Research paper

Daily affect dynamics predict early response in CBT: Feasibility and predictive validity of EMA for outpatient psychotherapy

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ABSTRACT

Background: Previous studies have shown that individual differences in affect dynamics during depressed patients' everyday lives allow the prediction of treatment outcome and of symptom reoccurrence in remitted patients. In this study, we analyze whether understanding patients' affective states and their fluctuation patterns helps predict early treatment response (until session 5).
Methods: Ecological Momentary Assessment (EMA) strategies allow in-depth analyses of real-time affective states and of their dynamics. Repeated assessments were made four times a day during a two-week period to capture real-life affective states (positive affect, PA and negative affect, NA) and dynamics (fluctuations in NA and PA) before the start of outpatient treatment of 39 patients. Due to the nested structure of the data, hierarchical linear models were conducted.
Results: PA/NA ratios, as well as fluctuations in NA predicted early treatment response, even when adjusting for initial impairment. In contrast, mean levels of NA or PA, as well as fluctuations in PA did not predict treatment response.
Limitations: The time between the EMA assessment and treatment onset varied between patients. However, this variation was not associated with early change.
Conclusions: The results suggest that pre-treatment affect dynamics could provide valuable information for predicting treatment response independent of initial impairment levels. Better predictions of early treatment response help to improve treatment choices early in the treatment progress.
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The majority of studies exploring evidence-based treatments have investigated questions of effectiveness and efficacy (Lambert, 2013). Recently, research attention has begun to address individual patterns of change in response to empirically validated treatments (e.g., Hayes et al., 2007; Lutz et al., 2005; Rubel et al., 2015; Stulz et al., 2007). Replicated findings have supported the description of the general pattern of treatment response as a log-linear decrease of impairment levels with an increasing number of sessions (e.g., Lambert et al., 2001; Stulz et al., 2013), as postulated by the dose-effect model (Howard et al., 1986). However, even if we assume this general pattern, we know that individual patients differ in their change trajectories and deviate from the general trend.

The study of the phenomenon of change that occurs at the beginning of treatment (early change) has gained increasing interest, often attempting to identify parameters of early change, e.g., early depressive symptoms, which may predict treatment

outcome (Cuijpers et al., 2005; Lutz et al., 2009). Early change patterns have been shown to be associated with outcome across different measures (Hunter et al., 2010), different treatment approaches (Crits-Christoph et al., 2001; Gunlicks-Stoessel and Mufson, 2011) and different diagnoses (Bradford et al., 2011; Hayes et al., 2007; Lewis et al., 2012;). By examining early change patterns, Rubel et al. (2015) were able to show that most change in patients' progress scores took place in an early phase of treatment.

Studies on early change patterns suggest that early response is indeed a critical predictor of treatment outcome (Lutz et al., 2009, 2014; Nordberg et al., 2014). However, at this point, little is known about how these differences in early response arise and which variables could explain them. Previous studies exploring the effects of patient characteristics and change trajectories have commonly used intake measures as predictors of slope variation, that is, how patients differ in change rates a) in general and b) specifically with regard to early response (e.g., Beutler et al., 2006; Clarkin and Levy, 2004; Cuijpers, et al., 2005; Lutz et al., 1999; Stulz at al., 2007). Patients' intake characteristics have been extensively investigated with regard to their predictive validity for

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treatment response (for an overview see Bohart and Wade (2013)). Of these patient variables, especially the intake score of the measure with which change is assessed stands out. There is compelling evidence that the strongest predictor of change in an outcome variable is the intake score of that variable (e.g., Lambert et al., 2002). More symptom distress has been found to be associated with more symptom change over the early and entire course of treatment, but also with a lower probability of reaching an impairment level comparable to a healthy population (Brown et al., 2001). Other intake variables have been shown to explain only a relatively small amount of additional variation beyond what is already explained by the intake score. In the present investigation, the following additional intake variables, which have shown predictive validity in prior research, are considered: therapist-rated functioning, prior psychotherapy, chronicity, and patients' treatment expectations. Higher clinicians' ratings of global patient status and better treatment outcome expectations have been repeatedly shown to be associated with more positive changes over the course of treatment (e.g., Constantino et al., 2011; Lutz et al., 2007). More prior psychotherapy and higher levels of chronicity have been related to slower rates of change in previous research (e.g., Lutz et al., 2007).

There is a contradiction between prevailing diagnostic criteria and our standard methods of clinical assessment: Diagnostic criteria often describe dynamic symptomatology over a certain period of time (e.g., manifest symptoms over a two-week period in depressed patients), whereas the assessments of these criteria typically apply clinical interviews or self-report questionnaires that ask patients to rate symptom distress retrospectively. These typically applied trait questionnaires often fail to capture the large variance of psychopathological processes (e.g., Bolger and Laurenceau, 2013; Verkuil et al., 2007). Findings on patient characteristics that influence change trajectories could be augmented by data collected from patients' or soon-to-be patients' ongoing daily experience, which would be more ecologically valid and not limited to the one-time impression of a clinical interviewer. To better understand the phenomenology and underlying mechanisms of psychopathology and to improve personalized care, methods that allow the collection of more ecologically valid data are needed (MyinGermeys et al., 2009; Trull et al., 2012). Ambulatory assessment strategies, which are applied regularly in the medical and psychophysiological fields, have recently gained growing attention in clinical psychology, psychiatry, and also psychotherapy research (e.g., Marzano et al., 2015, Palmier-Claus, 2011; Trull et al., 2012). One form of ambulatory assessment is the Ecological Momentary Assessment (EMA, Stone and Shiffman, 1994) method. EMA involves repeated event- or time-contingent inquiries of individuals in their natural environment, which are often carried out by electronic data assessment instruments (Fahrenberg, and Myrtek, 2001).

To our knowledge, the effects of daily life affective states and dynamics – assessed by EMA – on early response have not yet been investigated. However, several studies have explored different aspects of affect dynamics in daily life. For instance, Thompson et al. (2012) analyzed emotional variability in a sample of patients with major depressive disorder (MDD) and compared them to a healthy control group. EMA was conducted over seven days with eight daily prompts. In terms of NA, they found that, compared to healthy controls, depressed participants reported greater instability and greater reactivity to positive events, but comparable levels of inertia and reactivity to negative events. In terms of PA, the MDD and control groups did not differ significantly in their instability, inertia, or reactivity to positive or negative events. These results suggest that instability or fluctuation in negative affect might be an indicator of psychological functioning. Some EMA studies have applied EMA methods to investigate different

aspects of affective dynamics in everyday life with regard to treatment response, outcome, and relapse in remitted patients for specific diagnostic subgroups: For example, Peeters et al. (2010) analyzed whether emotional reactivity to daily life events functioned as a predictor of a) treatment response within the first month of psychotherapy (combined with pharmacotherapy if indicated) and of b) remission rates within 18 months in a sample of depressed patients. EMA was applied over 6 consecutive days 10 times daily before treatment onset to assess emotions and daily life events. They found that a) less emotional reactivity to negative and positive life events predicted higher depressive symptom severity after the first month of treatment and b) patients with less negative emotional reactivity to negative life events were less likely to recover from depression over the 18 months follow up. Wichers et al. (2011b, 2010) found that reductions in negative affect following the maximum daily increase of positive affect provided a means to discriminate between treatment responders (assessed after 8 weeks of treatment, defined as a 50% reduction on the Hamilton Depression Rating Scale, HDRS) and non-responders in a sample of depressed patients. They also found that higher negative affect following maximum increases in positive affect was associated with more depressive symptomatology at a six-month follow-up. Forbes et al. (2012) sought to predict the course and outcome of an eight-week open trial of CBT, pharmacotherapy, or a combination of the two by social interaction and affective dynamics in daily life in children and adolescents suffering from depression or anxiety. EMA was applied over four days before treatment onset. The results of this study showed that higher positive affect levels, lower negative affect levels, higher positive to negative affect ratios (PA/NA ratio) and more time spent with fathers predicted lower posttreatment severity of depressive and anxiety symptoms. Additionally, lower absolute levels of negative affect and higher PA/NA ratios predicted faster decreases in symptom severity over the course of the treatment while absolute levels of and fluctuations in PA did not.

The present study aims to broaden our knowledge regarding patient characteristics, which may serve as predictors of early treatment response. We applied EMA methods to collect real-time affective dynamics of patients who were – at the time of assessment – waiting to be treated at our outpatient clinic. In this study, we focused on the following research questions: do positive and negative affective states and their temporal dynamics (fluctuation), assessed via EMA before treatment onset, allow the prediction of early treatment response? More specifically, the following three hypotheses guided our work:

(a) Based on Forbes et al.'s (2012) findings, we expected lower absolute values of NA and higher PA/NA ratios to be associated with faster symptom reductions over the first five treatment sessions. Higher PA/NA ratios (i.e., more positive affect compared to negative affect) relate to higher levels of optimism and self-efficacy which in turn should increase patients' abilities to make use of therapeutic interventions already early in treatment. Along these lines, Larsen (2009) regards the PA/NA ratio as the core of emotional well-being. In accordance with the phase model of psychotherapeutic outcome enhanced emotional well-being (*remoralization phase*) is the first of three phases that psychotherapy patients undergo on their way to remission (Howard et al., 1993). The second phase focuses on symptom reduction (*remediation phase*) and the third phase on the restoration of the general level of functioning (*rehabilitation phase*). As a consequence, for patients with higher PA/NA ratios (i.e., optimism and emotional well-being) it might be able to skip parts of the *remoralization phase* and treatments can right away focus on symptom reduction.

(b) We hypothesized that fluctuations in NA (when controlled for initial impairment) predict poor early response, whereas fluctuations in PA are not tied to early treatment response. This hypothesis is based on Wicher's et al. (2010) findings that NA variability predicts future negative affective symptoms in remitted depressed patients and also takes into account Thompson's et al. (2012) findings that clinical samples display greater instability in NA.

(c) We hypothesized that these indices derived from EMA data would show incremental predictive validity beyond predictors derived from intake measures, which have shown predictive value in previous research.

1. Methods

1.1. Participants

The analyses were based on a sample of patients, who were treated with CBT at the university's outpatient clinic. All patients were assessed via EMA before the onset of their treatment (see procedure and design for further explanation) and underwent at least the first five sessions of treatment. Recruitment for EMA was conducted between October 2013 and April 2015 and screening for eligibility was carried out via the German Version of the Mini-International Neuropsychiatric Interview 5.0. (M.I.N.I.; Ackenheil et al., 1999). 100 patients were screened, of whom 63 were eligible for study participation and 61 agreed to participate in the study. Exclusion criteria included suicidality, current symptoms of PTSD, psychosis and mania. Patients were included if they were positively screened for an affective or an anxiety disorders in the M.I.N.I. Diagnoses were based on the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) Axis I Disorders (First et al., 2002), which was carried out after treatment onset. The majority of the sample was diagnosed with an affective (46.2%) or anxiety (38.5%) disorder as the primary diagnosis. Additional primary diagnoses were eating disorders (5.1%), and substance-related and addictive disorders (5.1%). For the diagnosis of personality disorders, the International Diagnostic Checklist for Personality Disorders (IDCL-P; Bronisch et al., 1996) was adopted, which identified 5.1% of the sample as having a personality disorder. Interviews were conducted by intensively trained independent clinicians with at least one year of clinical experience. These interviews were videotaped; interviews and diagnoses were discussed in expert consensus teams that included four senior clinicians; final diagnoses were determined by consensual agreement of at least 75% of the team members.

Of the patients, 59.0% were female, their age ranged from 19 to 60 years (mean=35.69, SD=11.48) and their impairment levels measured by the Global Severity Index of the Brief Symptom Inventory (BSI; Franke, 2000; German translation of Derogatis, 1975) ranged from .11 to 2.55 (mean=1.24, SD=.59).

The study was approved by the university's ethics committee and written informed consent was obtained.

1.2. Procedure and design

The study was carried out at the outpatient clinic of a university in southwestern Germany and the EMA period was integrated into the clinic's regular care process. After patients registered for treatment, they filled out a questionnaire package, which is part of the clinic's routine assessment. The questionnaire package included the BSI. In a second step, patients were screened for study eligibility with the Mini International Neuropsychiatric Interview, M.I.N.I. (Ackenheil et al., 1999) via telephone. The interview was carried out by trained clinicians. Those who were

found eligible for the study and agreed to participate (61% of the patients screened) were invited to the outpatient clinic to receive training for iPod use, which was used for the EMA data collection. Written informed consent was obtained during this session. The two-week EMA period started immediately after the training session and was part of the regular waiting period before the onset of treatment. Patients were signaled 4 times a day for 14 consecutive days (handover and return days not included). Audio signals followed a time-contingent sampling plan, with signals every four hours between 8:00 a.m. and 8:00 p.m. on weekdays and between 10:00 a.m. and 10:00 p.m. on weekends. Participants were contacted after the first two days of EMA to make sure that no problems arose; additionally they received a phone number they could call in case of questions or problems. After 14 days of data collection, participants returned the iPods. Participants were compensated with € 80 for completing the 14-d EMA period. After this session, patients routinely continued their waiting period before the onset of treatment. The waiting period (time between registration and treatment onset) was 150 days on average (SD=1.69). The time between EMA and treatment onset comprised on average about 2.3 months (SD=1.9). After the onset of treatment, the routine monitoring and diagnostic process began, during which patients reported well-being and impairment weekly. Data for the first five treatment sessions were available for this study.

1.3. Instruments and data collection

The following section describes all relevant measures that were included in the study. Measures are presented in the order of application.

1.3.1. Brief symptom inventory (BSI)

To assess overall impairment levels, the German version of the BSI (Franke, 2000) was administered at registration for treatment. The BSI is a 53-item short form of Derogatis' Symptom Checklist, which assesses patients' general impairment levels on nine symptomatic subscales using a five-point Likert scale ranging from 0 (not at all) to 4 (extremely). In this study, we used BSI's Global Severity Index only. The internal consistency of the BSI is $\alpha=.92$ and the retest-reliability is $r_{tt}=.90$ (Franke, 2000).

1.3.2. Affective states

Positive (PA) and negative affect (NA) were assessed during the EMA procedure. Participants rated eight momentary affective states on 5-point Likert scales (ranging from 1 – "a little or not at all" to 5 – "very"). Our choice of the EMA affect items was guided by the PANAS scales (Watson et al., 1988). Four items were assessed for the NA Scale and four for the PA scale (see Appendix A). As recommended by Shrout and Lane (2012) in the Handbook of Research Methods for Studying Daily Life, we estimated the reliability of the single items of each scale across time points of assessment (0–56) and the average reliability of each scale. The average reliability of the NA scale was $\alpha=.77$. The average reliability of the single items of the NA scale ranged from .60 to .90 at each time point of assessment. The reliability of the PA scale was $\alpha=.84$. The average reliability of the single items of the PA scale ranged from .76 to .90 at each time point of assessment. Mean PA, mean NA and mean PA/NA ratios as well as temporal fluctuations in affective states quantified by the mean squared successive differences (MSSD) in PA and NA were computed for each participant. MSSD is the average of the squared differences between successive observations at occasion i and $i+1$

$$MSSD = \frac{\sum_{i=1}^{n-1} (x_{i+1} - x_i)^2}{n - 1}$$

with n being the number of time points, X_i the PA or NA score of a patient at time point i, and X_{i+1} the PA or NA score of the same patient at the next time point ($i+1$). The MSSD is a preferred index of affective fluctuation in EMA studies, because it captures both variability and temporal dependency in a time series (Jahng et al., 2008). We only included within-day lags, as overnight lags represent structurally different lags with longer time periods and intervening night's sleep.

1.3.3. Treatment expectation

Treatment expectation was assessed by a single item before treatment onset: "How convinced are you that psychotherapy will help you deal with your problems? ". Answers ranged from 1 = "not at all" to 4 = "very much".

1.3.4. Prior psychotherapy

The extend of prior psychotherapeutic treatment was assessed by a single item before treatment onset: "How much psychotherapy have you had in the past?". Answers ranged from 1="none" to 6="more than a year".

1.3.5. Chronicity

Chronicity was assessed by a single item before treatment onset: "How long has the problem for which you are presently seeking treatment been a concern to you?". Answers ranged from 1="less than a month" to 6="more than 2 years").

1.3.6. Global assessment scale (GAS)

The German version of the therapist-rated Global Assessment Scale (GAS; Endicott et al., 1976) was applied at the first session of treatment. The GAS assesses overall functioning of an individual on a continuum from psychological or psychiatric illness to health measured on a scale ranging from 0 to 100.

1.3.7. Hopkins symptom checklist-11 (HSCL-11)

The HSCL-11 (Lutz et al., 2006) was administered at the beginning of each session. Patients fill out the questionnaire via touch screens, allowing for no missing items. This 11-item self-report inventory assesses symptomatic distress. It is a brief version of the SCL-90-R (Derogatis, 1992). The items are answered on a 4-point Likert scale ranging from 1 (not at all) to 4 (extremely). The mean of the 11 items represents the client's level of global symptomatic distress in the preceding week. It is highly correlated with the GSI ($r=.91$) and has high internal consistency ($\alpha=.92$; Lutz et al., 2006).

1.4. Data analytic strategy

The focus of our analyses was to predict early change over the first five sessions of treatment (i.e., sessions 1–5). Due to the nested structure of the data (sessions nested within patients), we conducted hierarchical linear models.

Prior research has shown that a log-linear transformation of session numbers, can parsimoniously approximate the average pattern of change over the whole course of the treatment and in an early treatment phase (e.g., Gibbons et al., 1993; Stulz et al., 2013).[1] In accordance, we modeled each patient's HSCL score as a function of session number (S) as follows:

Level 1: $\text{HSCL} - 11_{pt} = \beta_{0p} + \beta_{1p} * \text{Log}(S)_{ps} + e_{ps}$

Level 2: $\beta_{0p} = b_{00} + b_{01}*\text{GSI}_p + r_{0p}$

$$\beta_{1p} = b_{10} + r_{1p}[\, + b_{11}*\text{GSI}_p + \ldots + b_{1x}*Predictor_p] \tag{1}$$

At level 1, a patient´s p HSCL-11 score in session s was predicted by the patient specific intercept (β_{0p}; i.e., the HSCL-11 score before the first session), the patient specific rate of change (β_{1p}; i.e., the expected change in HSCL-11 scores per $\log_{10}$ of session number), and a session specific error (e_{pt}).[2] At level 2, patient specific intercepts and slopes are predicted by patient variables. In all models, a patient's intercept is modeled as their intake score deviation (r_{0p}) from the average overall intake score (b_{00}) and their intake GSI score (b_{01}). Patients' slopes are modeled as their individual deviation (r_{1p}) from the average overall slope (b_{10}). This model served as our unconditional base model (Eq. 1 excluding the square brackets). We then successively augmented Eq. (1) to test the cross-level interaction of eleven different level 2 predictors with regard to their predictive value for the slope factor (i.e., change from session one to five). First, we entered the intake GSI score (b_{11}) as an indicator for initial impairment. As initial impairment has been repeatedly found to be the strongest predictor of change, we tested all additional predictors above and beyond the effects of the GSI (b_{1x}). Of the ten additional predictors we added, three were the single items with regard to treatment expectation, prior psychotherapy and chronicity. As a fourth predictor, we used the therapist-rated GAS. To control for differences in the patients' waiting time between the EMA period and actual treatment onset, we entered this time span as a fifth predictor. The remaining five predictors were derived from the EMA period: Besides entering the PA/NA ratio, we also included fluctuation indices of PA and NA (measured by MSSD). Mean levels of PA and NA were also included as predictors to control for the influence of these variables.

By doing so, we followed a two-step approach: In a first step, we entered all of the aforementioned predictors, in addition to the GSI, separately into single-predictor models to test whether these significantly predicted early change (hypotheses a and b). In a second step, we included all significant predictors from the single-predictor models in a combined multi-predictor model to control for mutual covariation and estimate the total amount of explained slope variation, i.e., the differences between patients' change rates from sessions 1–5 measured by the HSCL-11 (hypothesis c). All level 2 predictors were grand-mean centered in all analyses. To better understand the structure of the relation for the significant interaction, we further probed the conditional effects. We used the Johnson–Neyman technique to assess the effect of interactions (Johnson and Fay, 1950; Preacher et al., 2006). This yields an estimate of the region of significance. Regions of significance define the specific values of the moderator at which the slope of the regression changes from non-significance to significance and vice versa.

All models were estimated using the software package HLM (Version 7, Raudenbush et al., 2011).

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[1] Additionally, we estimated a linear unconditional growth model and compared its model fit with the log-linear unconditional growth model. The deviance test ($\chi^2=10.755$; $p<.001$) as well as the AIC statistic ($\text{AIC}_{\text{linear}}=184.87$; $\text{AIC}_{\text{log-linear}}=174.12$) favored the log-linear model. A quadratic model could not be estimated due to non-convergence.

[2] As the HSCL-11 is a short version of the BSI and both are highly correlated, we first entered the GSI as an intercept predictor. Because β_{00} represents the overall HSCL-11 at the first session, the GSI extracts all reliable variation when entered into the level 2 model predicting intercept; therefore, no variation remained after including the GSI and no further predictors where entered for intercept variation.

2. Results

2.1. Descriptive statistics

Of the 61 patients who agreed to participate in the study, one dropped out during the EMA period and data from two participants were lost at transfer. Of the remaining 58 participants, 50 started treatment, 11 of whom dropped out before session five, so that EMA data from the remaining 39 patients was able to be used to predict change over the first five sessions of treatment. Descriptive statistics for all variables can be found in Table 1. A GSI score of 2.08 represents the impairment level of a common outpatient sample (Lutz et al., 2015).

Mean levels of PA and NA indicate that, on average, the amount of PA outweighed the amount of NA in the sample (t (38)$= -2.65$, $p < .05$). This relative dominance of PA over NA was also indicated by an average PA/NA ratio greater than one. Fluctuations in affect were significantly higher for PA than for NA (t (22)$= -5.13$, $p < .01$).

Bivariate correlations between the EMA predictors and symptom severity at treatment onset and registration were examined to test whether the EMA measures capture independent aspects of patients' psychopathology or are just a proxy for overall symptom impairment (Table 2). Three of the EMA measures were moderately correlated to the impairment scores at the two time points. A larger PA/NA ratio was associated to lower impairment levels, whereas higher mean scores of NA and stronger fluctuations in NA (MSSD NA) were associated with higher severity levels. As such, we can conclude that some of the EMA measures do share a significant portion of variance with patients' impairment levels. However, the largest parts of the respective variances do not overlap and we can exclude that the EMA indicators are a mere proxy of symptom severity.

2.2. Unconditional growth model

To test whether indices of affective states and of their temporal dynamics can improve predictions of early response, we first modeled the course of treatment response in an unconditional model. The fixed effect estimates indicated an average HSCL of 2.08 at the first session and a mean rate of change of $-.35$ ($SE = .02$; $p < .05$) HSCL scores per $\log_{10}$ of session number. This corresponds to a mean decrease in symptoms of approximately .6 SDs over the first five sessions. Participants differed in response rates over time, as indicated by a significant random effect for the time slope ($SD = .60$; $p < .001$). As expected due to the inclusion of the GSI as a level 2 covariate, there was no significant random effect for the intercept ($SD = .01$; $p = .97$).

Table 1
Descriptive statistics.

Measures	M	SD	Min	Max
Intake				
Initial Impairment	2.08	.63	1.08	3.33
Treatment Expectation	3.00	.56	2	4
Prior Psychotherapy	2.69	1.76	1	6
Chronicity	5.54	.88	2	6
Global Assessment	59.69	8.42	45	78
EMA-Period				
PA (4 items)	2.31	1.03	1.00	4.50
NA (4 items)	1.69	.69	1.00	3.25
PA/NA	1.72	.77	.56	3.50
MSSD PA	.65	.49	.06	2.04
MSSD NA	.28	.20	.02	.89

Note. For EMA period variables, means represent averaged momentary scores across individuals and across measurement occasions. MSSD scores were calculated within days.

Table 2
Bivariate correlations between EMA measures and symptom impairment at the start of the treatment and registration.

	1	2	3	4	5	6	7
1. Impairment at treatment start	1						
2. Impairment at registration	.499*	1					
3. PA/NA	−.391**	−.419*	1				
4. NA	.404**	.438*	−.659*	1			
5. PA	−.308	−.167	.581*	−.436*	1		
6. MSSD NA (within days)	.348**	.363**	−.289	.294	−.015	1	
7. MSSD PA (within days)	.012	.120	.376**	−.271	.087	.342**	1

* $p < .01$.
** $p < .05$.

2.3. Single predictor models for early treatment response

Testing the above-described variables as predictors of early change in single predictor models, initial impairment (GSI) significantly predicted differences in patients' slopes (see Table 3). When we tested all other predictors with regard to the slope factor separately, while controlling for initial impairment, the following predictors significantly explained slope variation beyond initial impairment: mean PA/NA ratio, mean levels of NA, and fluctuation in NA. As global assessment of functioning scores (GAF), prior psychotherapy experiences, treatment expectations, chronicity of problems, time span between EMA and treatment onset, mean levels of PA and fluctuations in PA did not significantly predict

Table 3
Results of multilevel single- and multi-predictor models testing the predictive quality of intake and EMA variables for early change.

	Fixed effects		Random effects	
	Estimate (SE)	p	SD	p
Single-Predictor Models				
HSCL-11				
Intercept	2.08 (.03)	< .001	.00–.01	
GSI	.82–.91 (.05)	< .01		
Time Slope	−.35 (.11 - .12)	< .01	.53–.61	
GSI	−.51 (.18)	< .01		
Treatment expectations	−.04 (.21)	.86		
Prior Psychotherapy	−.10 (.06)	.08		
Chronicity	.09 (.08)	.45		
GAF	−.02 (.01)	.06		
Weeks between EMA and treatment onset	−.00 (.00)	.62		
PA/NA	−.36 (.10)	< .001		
MSSD_NA	.99 (.44)	< .05		
MSSD_PA	−.14 (.17)	.41		
Mean_PA	−.08 (.08)	.30		
Mean_NA	.22 (.10)	< .05		
Combined Model				
HSCL-11				
Intercept	2.08 (.09)	< .001	.00	.97
GSI	−.91 (.04)	< .001		
Time Slope	−.35 (.09)	< .001	.49	< .001
GSI	−.74 (.20)	< .001		
PA/NA	−.36 (.14)	< .05		
MSSD_NA	−.78 (.36)	< .05		
NA	−.06 (.12)	< .65		

Note. N (Level 2)$= 39$. N (Level 1)$= 195$. Time as the logarithm of session number was entered as a level 1 predictor. All level 2 predictors were grand-mean centered. Coeff.$=$Regression Coefficient.
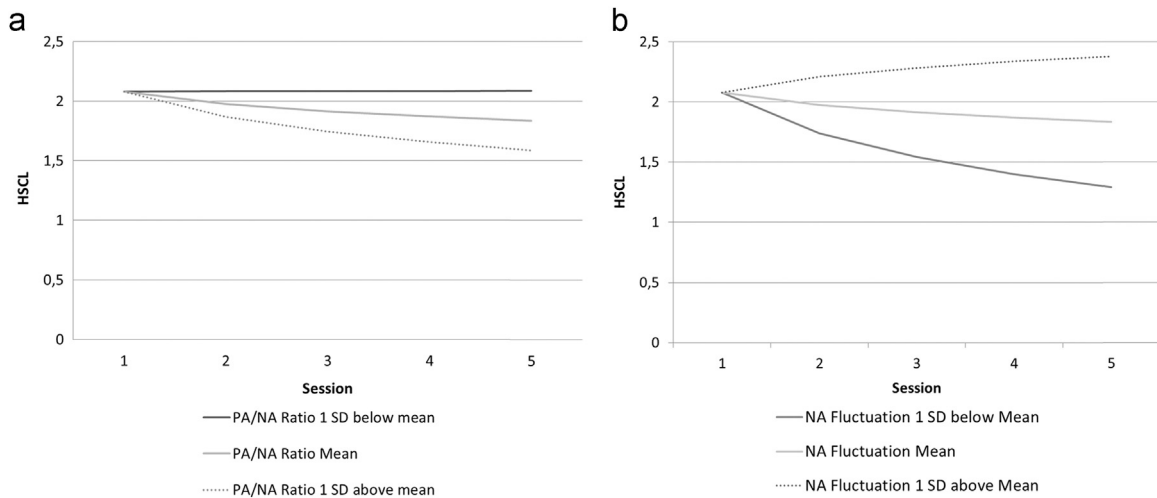* $p < .05$, *** $p < .001$.

Fig. 1. Rates of early response depending on daily life PA/NA ratios (Fig. 1a, left) and daily life fluctuation in NA measured by MSSD (Fig. 1b, right). Simple slopes are depicted for the grand-mean of the predictors (0) as well as one SD below (−1) and above (+1).

early change beyond the initial impairment scores, these variables were not integrated into the combined prediction model.

2.4. Combined model for early treatment response

The results displayed in the lower part of Table 3 indicate that besides mean levels of NA, all predictors from the previous models significantly explained variation in early change slopes. As expected from the high correlation between PA/NA ratios and NA ($r = −.659$; $p < .001$), NA did not have additional explanatory value beyond PA/NA. Table 3 indicates that patients with higher impairment levels at intake (measured by GSI scores) were estimated to improve more over the first five sessions. Higher PA/NA ratios and less fluctuation in NA (MSSD_NA) also corresponded to steeper rates of change over the first five sessions.

The relationship between the significant EMA predictors and rate of change is visualized in Fig. 1a and b. Fig. 1a indicates that patients with an average PA/NA ratio or a PA/NA ratio one standard deviation above the mean improve during the early treatment period, whereas patients with PA/NA ratios one SD below the mean do not. The opposite is true for the impact of fluctuations in NA on early response: Higher fluctuations in NA (measured by MSSD scores) correspond to slower rates of early response. Fig. 1b indicates that patients with scores one SD above mean fluctuation show increased impairment levels in the early treatment phase, whereas patients with average levels of fluctuation or with fluctuation levels one SD below the mean seem to improve.

For the standardized estimates of PA/NA ratios, the regions of significance are −6.16 at the lower bound and −.34 at the upper bound (simple slopes are significant outside this region). The upper bound corresponds to a value of 1.46 on the PA/NA predictor. This means that all patients with a PA/NA ratio of 1.46 or above are estimated to show improvements measured by the HSCL-11 over the first five sessions, whereas patients with a PA/NA ratio below 1.46 are not. 56.41% of the patients in the sample had a PA/NA ratio above 1.46. If the PA/NA ratio increases by 1 SD from the mean (corresponds to .77 points), while controlling for intake impairment, absolute levels of negative affect, and fluctuations in negative affect, the change slope becomes on average 1.8 times steeper. This steeper slope relates to about twice as much change from the first to the fifth session (average difference for a mean PA/NA ratio: .24 vs .44 for a PA/NA ratio 1 SD above the mean). This results in a standardized difference in symptom distress at session five of $d = .34$ for patients with a PA/NA ratio of 1 SD above the mean.

The regions of significance for fluctuations in NA (MSSD_NA) are −2.26 at the lower bound and .14 at the upper bound (simple slopes are significant inside this region). The upper bound corresponds to a value of .31 on the MSSD_NA predictor. This means that all patients with an NA fluctuation score of .31 or below are estimated to show improvements measured by the HSCL-11 over the first five sessions, whereas patients with an NA fluctuation score above .31 are not. 64.10% of the patients in the sample had a NA fluctuation score below .31. If the MSSD_NA decreases by 1 SD from the mean (corresponds to .20 points), while controlling for intake impairment, absolute levels of negative affect, and the PA/NA ratio, the change slope becomes on average 1.4 times steeper. This steeper slope relates to about 1.4 times higher symptom change scores from the first to the fifth session (average difference for a mean MSSD_NA score: .24 vs .35 for a MSSD_NA score 1 SD above the mean). This results in a standardized difference in symptom distress at session five of $d = .19$ for patients with a MSSD_NA score of 1 SD above the mean.

In order to compare the incremental predictive value of the real-time EMA measures with regard to the explanation of early response, we calculated the proportionate reduction of residual slope variance (Raudenbush and Bryk, 2002). First, we included the only significant intake predictor (intake impairment levels measured by GSI) and compared this model to the unconditional model. By including GSI scores, the slope variance was reduced by 16.10%. We then compared our full model with the more restrictive model encompassing only the intake predictor to test how much additional variance was explained by the EMA predictors. By entering the significant EMA predictors, a further 21.73% of the variation in slopes was explained.

3. Discussion

The prediction of early treatment response is an important goal of psychotherapy research. Prior research has shown that severity assessments at intake provide some indication of the expected course of improvement early in therapy (Beutler et al., 2006; Clarkin and Levy, 2004; Cuijpers, et al., 2005; Rubel et al., 2015; Stulz et al., 2007). In this study, we found that the assessment of affective states and dynamics in daily life prior to treatment allows for incremental prediction of early treatment response. Specifically, we showed that a) mainly the interplay of PA and NA – measured by PA/NA ratios – and not that much their sheer mean levels predicted early treatment

response. While controlling for initial impairment levels, negative affect, as well as fluctuations in negative affect, patients with higher PA/NA ratios showed steeper early change slopes. In addition, we showed that b) the inclusion of an index of daily life NA fluctuation improved predictions of early response beyond PA/NA ratios, with greater NA instability being tied to poorer response. In contrast, inclusion of an index of PA fluctuation had no such incremental contribution. Finally, we demonstrated that c) the affect dynamic predictors had incremental predictive power beyond intake measures, thereby improving prediction models of early response.

As we pointed out, there is existing literature on how intake indicators predict the course of treatment (e.g., Beutler et al., 2006; Clarkin and Levy, 2004; Cuijpers, et al., 2005; Rubel et al., 2015, Stulz et al., 2007). However, they all leave a substantial amount of unexplained variance. Previous studies have shown that especially initial impairment levels predict differences in treatment response (e.g., Rubel et al., 2014). