric conditions that overlap with borderline PD, including anxiety, depressive, substance use, and psychotic disorders (e.g., Conway, Rutter, & Brown, 2016; Hammen, 2005; Hlastala et al., 2000; Kim, Conger, Elder, & Lorenz, 2003; Monroe, Slavich, & Georgiades, 2014; Myin-Germeys & van Os, 2007). A comparatively small, but consistent, line of research attests to the connection between environmental stress and borderline PD (e.g., Stepp, Olino, Klein, Seeley, & Lewinsohn, 2013). Childhood maltreatment, in particular, has been implicated repeatedly in etiological theories of borderline PD (Cohen, Crawford, Johnson, & Kasen, 2005; Zanarini & Frankenburg, 1997). The vast majority of this research and theorizing, however, concerns *early* environmental adversity (cf. Distel et al., 2011; Jovev & Jackson, 2006); the degree to which stress exposure later in development influences the trajectory of borderline pathology remains unknown. There is, however, preliminary evidence from clinical studies that recovery from borderline PD is delayed by toxic environmental conditions such as abuse and exposure to violence (Zanarini, Frankenburg, Reich, & Fitzmaurice, 2010).

Whereas prior clinical studies focus on acute shifts in borderline PD symptoms, a complementary line of research shows that major life events can alter the course of the normal personality traits that form the scaffolding for borderline PD. Studies supporting social investment theory (Roberts, Wood, & Smith, 2005), for instance, document that marrying and entering the workforce stimulate personality maturation (e.g., increases in agreeableness and conscientiousness, decreases in neuroticism). Unemployment, on the other hand, produces the reverse trends (Bleidorn, 2015). Given the connections between five factor model personality traits and borderline PD risk (e.g., Widiger & Costa, 2002), such environmental transitions might be expected to shape the course of borderline pathology.

The longitudinal association between borderline PD and stress exposure may be bidirectional. People at risk for borderline PD are theorized to evoke, create, and select into

stressful environments (e.g., Hopwood, Donnellan, & Zannarini, 2010; Wilson, Stroud, & Durbin, 2017). This phenomenon, known as stress generation, originally was described in the context of depression, but has since been found to characterize anxiety, substance use, and antisocial behavior disorders (Conway, Hammen, & Brennan, 2012; Hammen, 2006; Liu & Alloy, 2010). These investigations show that psychopathology and related risk factors (e.g., cognitive biases) portend elevated rates of *dependent* stressors, or those that individuals—at least in part—trigger or maintain (e.g., romantic relationship breakup, layoff due to poor work performance). In contrast, stress generation theory holds that risk for mental disorder should not be prospectively associated with exposure to *independent* (or fateful) stressors (e.g., natural disaster, death of a loved one) (Hammen, 2006).

A number of prior longitudinal studies have connected borderline PD with the stress generation effect. Daley et al. reported that Cluster B (i.e., antisocial, borderline, histrionic, and narcissistic) PD symptoms were prospectively associated with interpersonal stressors—especially those initiated by participants—over 2 years in a sample of high school girls (Daley, Hammen, Davila, & Burge, 1998). This research group later found in a 4-year follow-up of the same sample that borderline PD symptoms predicted romantic relationship conflict, partner abuse, and unwanted pregnancy (Daley, Burge, & Hammen, 2000). Powers, Gleason, and Oltmanns (2013) extended these findings in a large community sample that was administered a comprehensive interview assessment for PDs and questioned about recent life stressors. They found that, after adjusting for PD comorbidity, only borderline PD symptoms were prospectively associated with interpersonal stressors over the 18-month study timeframe.

Current Study

We investigated for the first time the prospective, reciprocal influences of borderline pathology and stress exposure in adulthood. We assessed 1,630 community adults 3 times over a 5-year interval. At each wave, we collected interview and self-report measures of borderline pathology, and interviewers probed the nature and severity of recent stressors. We used autoregressive cross-lagged models to evaluate the longitudinal effect of stress exposure on borderline pathology, adjusting for continuity in borderline pathology (and vice versa).

We predicted a dynamic pattern of interrelations between these two constructs. Given available data that show environmental stressors contribute to the onset and maintenance of borderline PD (e.g., Stepp et al., 2013; Zannarini et al., 2006), we hypothesized that life stress would be associated with future increases in borderline pathology (i.e., a *stress exposure* effect). Also, based on prior theory and empirical work (e.g., Powers et al., 2013), we expected that borderline pathology would prospectively influence exposure to serious dependent, but not independent, stressors (i.e., a *stress generation* effect).

Methods

Sample

The sample consisted of 1,630 older adults (55% female) participating in the St. Louis Personality and Aging Network, a longitudinal study of personality, health, and aging in later life. Participants were between the ages of 55 and 64 ($M = 59.6$ years; $SD = 2.7$) when they entered the study. The majority of participants identified as White (65%), 32% as African American, and 2% as Hispanic or Latino; the remaining 1% identified as a different race (e.g., Asian, American Indian). Slightly more than half the sample (51%) was either married or in a serious relationship and had a bachelor's degree or higher (52%) (see Oltmanns, Rodrigues, Weinstein, & Gleason, 2014 for more description about the sample characteristics and study protocol).

Procedure

A baseline assessment consisted of an informed consent process including a thorough explanation of the procedures, followed by a battery of interviews and questionnaires administered over a 3-hour session. Subsequent to the baseline assessment, participants were contacted approximately every six months to complete a packet of questionnaires at home. At 2.5- and 5-year follow-up assessments, participants again completed a 3-hour in-person session mirroring the baseline protocol. Questionnaire measures were completed by paper-and-pencil or on a personal computer, either at home or in our lab. For the purpose of the current study, we analyzed PD data collected from the baseline, 2.5-, and 5-year assessments; stressful life event data were analyzed from the first 6-month follow-up, and the 2.5- and 5-year assessments. Stressful life events were not assessed at study baseline. All study procedures were approved by the Washington University in St. Louis Institutional Review Board.

Measures

Structured Interview for *DSM-IV* Personality (SIDP-IV; Pfohl, Blum, & Zimmerman, 1997)—The SIDP-IV is a semi-structured diagnostic interview designed to assess the PDs listed in *DSM-IV*. Trained interviewers (i.e., graduate students in clinical psychology and trained research assistants) rated each criterion on a 4-point, Likert-type scale ranging from 0 (*not present*) to 3 (*strongly present*). For the purpose of the present study, ratings on the nine borderline PD criteria were summed to create a continuous measure of borderline PD symptoms. All interviews were video recorded, and a subsample of 265 participants were randomly selected to be rated by independent judges. Interrater reliability coefficients were computed for each PD using a one-way random, average measures intraclass correlation coefficient. The reliability coefficient for borderline PD ranged from .77 to .89 across waves, indicating acceptable reliability.

Multi-source Assessment of Personality Pathology (MAPP; Oltmanns & Turkheimer, 2006)—The MAPP is an 80-item measure of personality pathology based on lay translations of the *DSM-IV* PD diagnostic criteria. Each criterion was rewritten into words that avoided the use of technical terms and psychiatric jargon. Listed in a quasi-random order, the items are rated on a 5-point, Likert-type scale ranging from 0 (*I am never*

like this) to 4 (*I am always like this*). In the current study, we summed responses to the nine borderline PD items to serve as a continuous indicator of borderline pathology. Coefficient alpha for the borderline PD items was .68 at each of the three assessment waves.

NEO Personality Inventory–Revised (NEO PI-R; Costa & McCrae, 1992)—The NEO PI-R is a 240-item instrument designed to assess the five major personality trait domains of neuroticism, extraversion, openness to experiences, agreeableness, and conscientiousness, as well as six trait facets that define each domain. The items are rated on a 5-point, Likert-type scale ranging from 0 (*strongly disagree*) to 4 (*strongly agree*). Following procedures described in Miller, Reynolds, and Pilkonis, (2004), borderline PD prototype scores were calculated by summing scores across the nine facet scales shown to be directly associated with BPD (i.e., anxiety, angry hostility, depression, impulsivity, vulnerability, openness to feelings, openness to actions, compliance [reverse scored], and deliberation [reverse scored]). For these nine facet scales, coefficient alphas averaged across time points ranged from .62 (compliance) to .83 (depressiveness) (median alpha = .69). Reliability estimates for the composite ranged from .73 to .74 across waves.

