

Using Repeated Daily Assessments to Uncover Oscillating Patterns and Temporally-Dynamic Triggers in Structures of Psychopathology: Applications to the *DSM–5* Alternative Model of Personality Disorders

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Articulating an accurate and clinically useful structure of psychopathology is a crucial and difficult task. Dimensions identified through cross-sectional factor analyses are increasingly being linked with temporally dynamic processes of social cognition, emotion regulation, symptom expression, and functional impairment to demonstrate how between-person structures and within-person dynamics can be integrated. The present research considers how structure and processes are integrated in the *DSM–5*, specifically in the alternative model for personality disorders (AMPD). Participants ($n = 248$) completed a 14-day electronic diary, and results indicated that personality impairments oscillated across days and were triggered by daily negative emotions and cognitive distortions. Importantly, some aspects of the AMPD model that are identified as potentially redundant in cross-sectional research are shown here to increment each other in the prediction of dynamic oscillations and triggers. Thus, longitudinal designs and temporally dynamic analyses may provide new and novel evidence to fully inform structures of psychopathology. Such research is a needed step in the integration of the structure and process in classification and diagnosis of psychopathology.

General Scientific Summary

This study found that the dimension of time (i.e., variability of symptoms across days) adds important nuance to distinguishing aspects of personality dysfunction. Also, the dimension of time can help us integrate the between-person and single occasion notions of psychopathology structure with the within-person and ever changing oscillations of impairments that patients experience in daily life.

Keywords: experience sampling methodology, multilevel modeling, personality inventory for *DSM–5*, *DSM–5* alternative personality disorder model, structure of psychopathology

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Articulating a more precise and accurate description of psychopathology is a fundamental task in clinical science. In recent years, an effort to identify the core dimensions of psychopathology has been pursued by investigators examining the phenotypic structure of psychopathology (e.g., Caspi et al., 2014; Harkness, Reynolds, & Lilienfeld, 2014; Krueger, 1999; Watson, Clark, & Chmielewski, 2008), and by the National Institute of Mental Health in its Research Domain Criteria which seeks to identify a

taxonomy of neuroscientific constructs that relate to psychopathology (Cuthbert & Kozak, 2013; Lilienfeld, 2014). In addition, the field increasingly recognizes that the static dimensional structures of psychopathology can be enhanced by also considering how psychopathology manifests across time (Hopwood, Zimmermann, Pincus, & Krueger, 2015). Integrating these static structures with temporally dynamic processes is a crucial avenue to better understand how psychopathology presents, maintains, and might ultimately be alleviated. A classification and diagnostic system that integrates structure and process also brings science and practice closer together because clinicians must go beyond a static diagnostic label and try to understand the mechanisms and triggers that exacerbate or mitigate symptoms over time in patients' daily lives.

The present research considers how structure and processes are integrated in the *Diagnostic and Statistical Manual of Mental Disorders (DSM–5; APA, 2013)*. In particular, the *DSM–5* alternative model for personality disorders (AMPD) is used as an example to demonstrate the value of integrating structure and dynamics for informing dimensional models of psychopathology.

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Structures and Dynamics in the *DSM-5*

In the *DSM-5*, a psychological disorder is defined categorically by its characteristic features based largely on the medical model of descriptive psychiatry (Decker, 2013). Over the last several decades, considerable attention has been given to articulate a more precise structure of the features of psychopathology, with the hope that a more efficient organization can reduce diagnostic comorbidity and enhance clarity of communication among clinicians (e.g., Krueger, 1999). One approach toward this goal employs factor analysis to common disorders, finding that diagnoses consistently organize into two broad spectra of internalizing and externalizing dimensions which reliably emerge across age, sex, ethnicity, and culture (for review see Wright et al., 2013). Other research demonstrates that structural analyses including lower base rate disorders such as schizophrenia reveal a third spectra of psychosis/thought disorder (Kotov et al., 2010; Wright et al., 2013), and if personality disorder criteria are included, fourth (pathological introversion/detachment) and fifth (antagonism) dimensions also emerge (Markon, 2010; Wright & Simms, 2015).

An important additional step needed to advance understanding of psychopathology is to consider a temporal dimension that allows for the integration of psychopathology structure and associated dynamic processes to inform a more evidence based and clinically useful diagnostic scheme (Hopwood et al., 2015). In the *DSM-5*, this might be represented through prototypical treatment course descriptions. For instance, some disorders can resolve within months (e.g., major depressive episode, adjustment disorder), certain features can resolve while others remain (positive vs. negative symptoms in schizophrenia), and other disorders represent an expected more chronic course (e.g., untreated obsessive-compulsive disorder, personality disorders).

Examining the stability (vs. variability) of symptoms can also bring to life several important dynamics in psychopathology. For instance, Hopwood, Zimmermann, Pincus, and Krueger (2015) described how the five dimensions of psychopathology can be thought of as set points of dysfunction that may become exacerbated during certain life circumstances that trigger impairment (Mischel & Shoda, 1995; Miskewicz et al., 2015). In fact, the *DSM-5* articulates several context-symptom patterns where psychopathology is exacerbated. Examples include posttraumatic stress disorder (e.g., reexperiencing trauma stimuli triggering avoidance behaviors), phobias (exposure to phobic material triggers withdrawal), and obsessive-compulsive disorder (disturbing thoughts triggering ritualistic undoing behaviors) to name a few.

Through symptom course and context-symptom patterns, the *DSM-5* already articulates how the dimension of time is important for understanding psychopathology. The *DSM* would be enhanced if these assumptions about time were grounded more centrally in empirical research. For instance, the *DSM* assumption that personality disorders are stable across time seems somewhat incomplete given the several longitudinal studies of personality disorder symptoms showing more nuanced results across the timescale of years (for a review see Morey & Hopwood, 2013). Several other psychological disorders in the *DSM* require a 6 month (minimum) timeframe of symptom experience in order to diagnose a psychological disorder. For many of these disorders, it is unclear how the timeline of 6 months was chosen, and whether that particular timeline maximizes the utility of the diagnosis.

Increasingly, researchers are collecting data from participants over short bursts of time (e.g., hours, days, weeks, etc.) and applying advanced statistical methods to better understand the course of symptom experience. For instance, Wright and Simms (2016) asked patients with a personality disorder to report their personality pathology over 100 days. They found that personality pathology was variable across days and that some aspects of personality pathology were more variable than others. However, a summary of a patient's overall level (average) and variability of personality pathology was relatively consistent across months, indicating personality dysfunction is a complex mix of stable and dynamic processes.

Designs such as this can reduce certain retrospective biases (e.g., mood state congruent recall, availability heuristic), and also improve ecological validity by examining patterns that occur in the naturalistic settings of participants' daily life. These methods could even be used to determine what length of time for symptoms is most appropriate to use in describing various psychological disorders.

Structures and Dynamics in the Alternative Model of Personality Disorders

Perhaps the best instantiation of *DSM-5* diagnostic criteria informed by empirical structural models of personality and psychopathology is the AMPD found in Section III. Some have even argued that the AMPD represents the vanguard of conceptualizing psychopathology since the model is consistent with the larger metastructure of psychopathology (Krueger, 2013). We consider how structure and dynamics are described in this alternative model (Krueger, Hopwood, Wright, & Markon, 2014).

Structure

The AMPD includes two criteria that articulate the dimensional components of personality features and dysfunction. Criterion B includes five pathological personality traits reflecting individual differences in how personality pathology manifests (Krueger, Deringer, Markon, Watson, & Skodol, 2012). These five traits (negative affectivity, detachment, antagonism, disinhibition, and psychoticism) are operationalized using the personality inventory for the *DSM-5* (PID-5; APA, 2013; Krueger et al., 2012), and research shows these dimensions are largely consistent with other pathological trait models (Van den Broeck et al., 2014; Wright & Simms, 2014), normal personality trait models (Helle, Trull, Widiger, & Mullins-Sweatt, 2016; Suzuki, Samuel, Pahlen, & Krueger, 2015; Thomas et al., 2013), RDoC constructs (Harkness et al., 2014), and appear to capture aspects of the internalizing, externalizing, and thought disorder spectra as well (Wright & Simms, 2015).

Criterion A describes impairments to self and interpersonal functioning and is thought to capture the common impairments in all patients exhibiting personality pathology (Bender, Morey, & Skodol, 2011). In doing so, this description of impairment arguably improves upon the generic distress and/or disability criteria often used in *DSM-5* diagnostic sets. Criterion A is operationalized using the Level of Personality Functioning Scale (LPFS; APA, 2013), where aspects of self-impairment (identity and self-direction impairment) and interpersonal impairment (empathy and

intimacy impairment) are organized along a single dimension of functioning.

The AMPD model thus improves upon the many limitations of the *DSM-IV* PD system (Morey, Skodol, & Oldham, 2014; Skodol, 2012), and also proposes a model that could be used more broadly to capture the structure of psychopathology. However, some researchers wonder whether Criterion A can (or should) be distinguished from Criterion B, because both share certain conceptual redundancies. For instance Criterion B identifies the trait of negative affectivity, which is conceptually similar to aspects of Criterion A identity impairment related to emotion regulation. Criterion B identifies the trait of detachment, which is conceptually similar to the aspect of Criterion A intimacy impairment. Some have proposed that Criterion A is thus redundant with Criterion B, and that perhaps Criterion A is unnecessary.

There is limited research examining whether Criterion A and B provide incremental validity in association with other relevant constructs and outcomes. Only two studies directly examined the AMPD using the LPFS and PID-5 measures from the *DSM-5*. Few et al. (2013) examined 109 patients with self-ratings of the PID-5 and clinician ratings of the LPFS and PID-5. Using only clinician ratings, Few et al. (2013) found the LPFS explained a significant amount of variance in *DSM-IV* PDs when entered alone, but failed to increment once the PID-5 traits were added to the model. Zimmerman et al. (2015) used informant ratings from community participants and therapists to evaluate the latent structure of the LPFS and PID-5. They found support for a bifactor structure of the LPFS, with a large general factor representing the overall severity of personality impairment. Evaluating the LPFS and PID-5 together, they found evidence for seven factors, the first two representing self and interpersonal impairment, and the remaining five roughly corresponding to the PID traits. However, they noted that several LPFS and PID traits had primarily loadings on unexpected factors, suggesting it is difficult to distinguish Criterion A and B. The incremental validity of Criterion A and B was not examined.

Although other research has examined the incremental validity of Criterion A and Criterion B, these studies substitute different proxy measures for the LPFS and/or PID-5, making it difficult to confidently generalize to the AMPD. For instance, Hopwood, Thomas, Markon, Wright, and Krueger (2012) measured Criterion A through a composite of the Personality Diagnostic Questionnaire (PDQ-4 items, self-report) and demonstrated some modest incremental validity over the PID-5 in predicting personality disorders. Hentschel and Pukrop (2014) used self-report measures representing alternative measures of Criterion A (GAPD; Livesley, 2006) and Criterion B (DAPP-BQ; Livesley & Jackson, 2009), finding Criterion A did not increment Criterion B in predicting personality disorder severity. Similarly, Bastiaansen et al. (2015) used self-report measures of Criterion A (SIPP-118; Verheul et al., 2008) and Criterion B (DAPP-BQ), to predict personality disorders, finding that criterion A incremented an average of 2% additional variance, and was insignificant for five of the 10 personality disorders. Finally, Creswell, Bachrach, Wright, Pinto, and Ansell (2016) also used the self-report GAPD and the PID-5, finding that problematic alcohol use was initially predicted by the GAPD, but was no longer significant once the PID-5 was added to the model. In short, there is limited evidence that Criterion A increments Criterion B in cross-sectional studies.

Dynamics

The alternative model suggests that patterns of personality dysfunction are relatively stable across time and circumstances, which is somewhat inconsistent with the extant literature that observes personality disordered patients demonstrating variability in self, social, and emotional difficulties (e.g., Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007; Wright, Hopwood, & Simms, 2015). Despite the broad assertion of stability, the individual descriptions found in Criterion A do suggest some modest instability in self-image (e.g., may show overidentification with others, overemphasis on independence, or vacillation between these), emotion regulation (e.g., emotions may be rapidly shifting or chronically despaired), and interpersonal connectedness (e.g., feelings about intimate involvement with others alternate between fear/rejection and desperate desire for connection). Thus, it will be important to examine how stable or unstable these features of personality psychopathology actually are.

As previously mentioned, the incremental validity of Criterion A and B is questionable, but prior research has primarily focused on how these measures predict cross-sectional variance in other trait based measures. The use of trait-based measures as outcomes is limited as it does not capture much of “life as it is lived” (Allport, 1942, p. 56). Thus, using longitudinal data might provide greater nuance in detecting the incremental validity of Criterion A and B to predict dynamic outcomes and context-symptom linkages.

The Present Study

The broad aim of this research is to articulate how the structure and dynamics of psychopathology can be integrated to predict impairment, using daily ratings across 2 weeks in the participants’ lives. Using the AMPD model, we also consider how such integration can illuminate the incremental validity of Criterion A and B that was not detected using single-occasion data and analyses. The specific analyses can be organized into Criterion A and B predicting variability of personality impairment, day-to-day fluctuations of personality impairments, and severity of personality impairments. We also examine how daily life triggers (e.g., emotions and cognitive distortions) exacerbate personality impairment, and whether Criterion A and B uniquely strengthen (i.e., moderate) those within-person associations.

Variability

Using a common method, we capture variability for each individual across their time series, the intraindividual standard deviation, or iSD. Our first hypothesis is that Criterion A and B will be associated with increased variability of personality impairments at the aggregate (iSD) level, and will increment each other.

Fluctuations

One limitation of an iSD is that it aggregates across the entire data series to form a summary statistic that may obscure fluctuations across days. We implement a new method using autocorrelations in a differential time-varying effect model to examine how a day with personality impairment impacts subsequent days. We use an exploratory framework and thus make no hypothesis about

the pattern of fluctuation, but our second hypothesis is that the fluctuations will be incrementally predicted by Criterion A and B.

Severity

Retrospective self-reports may contain biases that distort the participant's true experience. Therefore, taking an average of personality impairments reported daily over 2 weeks may be more accurate than collecting the same information using a single retrospective self-report. Our third hypothesis is that Criterion A and B will increment each other in predicting the severity of personality impairments experienced over 2 weeks.

Triggers

The cognitive-affective processing system model (CAPS; Mischel & Shoda, 1995) considers how personality patterns are shaped by tendencies toward particular cognitions and affective experiences. This model was extended to explain how personality impairment symptoms can be shaped by contexts (Eaton, South, & Krueger, 2009) such as daily hassles (Jarneck, Miller, & South, 2015), daily stress (Wright et al., 2015), rejection and abandonment (Miskewicz et al., 2015), and interpersonal perceptions (Roche, Pincus, Hyde, Conroy, & Ram, 2013; Wang, Roche, Pincus, Conroy, Rebar, & Ram, 2014). Consistent with the CAPS model, both psychodynamic (Yeomans, Clarkin, & Kernberg, 2015) and cognitive-behavioral (Beck, Davis, & Freeman, 2015; Linehan, 1993; Linehan, 2015) therapies for personality disorders highlight the importance of negative emotions and cognitive distortions as triggers that exacerbate personality impairment. Thus, our fourth hypothesis is that (a) daily negative emotions and cognitive distortions will be positively associated with personality impairment, and (b) Criterion A and B will moderate the strength of those associations.

