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Affect Dynamics as Predictors of Symptom Severity and Treatment Response in Mood and Anxiety Disorders: Evidence for Specificity

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Abstract: Studies of affect dynamics in psychopathology often focus on the prediction of broad constructs like subjective well-being and psychological health. Less is known about how fluctuation in affect over time relates to specific symptom measures (e.g., anxiety or depression), or how these domains change in treatment. A clinical sample of 32 adults with mood and anxiety disorders (13 generalized anxiety, 5 major depression, 14 comorbid) completed four daily assessments of positive (PA) and negative affect (NA) for 30 days prior to receiving cognitive behavioral treatment. Anxiety and depression symptom severity were assessed pre- and post-treatment. We calculated three metrics of affect dynamics for each person's PA and NA time series: (1) variability (experiencing emotional extremes, the standard deviation of a person's PA or NA vector); (2) instability (magnitude of point-to-point change in emotion, the vector's mean squared successive difference); and (3) inertia (the extent to which emotions self-perpetuate over time, the lag-1 autocorrelation of the vector). Multiple regression models were run to test dynamics of positive and negative affect as between-subjects predictors of symptom severity and pre-to-posttreatment change in symptoms. Findings suggest NA dynamics are unrelated to depression symptom severity or treatment response, but we observed a specific effect of NA instability (MSSD) on both severity and response of anxiety symptoms. All PA dynamics were unrelated to anxiety or depression symptom severity. However, variability, instability, and inertia of PA were all found to relate to treatment response for both anxiety and depression symptoms. Taken together, our results suggest that affect dynamics have some specificity in their relationship to clinically relevant phenomena such as symptom severity and treatment outcomes at the between-subjects level of analysis.

Introduction

Most forms of mental illness are related to problematic emotional patterns—difficulties with the frequency, intensity, expression, and regulation of emotions like fear, anger, and sadness. Because these patterns play out over time, during people's everyday lives, researchers have increasingly begun to collect intensive repeated measures data via ecological momentary assessment (EMA; Hamaker & Wichers, 2017). Within standard EMA paradigms, individuals are prompted by a smartphone or wearable device to provide self-report ratings of emotions many times over the course of several days or weeks, yielding person-specific time-series data that allows for modeling of the timevarying dynamics in emotion as they occur in daily life (see Carpenter, Wycoff, & Trull 2016; Moskowitz et al., 2009; Shiffman, Stone, & Hufford 2008; Trull & Ebner-Priemer, 2013 for reviews). Such models hold a great deal of potential for improving clinical care by providing a nuanced, person-specific understanding of complex idiosyncratic patterns in a client's emotions (Trull & Ebner-Priemer, 2009). Nevertheless, there is a need to be thoughtful about the ways in which EMA time-series data are leveraged for this purpose.

The multivariate time series generated by EMA methods provide a rich source of information related to individual phenomenology as a function of time, allowing researchers to not only investigate individual differences in level (e.g., severity or intensity), but also the temporal dynamics among variables-the differential patterns of variation and covariation that unfold from moment to moment. Still, a challenge remains to identify and extract useful units of information from a time series that can be used to inform the prediction of clinically-relevant phenomena, and several methodologies have been applied to this task to date (Epskamp et al., 2018; Fisher et al., 2019; Fisher, 2015; Fisher & Soyster, 2019; Heron & Smyth, 2010, Schiepek et al, 2016; Roche et al., 2014). With observations across many participants, variables, and time-points, EMA time-series data provide fertile ground for both idiographic and nomothetic analyses (Hamaker & Wichers, 2017). Researchers can examine between-persons distributions of within-person temporal patterns with a degree of granularity and precision that is not available from cross-sectional measurements. Thus, even at the between-subject level, EMA provides detail beyond basic differences in severity or intensity. However, with increased granularity and operational precision comes the responsibility for researchers to demonstrate that these levels of analysis provide predictive information above and beyond mean levels. Can intraindividual measures of fluctuation and variation provide incremental information about etiology, symptom severity, or treatment outcome (Wright & Zimmerman, 2019)? As the field continues to explore person-level temporal dynamics, there is a need to determine which of these metrics provide incremental predictive validity for understanding clinical phenomena between subjects.

The extant affect dynamics literature has provided a set of metrics that quantify intraindividual patterns of fluctuation in constructs like positive affect (PA) and negative affect (NA) in terms of their volatility, predictability, and constancy (Kuppens & Verduyn, 2017). As we've noted, these metrics are derived from the analysis of within-person time-varying processes; however, their aggregation across participants creates nomothetic distributions of idiographic temporal patterns, revealing rank-order differences in temporal dynamics between individuals or groups. Three of the most common affect dynamics in the literature include variability, defined as the within-person standard deviation (SD) of the PA or NA observations; instability, the mean squared successive difference (MSSD) of PA or NA; and inertia, which is defined as the lag-1 autoregression (AR) of the PA or NA vector.

Variability represents whether a person endorsed extremes of positive or negative emotion, regardless of when extreme ratings occurred. Instability is intended to capture the magnitude of point-to-point shifts in PA or NA; high instability means that a person tended to experience large shifts in PA or NA from one moment to the next. Thus, this metric reflects variability as a function of temporal order. Finally, inertia captures the extent to which emotions self-perpetuate or predict themselves over time. Crucially, the SD, MSSD, and AR are correlated processes. It has been demonstrated in simulated time-series data that for constant levels of variability, instability/MSSD and inertia/ AR are inversely related (Jahng, Wood, & Trull, 2008).

Affect Dynamics and Psychopathology

Mounting empirical evidence supports the notion that affect dynamics are associated with well-being and psychological health, broadly speaking (Kuppens & Verduyn, 2017; Trull, Lane, Koval, & Ebner-Priemer, 2015; Wichers, Wigman, & Myin-Germeys, 2015). A question remains regarding which specific types of affect dynamics are most relevant to psychopathology—and which (if any) metrics can outperform PA and NA mean levels in the prediction of negative psychological outcomes (Dejonckheere et al., 2019).

Affective instability (MSSD) and inertia (that is, lag-1 autoregression) are inversely related (Jahng, Wood, & Trull, 2008), so we might expect these metrics to have opposite effects on well-being. However, there is a paradox in the literature wherein high instability and high inertia are both associated with lower self-reported well-being and greater psychopathology (Houben, Van Den Noortgate, & Kuppens, 2015). Commonly, depression is the specific form of psychopathology under examination (cf. Koval, Pe, Meers, & Kuppens, 2013). One interpretation of this instabilityinertia paradox might be that optimal emotion fluctuation lies in the middle of a hypothetical continuum from emotional "flexibility" to emotional "rigidity". Perhaps instability and inertia occupy the two poles, respectively, of that continuum. That is, extreme instability may indicate maladaptive levels of emotional flexibility, changing too much in response to the environment, while extreme inertia may suggest emotional rigidity or not changing enough.

Koval, Pe, Meers, & Kuppens (2013) offer another explanation, arguing that the instability/inertia paradox results from statistical overlap in these constructs. In a depressed sample, they found that depressive symptoms were associated with greater inertia and variability of NA, but not instability. Bos, deJonge, & Cox (2018) replicated these findings; showing in a depressed sample that the association between depression and instability is accounted for by the SD, and the association between depression and SD is accounted for by mean level. A recent paper provides compelling evidence that perhaps affect dynamics only predict individual differences in well-being due to their association with mean levels of NA and PA. Dejonckheere and colleagues (2019) compared a range of metrics of affect dynamics including variability, instability, and inertia, finding across 15 studies (N = 1,777) that none of these dynamics added incremental value to the between-subjects prediction of well-being above and beyond mean level. These findings seem to indicate that instability may not be relevant to the prediction of psychological health.

There are other gaps in the literature which also remain

to be addressed. While affect dynamics have been studied in clinical samples for many years, studies modally focus on the relationship between affect dynamics and depression symptoms (Trull, Lane, Koval, & Ebner-Priemer, 2015) or subjective well-being (Houben, Van Den Noortgate, & Kuppens, 2015). More remains to be known about how affect dynamics relate to specific symptom measures and psychopathology more broadly. For example, much less is known about affect dynamics' relationship to anxiety disorders, despite the fact that anxiety disorders are more prevalent than depression (Kessler et al., 2005). Further, in the current literature, studies of NA dynamics and psychopathology are more common than studies that include PA dynamics. Some have found that PA dynamics are less relevant to depression symptoms: for instance, Thompson et al (2012) demonstrated that individuals with depression exhibited no significant differences from healthy controls in terms of their variability, instability, or inertia of PA. Koval, Sutterlin, & Kuppens (2015) showed that PA inertia is less relevant to depression than NA inertia. However, more remains to be known about how PA dynamics relate to other forms of psychopathology.

Affect Dynamics and Treatment Outcome

The experience of psychopathology can be conceptualized as a complex dynamical system: that is, a system with multiple stable states whose underlying conditions are influenced by many factors (Wichers, Wigman, & Myin-Germeys, 2015). In these systems, it may be difficult to identify exact mechanisms responsible for state transitions (for example, into or out of a depressive episode); but these transitions are often preceded by generic "warning signs" like rising autocorrelation and variance of a signal (Wichers, Wigman, & Myin-Germeys, 2015). Affect dynamics may therefore be a useful warning signal of imminent state transitions concerning psychopathology. A "state transition" is a major goal of psychotherapy-that is, to establish new, more adaptive patterns among emotions and behaviors. Thus, it may be useful to gain a more nuanced understanding of affect dynamics as predictors of change in treatment.

There are mixed findings concerning the relationship between specific affect dynamics and treatment response, and many gaps remain in the literature. Peeters et al (2010) demonstrated that depressed patients who exhibited less emotional reactivity to negative daily events were less likely to recover from MDD. Peeters and colleagues found that greater instability of NA was a positive sign for treatment outcome—those with higher instability exhibited greater reductions in symptoms following treatment. However, Husen et al (2016) demonstrated that greater instability of NA was linked to poorer response to CBT in a sample with mixed mood and anxiety disorder diagnoses. Of note, there were different patterns of comorbidity in these samples, indicating that affect dynamics may differentially relate to treatment outcome across forms of psychopathology (for instance, perhaps instability of NA is positively related to change in depression, but not in other forms of psychopathology).

With respect to PA dynamics, there is clear evidence—at least in depressed samples—that patterns of fluctuation in PA are related to treatment outcome. Wichers et al (2012) demonstrated that moments of increase in PA during an EMA time series are predictive of favorable response to treatment for depression. These authors found that moments of PA increases are actually more predictive of symptom remission for depressed individuals than moments of reduction in NA. This might suggest that variability or instability of PA, more than that of NA, is a positive indicator for treatment response. Relatedly, there is evidence that greater inertia of PA is associated with better current and future recovery from depression (Heller et al., 2009; Hohn et al., 2013). This remains to be extended to other forms of psychopathology beyond depression.

The Present Study

The present study aims to build upon and extend the prior literature by investigating NA and PA dynamics as predictors of 1) anxiety and depression symptom severity and 2) pre-to-post-treatment change in these symptoms in a clinical sample of adults with mood and anxiety diagnoses. We ran a series of hierarchical multiple regression models for each valence (PA and NA) and each symptom domain (anxiety and depression) beginning with instability of affect as the independent variable, and iteratively adding mean level, and the other affect dynamics (variability + inertia). Thus, we examined whether or not affect dynamics "beat the mean" (following Dejonckheere et al., 2019) in the prediction of symptom severity and treatment outcome.

While these analyses were largely exploratory in nature, several predictions followed from the literature. We predicted that instability would be associated with symptom severity and treatment outcome when modeled alone; however, consistent with Dejonckheere et al., 2019, we also hypothesized that mean levels of affect would be significantly associated with symptom severity, mitigating the effects of other dynamics.

Methods

Participants

This study utilized data from an open trial of a personalized modular intervention for depression and anxiety, the procedural details and outcomes of which have been recently published elsewhere (Fisher et al., 2019). Briefly, in this study participants with GAD and MDD completed four daily self-report assessments of affect, behavior, and symptoms during a 30-day period prior to receiving cognitive-behavioral treatment. They subsequently received interventions tailored to their symptom profiles as assessed during the EMA period.

Individuals experiencing symptoms consistent with possible GAD and MDD were recruited from the greater San Francisco Bay Area via flyers, referrals, and internet advertisements. After passing a brief telephone screening, 174 potential participants were invited to an in-person appointment where they completed a structured clinical interview to verify diagnosis and assess severity of symptoms. Inclusion criteria were a primary diagnosis of GAD or MDD, age between 18 and 65 years, and a web-enabled mobile phone. Exclusion criteria were any history of psychosis or mania, concurrent treatment or cognitive-behavioral treatment within the past 12 months, and as-needed medication. We calculated interrater reliability for diagnosis based on video recordings of the structured clinical interviews. The inclusion criteria, GAD and MDD, returned kappa values of 0.68 and 0.84, and percent agreement of 95% and 92%, respectively, with two mismatches for GAD and three mismatches for MDD.

In total, 57 individuals (33%) met inclusion criteria for the current study. Of these, 40 began treatment. Seven participants withdrew from the study during treatment, and one participant was unable to complete a post-treatment assessment, leaving 32 participants in the present sample who completed a full course of treatment and posttreatment assessment. Of the 32 individuals included in the present study, 20 (62.5%) identified as female, and the average age was 37.9 years (SD = 14.3). Sixteen participants (50%) identified as white, nine identified as Asian (28.1%), four (12.5%) identified as Latino/a, one identified as Black (3.1%), and two selected "other" (6.3%). In terms of diagnosis, 13 individuals were diagnosed with current primary GAD, 5 were diagnosed with current primary MDD, and 14 met criteria for co-primary diagnoses of both GAD and MDD. 16 of these participants met for at least one current comorbid disorder other than GAD or MDD; other comorbid diagnoses included agoraphobia (n = 2; 6.25%), persistent depressive disorder (n = 3; 9.38%), posttraumatic stress disorder (n = 1; 3.13%), social anxiety disorder (n = 10; 31.25), and specific phobia (n = 4; 12.5%).