List of Threatening Experiences Questionnaire (LTE-Q; Brugha, Bebbington, Tennant, & Hurry, 1985)—The LTE-Q consists of 12 stressful life events (e.g., major financial crisis, marital separation) found to be associated with severe or moderate long-term threat by consensus ratings (Bebbington, Tennant, & Hurry, 1981). The 12 items were presented in a checklist format along with two additional items added to the study protocol by the research team (i.e., victim of a serious crime, major changes in family responsibilities). The questionnaire instructed participants to report only those stressful events occurring in the past six months. If at least one event was checked on the LTE-Q, the participant was called by a trained interviewer who inquired about the nature of the event. Interviewers asked participants if the event occurred in the specified time frame (i.e., the preceding six months) and was a major event that caused considerable distress (see Gleason, Powers, & Oltmanns, 2012, for a full description of the follow-up interview). Events judged by interviewers to not meet both of these criteria were not counted as stressors.

Of those stressors confirmed by interviewers, we divided events into independent and dependent stressors. As indicated above, independent stressors are those that are unrelated to individuals' attitudes and behavior, whereas dependent stressors are influenced to some degree by individuals' actions (Hammen, 2006; see also Paykel, 1987). We categorized the following events as independent: personal illness or injury; serious illness or injury to a close relative; death of a partner, parent, or child; death of a close friend or another relative; victim of a serious crime; major changes in family responsibilities (e.g., caring for elderly parents). In contrast, the following were classified as dependent: marital separation; breaking off a steady relationship; conflict with close friend; unemployment; fired from a job; financial trouble; legal problems; something valuable was lost.

Statistical Analysis

An autoregressive cross-lagged (ARCL) model of the prospective associations between borderline pathology, dependent life stress, and independent life stress was estimated in

Mplus (version 7.11; Muthén & Muthén, 1998–2014) using the WLSMV estimator. Latent variable modeling was used to capture the shared variance among the multiple indicators of borderline PD, whereas both life stress variables were operationalized as the observed count of interviewer-confirmed stressful life events in the 6 months preceding each assessment wave. Between 0.0% and 1.7% of participants across waves endorsed 3 or more stressful events within each stress category (i.e., independent, dependent). Therefore, event counts of 2 or more were collapsed into a single response category, such that values for each stress variable could range from 0 to 2.

To evaluate model fit, we used the comparative fit index (CFI), the Tucker-Lewis index (TLI), the root mean square error of approximation (RMSEA), and the weighted root mean square residual (WRMR). Acceptable model fit was defined according to guidelines offered by Hu and Bentler (1999): RMSEA values close to 0.06 or below, CFI and TLI values close to .95 or above, and SRMR values close to .08 or below. Missing data were accommodated using full information maximum likelihood. Retention rates across 2.5- and 5-year follow-up waves were 78.4% and 66.1%, respectively. Attrition was not statistically significantly related to baseline levels of life stress or borderline pathology, $t_s < 1.96$, $p_s > .05$.

Results

Descriptive Statistics

Table 1 presents the rates of stressful life events and borderline PD features across study waves. Stress exposure was fairly common, in that approximately half of the sample endorsed at least 1 major stressor (dependent or independent) at any given assessment. The correlations in Table 1 illustrate that there was statistically significant, albeit modest, continuity in exposure to both dependent and independent stress, and moderate-to-large (depending on the indicator) continuity in borderline PD severity across time. Additionally, as expected, life stress and borderline pathology were associated cross-sectionally (median r for dependent events: .16; median r for independent events: .06).

Longitudinal Measurement Model

We tested for measurement invariance in borderline pathology, a precondition for ARCL modeling, using longitudinal confirmatory factor analysis (e.g., Horn & McArdle, 1992). In a first step, we specified a model in which all factor loadings were freely estimated across time. Second, we imposed weak measurement invariance by testing a model in which loadings of the same indicators (e.g., NEO₁, NEO₂, NEO₃) were constrained to equality over time. In both models, we allowed cross-wave error covariances for the same indicator to account for shared method variance.

The unconstrained confirmatory factor analysis fit the data well, $\chi^2(15) = 54.27$, $p < .001$; CFI = .99; TLI = .99; RMSEA = 0.04; SRMR = 0.02. Factor loadings were moderate-to-strong (λ range: .58–.77) and statistically significant at a .001 alpha threshold. Restricting factor loadings to equality across time in the constrained model did not significantly diminish model fit, $\chi^2_{\text{diff}}(4) = 8.90$, $p = .06$. This test therefore supported weak

measurement invariance, and we carried forward this constrained measurement model of borderline PD to ARCL analyses.

Autoregressive Cross-Lagged Model

The ARCL model estimated the prospective effects of study constructs on one another, adjusting for continuity across contiguous waves. Thus, the model included autoregressive paths (e.g., bpd_2 regressed on bpd_1) and cross-lagged paths (e.g., bpd_2 regressed on dep_1) (see Figure 1). Contemporaneous (i.e., within-time) error covariances were freely estimated to reflect shared causes of borderline pathology and life stress not included in this model, and the covariances of Time 1 borderline pathology with the two Time 1 life stress variables were also freely estimated.

This initial ARCL model fit adequately, $\chi^2(64) = 304.16$, $p < .001$; CFI = .98; TLI = .96; RMSEA = 0.05; WRMR = 1.15. To maximize model parsimony, we tested several sets of equality constraints to determine whether equating various parameters over time would cause a decrement in model fit (see Curran & Bollen, 2001). We sequentially restricted the borderline pathology autoregressive paths (i.e., regressions of bpd_2 on bpd_1 , and bpd_3 on bpd_2), life stress autoregressive paths, cross-lags of borderline pathology on life stress (i.e., bpd_2 on dep_1 , bpd_3 on dep_2 , bpd_2 on ind_1 , and bpd_3 on ind_2), cross-lags of life stress on borderline pathology, and contemporaneous error covariances to equality. None of these restrictions significantly reduced model fit, relative to the prior model in the sequence, $\chi^2_{diffs} < 3.84$, $ps > .05$. Further, the model CFI never changed appreciably as a result of these restrictions; the largest CFI change was an *improvement* of .004 unit associated with constraints on residual covariances (see Cheung & Rensvold, 2002). The final model, which imposed all equality constraints, fit the data well, $\chi^2(76) = 227.14$, $p < .001$; CFI = .99; TLI = .98; RMSEA = 0.03; WRMR = 1.18.

Figure 1 shows the standardized path coefficients from the final model. All autoregressive paths were statistically significant, reflecting continuity in each construct across waves. Autoregression for borderline pathology was especially large (standardized coefficients $> .90$). In contrast, the prospective effect of life stress on borderline pathology, net continuity in borderline pathology, was almost nil. The reverse cross-lagged pathway, reflecting the longitudinal contribution of borderline pathology to stress exposure, was statistically significant for dependent (standardized coefficients = .22 and .21), but not independent (standardized coefficients = .03 and .03), events. The unstandardized path coefficients and corresponding standard errors are presented in Table S1 in the online supplementary materials.

Alternate Models

In auxiliary analyses, we respecified the ARCL to include only a single indicator of borderline pathology. Thus, we estimated a series of three separate multivariate models—one each for the SIDP-IV, NEO PI-R, and MAPP—that represented borderline pathology as an observed, as opposed to latent, variable. Results from these analyses are presented in supplementary online materials (Tables S2–S4). The retest stability of borderline pathology was expectedly lower in these analyses, except that continuity in the NEO PI-R was only

modestly smaller (β range: .85–.88) than that of the latent borderline pathology variable. The statistically significant stress generation effect was conserved in these new models (β range: .13–.19). Finally, in contrast to the main analyses, we observed significant stress exposure effects (β range: .04–.15) for two (SIDP-IV, MAPP) out of the three observed indicators of borderline PD.

Discussion

We theorized that stress exposure and borderline pathology would be linked in a cycle of mutually reinforcing associations over time. In a large sample of older adults, we performed a multimethod evaluation of borderline PD and conducted interviews regarding recent stressful events 3 times over a 5-year span to evaluate these dynamic influences. Contrary to hypotheses, we found that exposure to stressful life events had virtually no effect on subsequent severity of borderline PD. On the other hand, borderline pathology prospectively predicted rates of dependent stressful life events above and beyond continuity in exposure to life stress.

This “nil effect” of stress exposure on borderline PD may be related to the considerable continuity in borderline pathology observed here. Differential (i.e., rank-order) stability—reflected in a retest correlation—for the borderline pathology construct exceeded .90 across waves. This level of persistence is much larger than estimates over similar intervals—generally in the range of .40 to .60—reported in younger, patient samples (Hopwood et al., 2010; Morey et al., 2007; for a review, see Morey & Hopwood, 2013). Additionally, in the Children in the Community Study, the corresponding estimates over 3 and 6 years were .44 and .36 for a representative sample of 816 adolescents (Johnson et al., 2000). The strong autocorrelation in the current study, in contrast, left little variation in borderline pathology to be explained by other exogenous factors (i.e., dependent and independent stress) at follow-up waves. This observation has key implications, we believe, for the design of future research on the longitudinal relations between life stress and borderline pathology. That is, in the context of elevated borderline PD continuity, the signal of any pathogenic effects of stressful events is likely to be so muted as to be undetectable in older adults. The influence of life stress theoretically will be more prominent in times when borderline PD is more fluid (i.e., has a larger time-varying component), such as during adolescence or following psychiatric intervention (cf. Cole et al., in press).

There are at least 2 (not mutually exclusive) possible reasons for the heightened stability of borderline pathology in the present study. First, our cross-lagged model included a latent variable reflecting borderline pathology, defined by multiple interview and questionnaire manifest indicators of borderline PD. This measurement approach maximized the estimated autoregression for borderline PD by ensuring that it was unattenuated for measurement error. By comparison, the continuity in the Structured Interview for *DSM-IV* Personality—the most popular (in prior research) indicator of borderline PD used in this study—was .53 and .63 over the 2 2.5-year intervals in these data. Second, to the best of our knowledge, the SPAN project is the first to examine the continuity of borderline pathology in older adulthood. Borderline PD—like other PD constructs—may be especially invariant during this developmental stage, when there is comparatively little change in many of the normative

personality traits undergirding borderline PD (e.g., Roberts & Mroczek, 2008) and several externalizing features (e.g., impulsive and suicidal behavior) relevant to the disorder are relatively infrequent (e.g., Stepp & Pilkonis, 2008; Stevenson, Meares, & Comerford, 2003).

The small influence of stress exposure observed here is at odds with an abundance of evidence on the deleterious effects of early environmental adversities on borderline PD liability. A large and consistent literature on childhood maltreatment shows that early abuse and neglect are strongly related to future risk for borderline PD (e.g., Carlson, Egeland, & Sroufe, 2009; Horesh, Ratner, Laor, & Toren, 2008; Zanarini et al., 1997), along with many other psychiatric problems (e.g., Green et al., 2010). Chronic stressors, such as academic failure and parent-child conflict, in adolescence and young adulthood have also been linked to borderline pathology (e.g., Conway, Hammen, & Brennan, 2015; Stepp et al., 2013). Further, behavioral genetic research has demonstrated that this association is, at least in part, causal, as opposed to an artifact of overlapping genetic vulnerabilities to stress exposure and borderline PD (Distel et al., 2011, cf. Berenz et al., 2013). Against this backdrop, then, it was unexpected to find essentially no contribution of life stress to change in borderline pathology. However, we believe it is significant that prior research has concentrated almost exclusively on the effects of life stress early in development. We are aware of no previous longitudinal research that has evaluated the reciprocal relations between life stress and borderline pathology after the transition to adulthood. It is possible that the nature of these dynamic associations changes across development to the point that stress exposure has a very limited influence on the maintenance of symptoms of borderline PD in older adults. Future developmental research with repeated measures of life stress and borderline PD is needed to resolve this question.

Whereas the stress exposure model was not supported here, there was evidence consistent with the stress generation model. Elevations in borderline pathology prospectively predicted increased rates of stressful life events, net continuity in life stress. Although the stress generation phenomenon originally was documented in the context of depression, it has since been detected in research on diverse emotional disorders (e.g., anxiety disorders) and other psychiatric conditions commonly comorbid with borderline PD (e.g., Conway et al., 2012; Liu & Alloy, 2010). Indeed, a small, but consistent, body of research has found robust stress generation effects for borderline and other cluster B PDs, albeit mostly among adolescents and young adults (e.g., Daley et al., 1998; Daley et al., 2000; Powers et al., 2013). Our results extend this prior work by showing that stress generation continues to operate in later adulthood and by adjusting our estimate of the size of the stress generation effect by controlling for stability of stress exposure (see, e.g., Hazel & Hankin, 2014). The magnitude of the observed effect was moderate (standardized coefficient $\approx .22$), and it exceeded the degree of continuity in both life stress constructs.

Although we did not find an increase in symptoms of borderline PD following exposure to stress, it remains likely that stress does have a harmful impact on the physical health and social adjustment of individuals who exhibit symptoms of the disorder in later life. Several other studies have documented the persistent nature of these difficulties in the lives of borderline PD patients (e.g., Keuroghlian & Zanarini, 2015). Previous papers from the SPAN study have reported a significant link between borderline pathology and impaired

physical health among older adults (Powers & Oltmanns, 2012), and we have found that the impact of personality on physical health is mediated by stressful life events (Iacovino, Bogdan, & Oltmanns, 2016). Therefore, conceptual models regarding the link between stress exposure and borderline PD across the lifespan should be expanded to consider the impact of stress on a variety of important life outcomes, above and beyond the presence and severity of the diagnostic symptoms that are used to identify the disorder itself.

Limitations

Our study benefitted from a representative community sample, multimethod assessment of borderline pathology, and favorable retention rates over 5 years of older adulthood. Several study limitations should be considered alongside these strengths, however. First, strong continuity in the borderline pathology latent variable might have restricted our ability to detect stress exposure effects, assuming they exist. In supplementary analyses, we found some evidence of (small) stress exposure paths when only one manifest variable was used to represent borderline pathology. It may be difficult for any predictor to explain significant incremental variance in a highly reliable outcome, and we therefore caution that our main results may not be an appropriate reflection of stress exposure effects in all research contexts or at all developmental stages.

Second, although interviewers confirmed that reported stressful life events had a meaningful impact on participants, they did not probe the full details of event occurrence. We therefore did not compute objective indices of the events' expected long-term impact and instead analyzed the count of major life events. This was a necessary strategy given the labor-intensive nature of "contextual threat" interview procedures for life stress (Brown & Harris, 1978; Hammen, 2006), relative to our sample size and follow-up structure (see also Dohrenwend, 2006). Along these same lines, we divided life events into dependent versus independent stressors based on judgment about whether certain classes of events *most often* depended on individuals' actions or not. Because we did not make this determination based on contextual information regarding each instance of a stressor, some individual stressors may have been misclassified. For example, a participant could have endorsed being the victim of a serious crime—an event we classified as most often independent of participants' actions—after being assaulted with a weapon; however, it could have been the case that the participant himself began a fight with an acquaintance after drinking heavily. With this context in mind, most raters would judge this particular event to be dependent, at least in part, on the participant.

Third, we expect these results to extend to the full population of older adults, but we caution that generalizability to other developmental groups should be tested empirically and not assumed. Older adulthood may be a period of increased stability for borderline PD, whereas adolescence and early adulthood are known for greater change (Bornovaalova, Hicks, Iacono, & McGue, 2009). Analogously, the landscape of stressful life events shifts across development as a function of changing social and occupational environments, possibly altering both stress exposure and stress generation effects. Adolescents, for instance, encounter more interpersonal conflicts with friends and romantic partners, whereas older adults report more health-related stressors (e.g., Jordanova et al., 2007; Rudolph &

Hammen, 1999). Fourth, stress exposure and stress generation hypotheses can be—and have been—tested over various timescales. Stress exposure, for instance, might have immediate effects on some outcomes (e.g., acute biological processes), whereas it could take years for other downstream consequences (e.g., shifts in neuroticism) to unfold (Bleidorn, Hopwood, & Lucas, 2016). We are not certain what the optimal timeframe is for evaluating connections between fluctuations in borderline pathology and stressful events. We advise future high-resolution, longitudinal research with repeated measurement occasions before and after significant life events to address this question.

Conclusion

Life stress does not appear to exacerbate borderline pathology in older adulthood, perhaps because PD is especially stable during this developmental period. In contrast, stress generation effects were robust, possibly setting the stage for continued functional impairment and further psychiatric problems. These findings require confirmation in other samples to determine whether the pattern of stress exposure and stress generation effects observed here is specific to older adults. It will also be important to examine the possible impact of stress exposure on other aspects of health as well as social and occupational impairment, which are often persistent areas of concern for people who experience borderline PD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Autoregressive cross-lagged model of borderline pathology and life stress over 5 years.

Regression paths with life stress variables as predictors are standardized with respect to the outcome variable; all other paths are fully standardized. IND = independent life stress; DEP = dependent life stress; BPD = borderline PD; NEO = NEO Personality Inventory–Revised; MAPP = Multi-source Assessment of Personality Pathology; SIDP = Structured Interview for *DSM-IV* Personality. Contemporaneous error covariances are omitted here for clarity of presentation (see Table S1 for full results). Observed borderline PD indicators are also omitted for clarity. The standardized factor loadings for the SIDP (lambdas = .61, .65, .71 at Times 1–3), MAPP (.71, .75, .69), and NEO (.75, .74, .71) were all statistically significant at the .001 alpha level. Dashed lines denote statistically nonsignificant effects. ** $p < .01$, *** $p < .001$.

Table 1

Pearson Correlations and Descriptive Statistics for Life Stress and Borderline Pathology

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	M	SD
1. DEP1	—														0.21	0.47
2. DEP2	.09	—													0.16	0.41
3. DEP3	.10	.11	—												0.12	0.36
4. IND1	.08	.08	.05	—											0.46	0.66
5. IND2	.03	.09	.04	.08	—										0.45	0.64
6. IND3	.07	.04	.06	.16	.14	—									0.47	0.65
7. NEO1	.20	.12	.15	.09	.00	.05	—								120.62	21.76
8. NEO2	.14	.16	.13	.06	.04	.03	.82	—							117.09	21.54
9. NEO3	.19	.13	.16	.10	.04	.06	.79	.82	—						120.37	21.96
10. MAPP1	.15	.05	.13	.06	.02	.05	.55	.46	.48	—					3.85	3.55
11. MAPP2	.10	.09	.11	.04	.06	.02	.46	.56	.47	.62	—				3.47	3.30
12. MAPP3	.15	.11	.12	.05	.00	.03	.43	.45	.54	.55	.59	—			4.11	3.50
13. SIDP1	.25	.12	.12	.09	.02	.05	.46	.39	.41	.42	.34	.30	—		1.14	1.86
14. SIDP2	.21	.14	.16	.09	.02	.06	.44	.48	.45	.46	.49	.41	.53	—	0.86	1.71
15. SIDP3	.21	.09	.16	.09	.09	.10	.38	.39	.44	.46	.45	.48	.51	.63	0.63	1.52

Note. DEP = dependent life events; IND = independent life events; NEO = NEO Personality Inventory-Revised; MAPP = Multi-source Assessment of Personality Pathology; SIDP = Structured Interview for DSM-IV Personality. The range of observed scores was 0–2 for life stress variables, 0–17 for SIDP, 0–33 for MAPP, and 53–225 for NEO. All correlations greater than |.06| were statistically significant at $p < .05$.