Although our hypotheses are explicitly organized around the incremental validity of Criterion A and B, we also consider how the patterns found in Criterion B (representing the larger structure of psychopathology) could be more broadly informative for connecting between-person structures of psychopathology to the within-person patterns of impairment experienced in daily life that are often the focus of treatment.

Method

Participants and Procedure

Participants were 248 adult ($M_{\text{age}} = 19.4$) college students (51% male). The majority of the sample identified as Caucasian (77%), with fewer identifying as Asian (9.7%), African American (3.6%), and Hispanic/Latin (2.8%). All participants agreed to participate in the study procedures (i.e., completing a baseline survey and 14 nightly diary records online) in exchange for course credit and a personalized summary of their responses at the conclusion of the study. After completing the baseline surveys, participants were sent an e-mail inviting them to begin their nightly diary records (completed online through a web site). Thereafter, participants were sent an e-mail reminder each night (between 5 p.m. and 8 p.m.) to complete the nightly diary record (n records = 3,479).

AMPD Measures

The Level of Personality Functioning Scale (LPFS; APA, 2013) is a 12-item measure that describes failures in self functioning such as identity (three items) and self-direction (three items), as well as failures in interpersonal functioning such as empathy (three items) and intimacy (three items). Items are rated along a 5-point scale, with unique descriptions attached to each point along the scale. Previous researchers have translated this assessment tool into 60 distinct German language items (Zimmerman et al., 2015); however, the *DSM-5* describes the LPFS in 12 distinct groupings, which we retain here to more directly map onto the *DSM-5*. Because the LPFS was designed to represent a single score, we take the average of the relevant scales ($\alpha = .83$). To remain as close to the *DSM-5* defined AMPD model as possible, this measure was constructed by copying the measure verbatim from the LPFS published in the *DSM-5*. Previous research demonstrated that undergraduate students can reliably utilize the LPFS to rate patients, and that these student ratings reliably detect the presence or absence of personality disorders (Zimmermann et al., 2014). It should be noted that a different LPFS brief form has recently been published (Hutsebaut, Feenstra, & Kamphuis, 2016). This measure was designed as a self-report, and is briefer than the original LPFS in that it changes the response format to a dichotomous yes or no response scale, and replaces the multisentence descriptions of the 12 aspects with just a single sentence. There is preliminary evidence for its reliability and validity, and this measure may well be a more efficient way of capturing the LPFS in self-report form (although some of the clinical nuance is lost by deleting the different clinical descriptions across each 0–4 rating point).

The Personality Inventory for *DSM-5* (PID-5; APA, 2013; Krueger et al., 2012) is a 220-item questionnaire with a 4-point response scale, resulting in 25 pathological traits. The five trait domains are calculated using only 15 of the pathological traits (three traits per domain), and because we planned to only use the domain scores, we collected only the 15 lower level traits necessary (resulting in 123 items to form the 15 traits). These five broad domains of personality trait pathology included negative affectivity ($\alpha = .83$), detachment ($\alpha = .82$), antagonism ($\alpha = .71$), disinhibition ($\alpha = .75$), and psychoticism ($\alpha = .79$). Research continues to emerge supporting the replicability of this five factor structure (Morey, Krueger, & Skodol, 2013; Wright et al., 2012), and providing evidence for criterion validity of the PID-5 (Dowdillo, Ménard, Krueger, & Pincus, 2016; Few et al., 2013; Hopwood, Thomas, Markon, Wright, & Krueger, 2012). As a supplemental analysis, we also average together the five PID traits to evaluate whether this single score ($\alpha = .80$) is more effective than the LPFS in predicting personality impairments (see also Somma, Fossati, Borroni, Markon, & Krueger, 2016).

Daily Measures

At the daily level, four items were chosen to represent aspects of personality impairment. All were answered on a -4 to $+4$ continuum using one point increments and including a zero point. The items captured impairment in identity ("Today my self-esteem was:" -4 low, $+4$ high), self-direction ("Today I was able to be myself despite pressure from others:" -4 strongly disagree, $+4$ strongly agree), empathy ("Today I understood (empathized with) other people's feelings and circumstances:" -4 strongly dis-

agree, +4 *strongly agree*), and intimacy (“Today I felt like other people cared about me:” -4 *strongly disagree*, +4 *strongly agree*). All items were rescored in the direction of pathology to facilitate interpretation. These daily items directly correspond to descriptions of self and interpersonal impairment noted in the *DSM-5*.

We also assessed daily affects, specifically depression (“Overall, today I felt:” -4 *depressed*, +4 *happy*), anxiety (“Overall, today I felt:” -4 *anxious*, +4 *calm*), and anger (“Overall, today I felt:” -4 *angry*, +4 *content*). These affects were also rescored in the direction of pathology. Finally, we assessed daily cognitive distortions using a dichotomous check box under the heading, “Thoughts checklist (check all that apply): Today I . . .” The distortions included magnification (*dwelled on a single negative event*), harsh critic (*was overly critical of myself*), and fortune telling (*assumed things would go badly for no reason*).

Statistical Analyses

Variability. We calculated aggregate variability by taking a within-person standard deviation (iSD) for the four outcomes, aggregating up so each individual has one variability score (just as they have one score for LPFS and PID traits). Because all scores were then at the between-person level, we calculated a series of hierarchical linear regressions where variability scores were regressed on the within-person mean of the outcome variable, the LPFS, and then the PID traits.

Fluctuations. To examine fluctuations in greater depth, we used a differential time-varying effect model (DTVEM), which is a multistage exploratory and confirmatory tool to inspect and diagnose optimal time lagged relationships within the generalized additive mixed model (GAMM) framework (Jacobson, Chow, & Newman, 2016). Our analysis uses DTVEMs to examine how personality impairment on a given day predicts fluctuations of personality impairment up to 7 days from then. This tells us about the ripple effect of a single day with personality impairment. We first examined the general pattern of the personality impairments across seven days, and then examined how LPFS and PID traits exacerbate or stabilize these fluctuations (see online supplemental materials for full details of model estimation).

Severity. This analysis examined how the overall severity of personality impairments across 14 days were associated with the LPFS and PID traits. Simply averaging each participant’s 14 days together would not account for error at the between and within-person levels. Instead, for each of the four outcomes we employed a multilevel model in SAS 9.3 (PROC MIXED; Littell, Miliken, Stoup, & Wolfinger, 1996), where the intercept represented the average score (severity) of the outcome for the sample, while simultaneously modeling error at the between and within-person level. We then added Criterion A and B measures (separately, and then together) to examine how each were associated with individual differences in personality impairment severity (see online supplemental materials for full equations and details). All level-2 variables (LPFS and PID traits) were sample centered to facilitate interpretation toward the prototypical participant.

Triggers. We employed multilevel models in SAS 9.3 (PROC MIXED) with missing data treated as missing at random to examine Criterion A and B moderating the within-person associations of emotions/cognitions and personality impairments. Specifically,

each of the four outcomes were predicted by depression, anxiety, and anger at level-1. These within-person associations were interacted with the LPFS and PID traits to test for cross-level moderations. Significant interactions were evaluated at 11 *SD* (or 0, 1 for dichotomous variables), and slope tests were calculated (Preacher, Curran, & Bauer, 2006). The second set of models was similar, except the three negative emotions were replaced with the three cognitive distortions. Following standard multilevel practice, all repeated measures were separated into a between-person component (within-person means across the repeated measures) and a within-person component (moment-to-moment fluctuations around those averages; Hofmann & Gavin, 1998; Schwartz & Stone, 1998). For the analysis, all between-person variables were sample-centered to facilitate interpretation of model parameters as representative to the prototypical participant. All nondichotomous within-person variables were person-centered to facilitate interpretation as fluctuations from that person’s average score. Dichotomous variables remained scored as zero or one to facilitate interpretation of the cognitive distortion as present or absent (see online supplemental materials for further details and statistical model).

Results

Descriptive statistics for the variables are listed in Table 1. The between-person variances ranged from 38% to 51%. The LPFS and PID traits had average scores toward the lower end of their respective scales. The LPFS was significantly correlated with the PID traits of negative affectivity, $r = .53, p < .05$; detachment, $r = .60, p < .05$; antagonism, $r = .25, p < .05$; disinhibition, $r = .52, p < .05$; and psychoticism, $r = .38, p < .05$. The strength of these associations are comparable to prior studies (e.g., Few et al., 2013), although this is the first to correlate self-report measures of the *DSM-5* Criteria A and B scales.

Table 1
Descriptive Statistics for Variables in Study

Variables	<i>M</i>	<i>SD</i>	<i>ICC</i>
Within-person			
Identity (ID)	-1.70	2.01	.51
Self-direction (SD)	-1.23	2.39	.25
Empathy (EPY)	-1.44	2.00	.39
Intimacy (INT)	-1.47	2.35	.38
Depressed (DEP)	-1.58	2.05	.42
Anxious (ANX)	-1.03	2.34	.41
Angry (ANG)	-1.78	1.99	.38
Magnification (MAG)	.26	.44	.31
Harsh critic (HC)	.28	.45	.35
Fortune telling (FT)	.13	.34	.24
Between-person			
Level of Personality Functioning Scale (LPFS)	.77	.58	
Negative affectivity (NA)	1.15	.64	
Detachment (DET)	.70	.51	
Antagonism (ANT)	.78	.45	
Disinhibition (DIS)	.72	.49	
Psychoticism (PSY)	.68	.48	

Note. Scores reported before centering, but after scoring in the direction of pathology. Continuous variables represented on -4 to +4 scale with zero point.

Variability

The variability results are presented in Table 2. As is common, the within-person mean was positively associated with the within-person variability for all models except self-direction, meaning a participant who reported higher average impairments tended to have more lability in those impairments across 14 days. After accounting for the within-person mean, the LPFS and PID traits explain a small amount of additional between-person variance for identity impairment (1%), self-direction impairment (9%), empathy impairment (<1%), and intimacy impairment (2%). The LPFS, when entered alone, was significant for self-direction impairment ($b = -0.21, p < .05$) and intimacy impairment ($b = -0.15, p < .05$), but was no longer significant once the PID traits were added into the model. Higher detachment scores were associated with lower variability in identity impairment ($b = -0.16, p < .05$) and self-direction impairment ($b = -0.28, p < .05$), and similarly higher disinhibition scores were associated with lower variability in self-direction impairment ($b = -0.19, p < .05$). In contrast, higher psychoticism scores were associated with higher variability in intimacy impairment ($b = 0.16, p < .05$).

Fluctuations

The results for the DVTEM moderation analyses are presented in Table 3. The general pattern of fluctuation appears to be a day-to-day oscillating pattern, where impairment today predicts more impairment tomorrow, less impairment that day after, more impairment the day after that, and so forth. Specifically, the general pattern is to find a positive autocorrelation on Days 1, 3, and 5 and a negative autocorrelation on Days 2, 4, and 6. For identity impairment, a positive autocorrelation is found for Days 1, 3, and 5, and a negative autocorrelation found for Days 4 and 6. Thus this generally fits the oscillating pattern described above (except Day 2 was not significant). For self-direction, Day 3

Table 2
Variability Models for Daily Personality Impairment

Variables	Identity <i>b</i>	Self-direction <i>b</i>	Empathy <i>b</i>	Intimacy <i>b</i>
Initial model				
iMN	.48	.09	.31	.41
Adj. R^2	.23	.00	.09	.17
Criterion A				
iMN	.48	.16	.30	.48
LPFS	.00	-.21	.04	-.15
Adj. R^2	.22	.04	.09	.18
Criterion A and B				
iMN	.48	.20	.32	.51
LPFS	.02	.00	.03	-.10
NA	.10	.01	.13	-.08
DET	-.16	-.28	-.12	-.14
ANT	-.07	.03	.04	.03
DIS	-.02	-.19	-.02	-.02
PSY	.15	.08	-.01	.16
Adj. R^2	.24	.09	.09	.19

Note. iMN = intraindividual mean; LPFS = level of personality functioning scale; NA = negative affectivity; DET = detachment; ANT = antagonism; DIS = disinhibition; PSY = psychoticism. Standardized beta coefficients reported. Bold = $p < .05$. Adj. R^2 reported with bold indicating the step was significant at $p < .05$.

Table 3
Moderation on the Temporal Stability of Personality Impairment

Variables	Identity		Self-direction		Empathy		Intimacy	
	Est.	SE	Est.	SE	Est.	SE	Est.	SE
Autoregressions								
Lag 1	.57	.03			.76	.03	.73	.02
Lag 2					-.46	.02	-.41	.02
Lag 3	.13	.03	.10	.02				
Lag 4	-.21	.03	-.06	.02				
Lag 5	.19	.02						
Lag 6	-.15	.01	-.14	.01	-.08	.01		
Moderators								
LPFS × Lag 1			.23	.03	.10	.03		
LPFS × Lag 2	-.09	.03	-.36	.03	-.06	.02	-.12	.02
LPFS × Lag 3	.08	.02	.34	.03				
LPFS × Lag 4			-.29	.03				
LPFS × Lag 5			.20	.02				
LPFS × Lag 6							-.05	.02
NA × Lag 1	.10	.02	.29	.02				
NA × Lag 2	-.10	.02	-.12	.02				
NA × Lag 3					.12	.02	.18	.02
NA × Lag 4							-.08	.02
DET × Lag 1							.08	.02
DET × Lag 4					-.09	.02	-.11	.03
DET × Lag 5					.12	.02	.10	.03
ANT × Lag 1	.11	.03						
ANT × Lag 2	-.31	.03						
ANT × Lag 3	.20	.03			.13	.03		
ANT × Lag 4	-.11	.03			-.08	.03		
DIS × Lag 3					.09	.03		
DIS × Lag 4					-.09	.03		
DIS × Lag 5							.07	.02
PSY × Lag 1			.24	.03	-.07	.02		
PSY × Lag 2			-.19	.03				
PSY × Lag 3							.14	.03
PSY × Lag 4							-.08	.03
PSY × Lag 6							-.08	.02

Note. Est. = unstandardized estimates of sample parameters; SE = standard error; LPFS = level of personality functioning scale; NA = negative affectivity; DET = detachment; ANT = antagonism; DIS = disinhibition; PSY = psychoticism. Note that many lag times and variables are not displayed as they were not estimated in the confirmatory stage 2 of DVTEM. Bold = $p < .05$.

(positive autocorrelation), Day 4 (negative autocorrelation), and Day 6 (negative autocorrelation) were significant, and had a similar oscillating pattern that appeared to be delayed until Day 3. Conversely, empathy and intimacy impairment evidenced the oscillating pattern for Day 1 (positive autocorrelation) and Day 2 (negative autocorrelation) only. Notably, all significant lag coefficients consistently exhibited day-to-day oscillating patterns.

Several LPFS and PID traits moderated these associations, and in all cases, the moderations *exacerbated* the oscillating patterns. That is, every moderation that existed on Days 1, 3, and 5 was a positive coefficient (i.e., exacerbating a positive autocorrelation), and every moderation that existed on Days 2, 4, and 6 was a negative coefficient (i.e., exacerbating a negative autocorrelation). The most common moderator was the LPFS (11), followed by negative affectivity (seven), antagonism (six), detachment (five), psychoticism (five), and disinhibition (three).

Identity impairment oscillations were exacerbated for Days 1–2 (negative affectivity), Days 2–3 (LPFS), and Days 1–4 (antagonism). Self-direction impairment oscillations were exacerbated for

Days 1–5 (LPFS) and Days 1–2 (NA, PSY). Empathy impairment oscillations were exacerbated for Days 1 (PSY), 1–2 (LPFS), 3 (NA), 3–4 (ANT, DIS). Intimacy impairment oscillations were exacerbated for Days 1 and 4–5 (DET), 2 and 6 (LPFS), 3–4 (NA), 5 (DIS), and 3–4, and 6 (PSY).

Comparing LPFS moderations with the PID traits, the same pattern for Days 1–2 appear with LPFS and antagonism on identity impairment oscillations. A different pattern emerges for self-direction impairment, whereby the LPFS predicts moderations longer (through Day 5) compared with other traits (not significant after Day 2). The LPFS moderated empathy impairment earlier (Days 1–2) compared with the PID traits. Finally, the LPFS is the only moderator for Day 2 of intimacy impairment.

Severity

The severity results are presented in Table 4. When the LPFS is used alone, it is significantly associated with all four outcomes, explaining between 13% and 23% of between-person variance. When the PID traits are used alone, at least one trait is significantly associated in each model, explaining between 14% and 37% of between-person variance. A higher PID trait score was usually associated with greater personality impairment (except for antagonism decreasing identity impairment, and psychoticism decreasing intimacy impairment). When the LPFS and PID traits were modeled together the LPFS remained significant and explained additional between-person variance beyond the PID traits for identity (2%), self-direction (2%), empathy (5%), and intimacy (5%). Similarly, the PID traits explained additional between-person variance beyond the LPFS for identity (16%), self-direction (3%), empathy (5%), and intimacy (3%). Some have argued that an average of the PID domains could instead be used in place of the LPFS (Somma et al., 2016). However, when scored this way, the PID average score explained only half the variance compared to the five PID traits entered separately (see Table 2), and failed to increment the LPFS in self-direction impairments, empathy impairments, and intimacy impairments.

Triggers

The results for the emotional triggers are presented in Table 5. Higher daily depression and daily anger increased personality impairments. Thus, for the typical participant, days when they reported being sadder and angrier compared with their usual day tended to coincide with reporting more personality impairments than usual. Several of these within-person associations were moderated by LPFS and PID traits.

There were no moderations for identity impairment. For self-direction impairment, days with higher depression increased self-direction impairment only for those with higher Psychoticism scores ($b = 0.29, p < .05$), relative to lower scores ($b = 0.09, p > .05$). Days with higher anxiety increased self-direction impairment only for those with higher LPFS scores ($b = 0.11, p < .05$), relative to lower scores ($b = -0.06, p > .05$). Days with higher anger increased self-direction impairment only for those with low antagonism scores ($b = 0.23, p < .05$), relative to higher scores ($b = 0.01, p > .05$).

There were no moderations for empathy impairment. For intimacy impairment, days with higher depression increased intimacy

Table 4
Associations With Severity of Personality Impairments

Variables	Identity		Self-direction		Empathy		Intimacy	
	Est.	SE	Est.	SE	Est.	SE	Est.	SE
LPFS only								
Intercept, γ_{00}	-1.70	.08	-1.22	.08	-1.44	.08	-1.47	.09
LPFS, γ_{01}	1.20	.14	.75	.14	.94	.13	1.12	.15
Level 1 error, e_{di}	1.98	.05	4.29	.11	2.45	.06	3.42	.09
Level 2 error, u_{oi}	1.60	.16	1.23	.14	1.27	.13	1.69	.17
Pseudo R^2	.23		.13		.19		.20	
PID-5 only								
Intercept, γ_{00}	-1.70	.08	-1.22	.08	-1.44	.08	-1.47	.09
NA, γ_{01}	.83	.15	.43	.15	-.07	.15	.23	.17
DET, γ_{02}	1.18	.18	.61	.19	.92	.18	1.14	.21
ANT, γ_{03}	-.59	.21	-.22	.21	.23	.21	-.05	.24
DIS, γ_{04}	.20	.23	.27	.23	.49	.23	.35	.26
PSY, γ_{05}	-.44	.23	-.38	.23	-.32	.23	-.54	.26
Level 1 error, e_{di}	1.98	.05	4.29	.11	2.45	.06	3.42	.09
Level 2 error, u_{oi}	1.31	.13	1.22	.14	1.27	.13	1.73	.18
Pseudo R^2	.37		.14		.19		.18	
LPFS and PID5								
Intercept, γ_{00}	-1.70	.08	-1.22	.08	-1.44	.07	-1.47	.09
LPFS, γ_{01}	.49	.18	.42	.18	.64	.18	.78	.21
NA, γ_{02}	.72	.15	.33	.16	-.22	.15	.05	.18
DET, γ_{03}	.96	.20	.43	.20	.63	.19	.78	.23
ANT, γ_{04}	-.57	.20	-.21	.21	.25	.20	-.02	.23
DIS, γ_{05}	.06	.23	.15	.24	.31	.23	.12	.26
PSY, γ_{06}	-.40	.22	-.35	.23	-.27	.22	-.47	.26
Level 1 error, e_{di}	1.98	.05	4.29	.11	2.45	.06	3.42	.09
Level 2 error, u_{oi}	1.28	.13	1.19	.14	1.20	.13	1.62	.17
Pseudo R^2	.39		.16		.24		.23	
PID average only								
Intercept, γ_{00}	-1.70	.09	-1.22	.08	-1.44	.08	-1.47	.09
PID average, γ_{01}	1.50	.23	.86	.21	1.15	.20	1.16	.24
Level 1 error, e_{di}	1.98	.05	4.29	.11	2.45	.06	3.42	.09
Level 2 error, u_{oi}	1.75	.17	1.31	.15	1.37	.14	1.91	.19
Pseudo R^2	.16		.07		.12		.09	
PID avg. and LPFS								
Intercept, γ_{00}	-1.70	.08	-1.22	.08	-1.44	.08	-1.47	.09
LPFS, γ_{01}	.94	.18	.64	.17	.75	.17	1.04	.19
PID average, γ_{02}	.62	.27	.27	.26	.46	.25	.20	.29
Level 1 error, e_{di}	1.98	.05	4.29	.11	2.45	.06	3.32	.09
Level 2 error, u_{oi}	1.57	.15	1.23	.14	1.26	.13	1.69	.18
Pseudo R^2	.25		.13		.20		.20	

Note. LPFS = level of personality functioning scale; NA = negative affectivity; DET = detachment; ANT = antagonism; DIS = disinhibition; PSY = psychoticism; PID average = average of five PID domains; Est. = unstandardized estimates of sample parameters; SE = standard error. Pseudo r^2 calculated as reduction in level-2 error from an empty model, divided by error in the empty model. Bold = $p < .05$.

impairment, and this association was strengthened for those with lower disinhibition scores ($b = 0.36, p < .05$), relative to higher scores ($b = 0.14, p < .05$). This effect was most striking at the lower end of depression (i.e., days with less sadness), where those who are not disinhibited appear to report less intimacy problems compared with those who are more disinhibited.

The results for cognitive distortion triggers are presented in Table 6. Unlike the emotion triggers, cognitive distortions evidenced a more diverse pattern with all three distortions increasing identity impairment, only magnification increasing self-direction impairment, magnification and fortune telling increasing both empathy impairment and intimacy impairment. However, several moderations of these day-to-day associations existed.

Table 5
Daily Emotional Contexts Influencing Personality Impairments

Variables	Identity		Self-direction		Empathy		Intimacy	
	Est.	SE	Est.	SE	Est.	SE	Est.	SE
Level 1								
DEP, γ_{10}	.45	.02	.19	.04	.13	.03	.25	.03
ANX, γ_{20}	.02	.02	.03	.03	-.01	.02	-.02	.02
ANG, γ_{30}	.14	.02	.12	.04	.16	.03	.09	.03
Level 2								
Intercept, γ_{00}	-1.70	.04	-1.22	.07	-1.44	.07	-1.48	.07
DEP μ , γ_{01}	.87	.007	.05	.11	.16	.11	.52	.12
ANX μ , γ_{02}	.10	.06	-.04	.09	-.05	.08	-.23	.09
ANG μ , γ_{03}	-.09	.08	.50	.12	.42	.11	.36	.13
LPFS, γ_{04}	.01	.11	.15	.17	.37	.16	.45	.18
NA, γ_{05}	.06	.10	.01	.16	-.54	.14	-.30	.16
DET, γ_{06}	.18	.12	.09	.19	.27	.18	.29	.20
ANT, γ_{07}	-.29	.12	-.12	.19	.33	.18	.14	.20
DIS, γ_{08}	.05	.14	.21	.22	.34	.20	.09	.23
PSY, γ_{09}	.04	.13	-.16	.21	-.07	.20	-.23	.22
Moderators								
DEP × LPFS, γ_{11}	-.01	.06	-.13	.09	.06	.08	.07	.09
DEP × NA, γ_{12}	.07	.05	.08	.07	-.07	.06	.11	.07
DEP × DET, γ_{13}	.05	.06	-.01	.09	.05	.08	.05	.09
DEP × ANT, γ_{14}	-.07	.06	.15	.09	.09	.07	.13	.08
DEP × DIS, γ_{15}	-.03	.07	-.14	.10	-.02	.08	-.22	.09
DEP × PSY, γ_{16}	.01	.07	.21	.10	-.09	.09	-.05	.10
ANX × LPFS, γ_{21}	-.02	.04	.15	.06	-.04	.05	.05	.06
ANX × NA, γ_{22}	.05	.03	-.09	.05	-.03	.04	-.03	.05
ANX × DET, γ_{23}	-.02	.04	-.09	.07	.05	.05	.09	.06
ANX × ANT, γ_{24}	.06	.04	-.07	.07	-.03	.05	-.02	.06
ANX × DIS, γ_{25}	-.06	.05	.08	.08	.00	.06	.06	.07
ANX × PSY, γ_{26}	.02	.04	.02	.08	.04	.06	-.13	.07
ANG × LPFS, γ_{31}	.02	.05	.11	.09	.05	.07	-.06	.08
ANG × NA, γ_{32}	-.07	.04	-.04	.08	.10	.06	.11	.07
ANG × DET, γ_{33}	-.02	.05	-.07	.10	-.14	.07	-.14	.08
ANG × ANT, γ_{34}	-.09	.05	-.24	.09	-.08	.07	-.08	.08
ANG × DIS, γ_{35}	.07	.06	.00	.10	.11	.08	.10	.09
ANG × PSY, γ_{36}	.09	.06	.05	.10	-.06	.08	.07	.09
Random effects								
Level 1 error, e_{di}	.89	.02	3.91	.10	2.10	.06	3.00	.08
Level 2 error, u_{0i}	.43	.05	.94	.11	.90	.10	1.13	.12
Level 2 error, u_{1i}	.05	.01	0		.03	.01	.03	.01
Level 2 error, u_{2i}	.01	.00	.01	.01	0		0	
Level 2 error, u_{3i}	.02	.01	.04	.02	.03	.01	.02	.01

Note. Est. = unstandardized estimates of sample parameters; SE = standard error; LPFS = level of personality functioning scale; NA = negative affectivity; DET = detachment; ANT = antagonism; DIS = disinhibition; PSY = psychoticism; DEP = daily level depression; ANX = daily level anxiety; ANG = daily level anger. Parameters person-centered (level-1), sample-centered (level-2). Some models required level-2 variances set to 0 to converge. Bold = $p < .05$.

For identity impairment, the magnification distortion increased identity impairment, and this association was strengthened for those with higher negative affectivity scores ($b = 1.02, p < .05$), relative to lower scores ($b = 0.44, p < .05$). The harsh critic distortion increased identity impairment, and this association was strengthened for those with lower antagonism scores ($b = 0.80, p < .05$), relative to higher scores ($b = 0.43, p < .05$). The fortune telling distortion increased identity impairment only for those with higher LPFS scores ($b = 1.04, p < .05$), relative to lower scores ($b = 0.28, p > .05$), and only for those with lower detachment scores ($b = 0.97, p < .05$), relative to higher scores ($b = 0.34, p > .05$).

For self-direction impairment, the harsh critic distortion increased self-direction impairment for those with lower antagonism scores ($b = 0.47, p < .05$), relative to higher scores ($b = -0.07, p > .05$). For empathy impairment, the magnification distortion increased empathy impairment only for those with higher disinhibition scores ($b = 0.66, p < .05$), relative to lower scores ($b = -0.02, p > .05$) and only for those with lower psychoticism scores ($b = 0.59, p < .05$), relative to higher scores ($b = 0.05, p > .05$).

For intimacy impairment, the magnification distortion increased intimacy impairment only for those with higher negative affectivity scores ($b = 0.62, p < .05$), relative to lower scores ($b = 0.06, p > .05$). The harsh critic distortion increased intimacy impairment

Table 6
Daily Cognitive Distortion Contexts Influencing Personality Impairments

Variables	Identity		Self-direction		Empathy		Intimacy	
	Est.	SE	Est.	SE	Est.	SE	Est.	SE
Level 1								
MAG, γ_{10}	.73	.08	.34	.11	.32	.10	.34	.11
HC, γ_{20}	.62	.08	.20	.11	.22	.09	.18	.10
FT, γ_{30}	.66	.12	.18	.14	.22	.13	.37	.14
Level 2								
Intercept, γ_{00}	-2.17	.07	-1.41	.08	-1.60	.08	-1.70	.09
LPFS, γ_{01}	.45	.17	.35	.20	.69	.19	.81	.21
NA, γ_{02}	.39	.15	.20	.17	-.30	.16	-.33	.18
DET, γ_{03}	.89	.19	.39	.22	.55	.21	.67	.24
ANT, γ_{04}	-.24	.20	.14	.23	.32	.22	.06	.25
DIS, γ_{05}	-.01	.23	.18	.26	.16	.24	.32	.28
PSY, γ_{06}	-.52	.22	-.59	.25	-.09	.24	-.50	.27
Moderators								
MAG × LPFS, γ_{11}	-.29	.19	.04	.25	-.31	.22	-.08	.25
MAG × NA, γ_{12}	.45	.16	.21	.22	-.10	.19	.44	.22
MAG × DET, γ_{13}	.13	.20	-.08	.26	.29	.23	.01	.26
MAG × ANT, γ_{14}	-.35	.20	-.26	.27	-.05	.23	.02	.27
MAG × DIS, γ_{15}	.11	.22	.02	.30	.70	.26	.11	.30
MAG × PSY, γ_{16}	.16	.23	.31	.31	-.57	.27	-.45	.31
HC × LPFS, γ_{21}	-.12	.21	.07	.26	-.08	.24	-.38	.24
HC × NA, γ_{22}	-.09	.17	-.13	.22	.19	.19	.52	.19
HC × DET, γ_{23}	.17	.21	.02	.26	-.01	.23	.40	.24
HC × ANT, γ_{24}	-.42	.20	-.60	.26	-.02	.23	.01	.23
HC × DIS, γ_{25}	.18	.24	.17	.31	-.21	.27	-.33	.28
HC × PSY, γ_{26}	-.05	.22	.33	.29	-.10	.25	.19	.26
FT × LPFS, γ_{31}	.66	.29	-.02	.34	.33	.31	.11	.35
FT × NA, γ_{32}	-.26	.23	.07	.26	-.36	.24	.09	.27
FT × DET, γ_{33}	-.61	.28	.22	.32	-.31	.30	-.03	.34
FT × ANT, γ_{34}	.20	.29	-.06	.32	-.31	.31	.07	.34
FT × DIS, γ_{35}	-.34	.32	-.62	.37	.00	.34	-.74	.38
FT × PSY, γ_{36}	.28	.31	.17	.36	.14	.34	.40	.37
Random Effects								
Level 1 error, e_{di}	1.56	.04	4.21	.11	2.25	.06	3.21	.08
Level 2 error, u_{0i}	1.10	.12	1.19	.14	1.22	.13	1.56	.17
Level 2 error, u_{1i}	.37	.12	.22	.19	.47	.15	.56	.19
Level 2 error, u_{2i}	.36	.12	.04	.17	.32	.14	0	
Level 2 error, u_{3i}	.74	.21	0		.61	.23	.54	.25

Note. Est. = unstandardized estimates of sample parameters; SE = standard error; LPFS = level of personality functioning scale; NA = negative affectivity; DET = detachment; ANT = antagonism; DIS = disinhibition; PSY = psychoticism; MAG = magnification distortion; HC = harsh critic distortion; FT = fortune telling distortion. Level-2 variables sample centered. Some models required level-2 variances set to 0 to converge. Bold = $p < .05$.

only for those with higher negative affectivity scores ($b = 0.52$, $p < .05$), relative to lower scores ($b = -0.16$, $p > .05$). The fortune telling distortion increased intimacy impairment only for those with *lower* disinhibition scores ($b = 0.73$, $p < .05$), relative to higher scores ($b = 0.01$, $p > .05$).

Discussion

The current study demonstrated the utility of integrating structure and dynamic processes to inform dimensional models of psychopathology. Specifically we examined whether the *DSM-5*'s Criterion A (LPFS) and Criterion B (PID traits) offer unique information in how personality impairment is experienced in daily life. We hypothesized that (H1) LPFS and PID traits would be associated with greater variability in personality impairments, (H2) LPFS and PID traits would be associated with daily fluctuations in personality impairment, (H3) the LPFS and PID traits would increment each other in predicting the severity of personality impairments reported in daily life, (H4a) cognitive and emotional triggers would worsen personality impairment that day, and (H4b) LPFS and PID traits would strengthen those triggers in unique ways.

Hypothesis 1 (H1) was not supported, in that higher LPFS and PID traits were mostly related to *lower* variability, not higher variability. This finding is consistent with the assumptions of the *DSM-5* that elevated levels of personality dysfunction should relate to more chronic (i.e., less variability) dysfunction, however only a small amount of variance was explained. Also inconsistent with our hypothesis, the LPFS did not increment the PID traits. It might be that aggregating up to represent variability as a single score leads to imprecision that masks the more nuanced aspects of temporally dynamic fluctuations (Ram & Gerstorf, 2009). A more precise method was used for H2.

H2 was supported, in that daily fluctuations were predicted by both the LPFS and PID traits. A robust pattern of day-to-day oscillations were found for all daily measures of personality impairment. Conceptually, this suggested a day with higher personality impairment creates a ripple effect that intensifies symptoms the next day, weakens the day after, intensifies the day after that, and so forth. Finding this pattern in daily life is somewhat inconsistent with the *DSM-5* notions of stability in personality impairment, and may highlight the need for a more nuanced perspective at this daily timescale. Notably, the oscillating pattern was *always* intensified by the LPFS and PID traits. The LPFS was the most common moderator intensifying the oscillation patterns, and in several cases appeared to operate on a different timescale compared to any of the other PID traits.

H3 was supported, suggesting that both aspects of the AMPD model can capture personality impairments as they are experienced in daily life. Although the amount of additional variance explained from the LPFS was small once accounting for the PID traits, the LPFS (except for identity impairment) explains essentially the same amount of variance as all five PID traits combined. It is perhaps unsurprising that when entered together, a 123-item measure with five variables is more effective than a single score from a 12-item measure. Thus, in relative terms, the briefer LPFS self-report performed well. Also, the LPFS predicted higher personality impairment across all domains (identity, self-direction, empathy, and intimacy), suggesting this measure is effective at

capturing the full range self and interpersonal impairment in daily life. It might suggest that the LPFS could be used as an efficient screening instrument for personality impairment.

If Criterion A is to be eliminated from the AMPD, our research suggests the PID average score (Somma et al., 2016) would be a poor substitute and we do not suggest it be used in this way. When the PID traits are scored as a total score, the amount of variance explained drops by roughly 50% compared with the PID traits separately. When entered together with the LPFS, the LPFS is a stronger predictor and renders the PID average score nonsignificant (except for identity impairment). Moreover, the clinical benefits of describing patients along the five pathological traits are lost when aggregating up to a single average score.

H4a was supported in that daily personality impairments were impacted by daily emotions and cognitive distortions. H4b was supported in that the LPFS and several PID traits moderated these within-person associations, finding that many times these triggers were *only* significant or were strengthened significantly when the LPFS or PID traits were elevated. In particular, elevations on the LPFS were related to anxiety triggering self-direction impairments (when that effect was not significant on its own, or in individuals with lower LPFS scores) and fortune telling triggering identity impairments, both of which reflect a fragile self-functioning capacity. Of note, the LPFS was the only scale to moderate anxiety, highlighting one way in which the LPFS is unique from any of the PID traits. Taken together, this means the AMPD model not only predicts overall severity of personality impairments, but also can predict how daily triggers can exacerbate personality impairments (Jarneck et al., 2015; Miskewicz et al., 2015; Sadikaj, Russell, Moskowitz, & Paris, 2010; Wang et al., 2014; Wright et al., 2015).

In summary, this research is among the first to demonstrate that both Criterion A and Criterion B of the *DSM-5* AMPD predict personality impairments in daily life. Moreover, temporally dynamic analyses suggests Criterion A and B are distinct in their prediction of fluctuations at unique time points, severity, and emotional/cognitive triggers. Thus, temporally dynamic analyses may provide new avenues necessary to detect differences in Criterion A and B that are missing in cross-sectional designs.

Criterion B and the Structure of Psychopathology

If the PID-5 is a marker for the larger structure of psychopathology, then the present results provide important insights into how the structure of psychopathology relates to impairment in daily life. Harkness, Reynolds, and Lilienfeld (2014) provide a compelling adaptive systems framework where the dimensions of Criterion B are understood as representing failures in the evolutionary system of danger detection (negative affectivity, internalizing anxious disorders), resource acquisition (detachment, internalizing depressive disorders), agenda protection (antagonism, personality disorders), cost-benefit analysis (disinhibition, externalizing disorders), and reality modeling for action (psychoticism, thought disorders). Within this framework the PID traits serve as markers for larger systems of psychopathology.

The variability and fluctuation analyses reveal several unique patterns, most notably the persistent nature of self-impairment for detachment. In the variability analyses detachment related to less variability in self impairment (both identity and self-direction). The fluctuation analyses showed no moderation effects for detach-

ment on self-impairment. These findings are consistent with the trigger analyses, which showed that the fortune telling distortion did not enhance self-direction impairment for those higher in detachment because self-direction impairment was persistently higher regardless of distortions used that day. The severity analysis demonstrated that detachment was the most consistent predictor of self and interpersonal impairment and the only PID trait that was significant across all four models. Taken together, it highlights the self-sustaining nature of depressive internalizing disorders, and their broad impairment across life activities.

The trigger analyses reveal how within-person triggers of daily impairment can be exacerbated by higher levels of these dimensions of psychopathology. For instance, higher negative affectivity revealed a vicious circle whereby the magnification distortion increased identity and intimacy impairments. These findings are consistent with the notion of an aberrant danger detection system misfiring and causing life impairment through overuse, and an expected pattern among a marker of anxious laden internalizing disorder features.

The magnification distortion was related to greater empathy impairment as a main effect which may represent how distorted cognition can impact interpersonal functioning. This effect occurs with those higher in disinhibition (which may capture how short-term pain is prioritized over long-term relationship building), and is not present with those higher in psychoticism (which may reflect weaker insight into the role of their cognitions impacting relationships, and is consistent with deficits in reality modeling). Thus, these patterns reveal how different spectra of disorders (externalizing vs. psychotic) can relate to context-symptom patterns in different ways.

The Promise of Temporally-Dynamic Data to Refine the Structure of Psychopathology

The present study assumed the structure of psychopathology can be defined through static dimensions, and proceeded to examine how temporally dynamic data could inform how these static dimensions relate to patterns and outcomes over time in daily life. In contrast to cross-sectional evaluations, the current approach provided ample evidence for the discriminant validity of the *DSM-5* AMPD dimensions spanning Criteria A and B. We believe these methods hold considerable promise to articulate nuanced patterns in other psychological disorders as well. As such, it might be useful for DSM and RDoC constructs to be examined using these methods, particularly those constructs which are presumed to fluctuate at a particular timescale or be associated with specific context-symptom patterns.

However, one can also consider temporal dynamics to *define* the structure of psychopathology. For instance, Wright, Beltz, Gates, Molenaar, and Simms (2015) examined traits at the daily level, finding that a between-person structure revealed internalizing and externalizing factors, while a within-person structure revealed four factors that resembled the PID model (absent psychoticism). Others have examined how within-person variability and density distributions of personality states serve as an organizing within-person structure for personality and psychopathology (Cervone, 2005; Fleenor, 2001; Fleenor & Jayawickreme, 2015). In the *DSM*, dynamic concepts of psychopathology already exist through criteria reflecting identity diffusion and affective instability. How-

ever, the five traits that efficiently organize individuals at the between-person level may not be the constructs needed to efficiently organize patterns at the within-person level. For instance, Cain, Meehan, Roche, Clarkin, and De Panfilis (2016) suggest that at the within-person level, the relevant constructs are the self-representation, other representation, and affect system, and demonstrate how these within-person associations can illuminate patterns hidden when only analyzing between-person structures.

Another interesting extension is to define the structure of psychopathology through the triggers, that is, within-person associations among contexts and symptoms. The present study used affect and cognitive distortions as the relevant contexts given the prominence of these contexts in both psychodynamic and cognitive-behavioral theories of personality dysfunction. However, development of a taxonomy of relevant contexts (situations) producing psychological dysfunction is needed (e.g., Rauthmann, Sherman, & Funder, 2015), along with theories that specify within-person patterns of dysfunction (e.g., Eaton et al., 2009; Hopwood, Pincus, & Wright, in press; Pincus & Hopwood, 2012), and statistical methods to model how symptoms develop, coalesce into patterns, and reinforce or satiate across time (Jacobson et al., 2016; Wickham & Knee, 2013).

Ultimately, the clinician is applying their understanding of the structure of psychopathology to an individual patient. Several researchers have articulated methods to use temporally dynamic data to arrive at a patient-specific conceptualization of symptoms for depression (Wichers, 2014), depression and anxiety (Fisher & Boswell, 2016), personality pathology (Wright et al., 2015), marital discord and interpersonal problems (Roche, Pincus, Rebar, Conroy, & Ram, 2014), and substance abuse (Zheng, Cleveland, Molenaar, & Harris, 2015). However, these statistical methods are often complex and not readily adopted by practicing clinicians. We believe the next frontier in psychological diagnosis will be to develop a taxonomy of within-person patterns, and then a highly accessible assessment approach that can capture the nomothetic and idiographic clinical insights derived from such rich data (Roche & Pincus, 2016).

Limitations and Future Directions

This research has several limitations, which can be organized around participant sample, measures/constructs chosen, timescale, and analyses. Regarding the sample, using student participants that were relatively homogenous in racial composition limits the generalizability to both clinical and more diverse samples. It is possible that a more pathological sample would evince different patterns of fluctuations or variability in symptoms, making it important to examine these findings using clinical populations. However, other research suggests that structural relationships between variables do tend to generalize in clinical and nonclinical samples (O'Connor, 2002), and that late adolescence to early adulthood marks a crucial developmental time period where psychopathology may take form (Schulenberg, Sameroff, & Cicchetti, 2004).

Measures. A strength of this research was that the variables chosen for Criteria A and B directly correspond to the *DSM-5* descriptions. However, the LPFS was initially designed as a clinician rated measure, and this research would have been strengthened if we were able to capture self-reports and informant ratings

of Criteria A and B. It might also be interesting to reexamine these analyses using the more specific facets of Criterion A (i.e., scoring identity, self-direction, empathy, and intimacy separately) and Criterion B (i.e., facets rather than domain scores). As with any self-report measure, participant data is limited by the participant's insight and ability or desire to report problems. We could have also used a normal range measure for Criterion B, as research has found that the incremental validity of Criterion A predicting personality impairment increases when paired with a normal range personality trait measure (Hentschel & Pukrop, 2014). We chose against this in order to remain as close as possible to the AMPD model described in the *DSM-5*. The outcome measures chosen here also corresponded closely to Criterion A descriptions of impairment. A more thorough approach could have also included outcome variables characteristic of Criterion B (e.g., drug use for externalizing disorders represented through disinhibition, verbal arguments represented through antagonism, etc.).

Timescale. Future research could also consider collecting the LPFS and PID traits over time to observe differences in variability and prediction of daily impairments. Choosing a different timescale (e.g., moment-to-moment, weekly, monthly) could change these results and thus our findings do not generalize beyond the daily timescale. A more complete study could capture several timescales at once (e.g., Ram et al., 2014) to truly identify at which timescale(s) these processes are most relevant.

Analyses. Finally, we relied upon strong assumptions, including homogeneity of processes across individuals, and that between-person differences organize into a multivariate normal distribution in order to obtain estimates of within-person associations with so few observations. Future research should collect more than 14 assessments for each individual, which would allow for more sophisticated analyses. We also explored dozens of cross-level interactions simultaneously, and therefore the results should be taken cautiously. However, we felt running these interactions simultaneously were preferable over running dozens of separate models for each cross-level interaction. Despite these limitations, we feel that the present research is an important first step in using the self-report version of the LPFS, distinguishing Criterion A and B using ecologically valid indicators in daily life, and considering how temporally dynamic analyses can evolve our conceptions of the structure of psychopathology from static pictures to moving images.

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