Measures

Anxiety Disorders Interview Schedule for DSM-V. (ADIS-V; Brown & Barlow, 2014). The ADIS-5 is a semistructured clinical interview that is designed to diagnose current anxiety, mood, and related disorders according to new DSM-5 criteria. This version of the ADIS-5 builds upon previous versions (the ADIS, ADIS-R, and ADIS-IV for DSM-III, DSM-III-R, and DSM-IV, respectively), which had well-established reliability. The ADIS-IV demonstrates good-to-excellent interrater reliability for DSM- IV disorders (kappa ranging from 0.67 to 0.86, with the exception of dysthymia, kappa = .31).

Depression Anxiety and Stress Scales. (DASS; Lovibond & Lovibond, 1995). The DASS is a 42-item selfreport measure that captures three related dimensions of

symptom severity: depression, anxiety, and stress. In line with the tripartite model (Clark & Watson, 1991) these subscales were created to measure symptoms unique to anxiety and depression. The depression subscale captures anhedonia or low PA unique to depression, the anxiety subscale captures hyper-arousal unique to anxiety, and the stress subscale assesses high NA symptoms shared across both categories. Therefore, to capture the full range of self-reported depression and anxiety syndromes, the present study utilized the depression + stress subscales to measure self-reported depression severity, and the anxiety + stress subscales to measure self-reported anxiety symptom severity. As each of the three subscales consists of 14 items, our depression and anxiety composites (each of which included its respective subscale + the stress subscale) were each indicated by 28 items. Items were rated on a 4-point Likert scale ranging from 0-3 with the anchors "did not apply to me at all", "applied to me to some degree or some of the time", "applied to me to a considerable degree or a good part of the time", and "applied to me very much or most of the time". Total scores for each subscale of the DASS were calculated by summing across the 14 items that made up each subscale. The stress subscale was added to both the depression and anxiety subscales to generate two 28-item measures of depression and anxiety, respectively. Scores on each of these two measures could range from 0 to 84. In the present sample, the average DASS anxiety score was 34.41 (SD = 14.37) and the average DASS depression score was 44.34 (*SD* = 13.29).

Daily Affect via EMA. During each of the four daily EMA survey pings, participants rated their experience of survey items over the preceding hours using a 0-100 visual analog slider with the anchors "not at all" and "as much as possible" for the 0 and 100 positions, respectively. Surveys contained the symptoms in the DSM-5 criteria for GAD and MDD (down and depressed, hopeless, loss of interest or pleasure, worthless or guilty, worried, restless, irritable, difficulty concentrating, muscle tension, fatigued), as well as an additional 11 items measuring positive affect (positive, energetic, enthusiastic, and content), negative affect (angry and afraid), rumination (dwelled on the past), behavioral avoidance (avoided people, avoided activities, and procrastinated), and reassurance seeking (sought reassurance). In the present study, the four PA items (positive, energetic, enthusiastic, content) and seven NA items (angry, irritable, worthless/guilty, frightened/afraid, down/depressed, worried, hopeless) were averaged at each observation to create a composite PA and composite NA variable at each observation. Of note, worthless/guilty, frightened/afraid, and down/depressed were measured as couplets in a single item (e.g., "felt worthless or guilty") to reflect the language used in clinical assessment for anxiety and depression. These composite affect measures were used to generate affect dynamics variables in the present study. In the present sample, within-person reliability for PA ranged from α = 0.50 to $\alpha = 0.92$ (*M* = 0.81, *SD* = 0.09); within-person reliability for NA ranged from $\alpha = 0.45$ to $\alpha = 0.93$ (M = 0.79, SD = 0.12).

Procedure

Clinical Interview. Interested individuals contacted the Idiographic Dynamics Laboratory at the University of California, Berkeley, and trained research assistants administered a brief telephone screening interview to assess eligibility for the study. Those who were eligible after the initial screening were invited to an in-person appointment for a structured clinical interview. The ADIS-5 was administered by graduate students in clinical psychology supervised by a doctoral level clinical psychologist. At this appointment, participants also completed a battery of self-report symptom measures including the DASS.

EMA Surveys. After enrolling in the study, participants' mobile phone numbers were entered into a secure webbased survey system which prompted participants to answer survey questions four times per day during waking hours. The precise sampling window was tailored to participants' self-reported wake up times, and during this window, they received surveys approximately every 4 hours. The exact time of the ping was randomized within a 30-min window. Participants had a time limit of approximately four hours to respond to each survey; each survey expired once the subsequent survey was sent. Each survey prompt was sent to participants as a text message containing a hyperlink to a web-based survey. Every time pings were sent to participants, the back-end of the system recorded a time stamp, whether the participant completed the survey or not. Participants were instructed to complete surveys for a minimum of 30 days (the total number of days ranged from 29 to 42; M = 34.25).

Personalized Treatment. Following the 30-day EMA period, participants received a course of modular cognitivebehavioral treatment for mood and anxiety disorders. Treatment was personalized via selection of relevant modules from the Unified Protocol (Barlow et al., 2017) to address the most relevant symptoms and mechanisms for each participant (Fisher et al., 2019; Fisher & Boswell, 2016; Fernandez, Fisher, & Chi, 2016). The average number of sessions delivered in the study was 10.38, ranging from 4 to 14 (mode = 9). Within days of completing treatment, participants completed an in-person follow-up assessment appointment to assess change in diagnosis and symptom experience. At this appointment, trained graduate students again administered a diagnostic structured clinical interview and participants completed various self-report instruments including the DASS.

Data Preparation and Analysis. Data were processed and analyzed using R (version 3.5.3; R Core Team, 2019). Complete R syntax as outlined in this paper is available in the supplementary material on OSF [https://osf.io/34xyh/]. First, composite PA and composite NA were calculated for each observation of each participant's time series by taking

the average of PA and NA items respectively at each observation. PA items included energetic, enthusiastic, content, positive (for PA) and angry, irritable, worthless/guilty, frightened/afraid, down/depressed, worried, hopeless (for NA). To account for any mean level shifts over time, linear trends were removed from the PA and NA composite measures by regressing each measure on time and extracting the residuals. Next, each participant's positive and negative affect dynamics (variability, instability, and inertia) and mean levels were calculated from their de-trended EMA time series, using the PA and NA composite vectors. Missing observations (e.g., those for which the participant failed to respond during the correct four-hour time window) were excluded as a function of listwise deletion; however, their timestamp was retained in order to maintain approximately even spacing.

Variability was calculated as the standard deviation of the PA and NA vectors for each person. Importantly, variability is a measure without temporal dependence. High variability indicates that a participant used a wider range of points on the scale to rate their emotions, regardless of when in the time series any extreme or outlying ratings occurred.

Night-to-morning lags were removed before calculating inertia and instability, as these measures rely on temporal dependence. Inertia was defined as the lag-1 autocorrelation of the PA/NA composite variables. In this sense, inertia indicates the extent to which emotions self-perpetuate over time, as a measure of the extent to which variance in PA/NA as observed at time T can be predicted from the same variable at the previous observation (T-1). To calculate inertia, we used the AutoR function in the psych package in R. Given that the presence of a time-dependent trend can inflate estimates of the strength of the autoregression (Jahng, 2008), PA and NA variables were first de-trended by regressing these variables on cumulative time. The lag-1 autoregression was run on the residuals-that is, PA and NA with the linear time-dependent trend removed. To calculate instability, we used the mean squared successive difference (MSSD) as calculated via the psych package in R (Revelle, 2017). Instability indicates the average magnitude of moment-to-moment shifts in intensity of PA or NA within a person's EMA time series.

Affect Dynamics and Symptom Severity. To assess relationships between symptom severity at baseline and each measure of affect dynamics, multiple regression models with anxiety and depression symptom severity regressed on emotion dynamics were estimated in an iterative fashion, adding predictors one at a time to an increasingly complex model. Separate models were constructed for each affective valence (PA and NA) and for each symptom domain (anxiety and depression) yielding a total of four hierarchical models. We began with instability (MSSD) of affect as the only predictor of each symptom domain, and iteratively added mean levels, variability, and inertia.

Affect Dynamics and Treatment Outcome. Next, we

examined effects of affect dynamics on treatment outcome by using a similar iterative modeling approach as described above. These models were constructed in a similar fashion, except that treatment outcome rather than symptom severity was modeled as the dependent variable, with baseline symptom severity included as a covariate in each stage. As with the symptom severity models, we constructed four total models of treatment outcome, for each affective valence (PA and NA) and symptom domain (anxiety and depression). With the same rationale as described above, instability was tested first, and then mean levels, instability, and inertia.

Treatment outcome was defined as the change scores (post-treatment scores minus pre-treatment scores) for each of the two symptom domains (anxiety and depression). Change scores were chosen as the outcome metric of interest to allow us to assess whether affect dynamics pretreatment have an effect on one's general malleability to change during treatment, and baseline severity for each domain was modeled as a covariate to account for where on the scale that change occurs. In these treatment response models, the direction of effect is negatively valenced, such that negative numbers of a larger magnitude indicate greater change in symptoms during treatment. Therefore, if an affect dynamic variable is negatively associated with treatment outcome this reflects that higher levels of the emotion dynamic variable are associated with more pronounced change in treatment.

Results

For the sample of 32 treatment completers, the total number of observations ranged from 110 to 158 (M = 129.75, SD = 12.05). The percentage of missing data ranged from 0% to 31.8% (M = 12.39%, SD = 0.08%). Group-aggregated descriptive statistics (means, SDs, and ranges) for all affect dynamics variables are presented in Table 1 and visualized as distributions in Figures 1a and 1b. Descriptive statistics by diagnostic group for all outcome measures (baseline and change scores for the DASS anxiety and depression measures) are provided in Table 2.

Table 1. Descriptive statistics for affect dynamics.

	Between-Persons Summary Statistics (aggregated across the sample)			
Affect dynamics Metric (within each time series)	M	SD	Min	Max
PA Mean Level	39.24	9.59	18.14	62.52
PA Variability (SD)	14.50	4.14	6.23	22.76
PA Instability (MSSD)	178.33	115.26	33.83	536.84
PA Inertia (AR-1)	0.37	0.19	-0.02	0.83
NA Mean Level	40.61	14.28	8.29	74.63
NA Variability (SD)	13.42	3.56	6.18	19.16
NA Instability (MSSD)	136.66	80.15	22.38	352.74
NA Inertia (AR-1)	0.45	0.19	-0.03	0.79

Table 2. Correlations among affect dynamics variables

	1	2	3	4	5	6	7	8
1: PA Mean Level	1							
2: PA Variability	0.19	1						
3: PA Instability	0.28	0.81***	1					
4: PA Inertia	-0.03	0.21	-0.22	1				
5: NA Mean Level	-0.27	-0.25	-0.28	-0.16	1			
6: NA Variability	0.01	0.69***	0.53**	0.24	0.00	1		
7: NA Instability	0.00	0.56***	0.58***	-0.15	0.05	0.79***	1	
8: NA Inertia	0.05	0.12	-0.15	0.71***	0.04	0.31	-0.24	1

Note. * *p* < .05; ** p < .01; *** *p* <.001

Panel A: Mean Levels.



Panel B: Variability (SD)



Figure 1a. Distributions of within-person affect dynamics across the sample. Mean levels and variability

Panel C: Instability (MSSD)



Panel D: Inertia (AR-1)



Figure 1b. Distributions of within-person affect dynamics across the sample. Instability and inertia.

	β	<i>t</i> -value	<i>p</i> -value	d		
MSSD alone						
MSSD (instability)	-0.19	-1.55	0.13	-0.39		
MSSD + PA mean levels						
MSSD (instability)	-0.19	-1.44	0.16	-0.36		
Mean	-0.53	-0.03	0.97	-0.01		
MSSD + PA mean levels + other dynamics						
MSSD (instability)	0.53	1.93	0.07	0.49		
Mean	-0.02	-0.19	0.85	-0.05		
SD (variability)	-0.77	-2.84	0.009	-0.71		
AR (inertia)	0.043	2.62	0.01	0.66		

Table 3. PA Dynamics and Depression Treatment Response

Note. PA = positive affect; MSSD = mean squared successive difference; d = Cohen's d

Table 4. PA Dynamics and Anxiety Treatment Response

	β	<i>t</i> -value	<i>p</i> -value	d		
MSSD alone						
MSSD (instability)	-0.25	-2.27	0.03	-0.57		
MSSD + PA mean levels						
MSSD (instability)	-0.21	-1.81	0.08	-0.46		
Mean	-0.15	-1.27	0.21	-0.32		
MSSD + PA mean levels + other dynamics						
MSSD (instability)	0.52	2.36	0.03	0.59		
Mean	-0.18	-1.74	0.09	-0.44		
SD (variability)	-0.81	-3.67	0.001	-0.92		
AR (inertia)	0.34	2.54	0.02	0.64		

Note: PA = positive affect; MSSD = mean squared successive difference; d = Cohen's d

Affect Dynamics and Symptom Severity

Greater mean levels of NA were associated with more severe depression symptoms ($\beta = 0.46$, p = 0.01); no other affect dynamics beyond mean level were found to relate to depression symptom severity. However, for anxiety symptoms, greater instability of NA was significantly associated with worse symptoms ($\beta = 0.49$, p = 0.002), even when controlling for mean levels of NA ($\beta = 0.34$, p = 0.03). In the final model, when variability and inertia were added, the effect of instability fell below the threshold of statistical significance (p = 0.10). No significant associations were found between any PA dynamics (including mean levels) and anxiety symptom severity.

Affect Dynamics and Treatment Response

NA and PA dynamics were tested as predictors of pre-to-

posttreatment change in depression and anxiety symptom scores, controlling for baseline symptom levels. Of note, in these models we utilize change scores as the dependent variable (posttreatment – pretreatment); therefore, as negative scores of a greater magnitude reflect more change in treatment, negative beta coefficients indicate a direct (rather than an inverse) relationship between the independent variable and treatment response. Baseline symptom severity was significantly associated with treatment response across all models, such that individuals with greater severity of symptoms demonstrated more pronounced change after treatment. The number of sessions varied between participants, but when this was added as a covariate it did not impact the observed effects.

We found no metric of NA dynamics to be significantly associated with change in depression symptoms. However, instability/MSSD of NA was significantly associated with change in anxiety symptoms ($\beta = -0.32$, p = 0.02); indicating that greater instability of NA prior to treatment predicted more pronounced change in anxiety symptoms during treatment. However, when variability and inertia were added in the final iteration of this model, the effect of instability fell below the threshold of statistical significance. No other metric of NA dynamics, including mean levels, significantly predicted change in anxiety symptoms.

Most of the significant relationships observed in the present analyses were PA dynamics predicting treatment response (see Tables 3 and 4). Variability, instability, and inertia of PA pre-treatment were all significantly associated with change in anxiety symptoms ($\beta s = -0.80, 0.52$, and 0.34 respectively; all ps < 0.05). Similarly, we found that PA variability and inertia were significantly associated with change in depression ($\beta s = -0.77$ and 0.43 respectively; ps < 0.05). This indicates that individuals with greater variability in PA pre-treatment had more pronounced change in their depression and anxiety symptoms during treatment. The opposite was true for inertia (greater inertia of PA was associated with less change in depression and anxiety symptoms). We found no significant effects of mean levels of PA in these models.

Discussion

The present study examined dynamics of NA and PA as predictors of anxiety and depression symptom severity, as well as pre-to-posttreatment change in symptoms, in a cohort of individuals diagnosed with mood and anxiety disorders who subsequently received CBT. For each valence and symptom domain, we ran a set of multiple regression models beginning with instability of affect as the only predictor, iteratively adding mean levels of affect, variability and inertia. We found that greater instability (MSSD) of NA was associated with greater anxiety symptoms, and more pronounced change in anxiety from pre-to-post treatment, even when controlling for mean levels of NA. Conversely, NA dynamics were not shown to be related to depression symptom severity or treatment response. While PA dynamics were not related to depression or anxiety symptom severity at baseline, we found that all three PA dynamics (variability, instability, inertia) were significantly associated with pre-to-posttreatment change in anxiety and depression symptoms.

Taken together, our findings point to specificity in the relationships between affect dynamics and symptom domains. It may not be accurate to say that affect dynamics are related to well-being and psychological health generally. Dejonckheere et al. (2019) illustrated this by demonstrating that affect dynamics did not add meaningful incremental value predictive value for psychological health, above and beyond mean levels. Crucially, their study defined psychological health as a composite including subjective well-being, depression symptoms, and borderline personality symptoms. In the present study, we took a narrower ap-

proach to defining psychological health, examining the relation between affect dynamics and specific symptom measures that assess depression and anxiety pathology separately. Consistent with Dejonckheere et al., we did not find a relationship between NA dynamics and depression symptom severity or treatment response. However, we did show that instability of NA exhibited a significant direct relationship with anxiety symptom severity and the extent to which anxiety symptoms change in treatment. Further, we found that PA dynamics are generally unrelated to baseline symptom severity, but that all three dynamics of PA were significantly associated with treatment response for both anxiety and depression. In short, while we cannot say that affect dynamics are linked to well-being broadly, there is evidence for more granular relationships: NA instability may be linked to severity and response of anxiety symptoms, and PA dynamics may be linked to treatment outcome-but not baseline severity-of anxiety and depression.

The finding that PA dynamics (variability, instability, and inertia) are related to treatment response for both anxiety and depression is consistent with prior findings. To wit, it has been shown that greater reactivity to positive events leads to better treatment outcomes for depression (Wichers et al., 2012) and that higher inertia of PA is also linked to better outcomes for depression (Heller, 2009; Hohn et al., 2013). However, it is surprising that the direction of effects in the present study is counter to what is predicted by the literature. As expected (given Wichers et al., 2012) we showed that greater variability of PA was associated with more pronounced treatment response. Contrary to expectation, however, greater instability and inertia of PA were both associated with less pre-to-posttreatment change in anxiety and depression symptoms. This echoes the instability-inertia paradox in the literature concerning the prediction of well-being. Until future studies can examine this further, it might be prudent to interpret the finding for PA variability, as this is consistent with the literature: greater variability in PA is linked to better treatment outcomes for anxiety and depression. Clinicians may therefore want to cultivate awareness of and responses to PA such as encouraging mindfulness and savoring positive experiences (Feldman, Joorman, & Johnson, 2008) as a strategy in treatment.

Limitations

A handful of limitations in the present study must be noted. First, although we had a large number of observations per-person yielding high within-person statistical power, our sample size of 32 treatment completers provided relatively low between-subject statistical power in the present study. It is therefore possible that we were unable to detect some effects that would be observable with a larger sample. For instance, in our symptom severity models, we did not consistently find significant associations between mean levels of affect and symptom experiences. We might expect, given findings by studies such as Dejonckheere et al (2019), that such effects would emerge. The present study utilized specific symptom measures rather than more general measures of self-reported well-being, so it is possible that this lack of associations with mean levels is due to the fact that specific symptoms are less associated with mean levels of affect than with broader measures of "psychological health", but it is also possible that this is a function of low statistical power. However, it is worth noting that the effects of PA dynamics on treatment response for both symptom domains were detected despite low power. Thus, perhaps these findings are particularly robust.

It should also be noted that in the present study, affect dynamics were measured pre-treatment, during a 30-day window between diagnosis and the beginning of CBT intervention. Therefore, conclusions about affect dynamics' associations with change in treatment should be interpreted accordingly. PA dynamics before treatment begins may predict an individual's response to treatment, and this information may be utilized clinically to generate hypotheses about a person's likely responses to intervention based on their pre-treatment patterns. However, as participants did not continue completing EMA measurements during treatment, we are unable to answer questions about how affect dynamics changed during therapy as a function of intervention, or how these changes may have been associated with change in symptoms. This merits further consideration in future work, especially in context of the suggestion that, considering psychopathology as a dynamical system (cf. Wichers, Wigman, & Myin-Germeys, 2015) affect dynamics may herald the onset of state-transitions within the system.

Future Directions

The present study raises many questions that should be explored in future work. First, the study's primary findings-the effect of NA instability on anxiety symptom severity and treatment response, and the effect of all PA dynamics on treatment response for anxiety and depression-should be investigated further in a larger sample with more between-subjects statistical power. It would be worth exploring whether specific diagnostic category or patterns of comorbidity influence these results. One conclusion that begins to emerge from the present findings is specificity in the relationship between affect dynamics and specific symptom domains. In most previous studies, affect dynamics have been explored as predictors of broad, general constructs like "well-being" and "psychological health", with many studies utilizing composite measures that comprise variables such as depression symptoms, borderline symptoms, and subjective well-being. It may be worth considering exploring the relationship between affect dynamics and psychopathology with finer-grained detail in future studies. For example, we showed that NA instability was specifically connected to anxiety symptoms, but not depression symptoms. It may be worthwhile to unpack the NA construct into discrete emotions: perhaps we would then discover more meaningful relations between emotion dynamics and specific forms of psychopathology. Examining links between affect dynamics and psychopathology with more granularity might reveal that dynamics of sadness or guilt relate specifically to depression, dynamics of excitement or enthusiasm relate to mania in bipolar disorder, or dynamics of anger or irritability relate to externalizing psychopathology.

While a handful of studies, including the present one, have examined affect dynamics as predictors of treatment response, such studies are less common in the literature. Our findings (and others) suggest that affect dynamics-in our case, specifically PA dynamics-may be relevant predictors of how someone will respond to CBT intervention. If explored further this could offer potential clinical application in the realm of personalizing intervention: it may be the case that certain types of affect dynamics predict better versus worse response to different modalities of intervention. If this were the case, measuring affect dynamics during a brief pre-treatment window could be useful in tailoring intervention to be optimally effective for each individual. In addition, while the present study did not measure affect dynamics during treatment, future studies could take this approach. This might be a useful indicator of progress in treatment (for example, clinicians could benefit from knowing whether or not an intervention is changing affect dynamics over time).

Finally, while the present study examined affect dynamics as a between-persons predictor of symptoms and treatment response, it is possible that analyzing these relationships at the within-person level would yield very different conclusions. Future research should explore this further. For example, if symptoms were measured daily and affect were assessed multiple times daily, within-person associations between symptoms on day t and variability, instability, and inertia of affect as captured within the day t window could be estimated. It would be useful for person-oriented clinical science to better understand the how affect dynamics contribute value to the between-person and withinperson prediction of clinically relevant phenomena; and it would be interesting to compare the differences in these predictive relationships from the between-persons to the within-persons level. Empirical studies have repeatedly demonstrated that distributions of idiographic estimates are highly variable compared to nomothetic ones (Fisher, Jeronimus, & Medaglia, 2018), so a reasonable hypothesis is that affect dynamics exhibit idiosyncratic relationships with symptoms throughout the population. Future research must be done to determine what metrics are relevant predictors of specific clinical phenomena at the within-person level.

Declaration of conflicting interests

The authors declare that there is no conflict of interest.

Author contributions

HB conducted the main analyses and served as the primary author of the manuscript. PS and AF assisted with data management and analysis, conceptualization of study aims and preparation of the final manuscript.

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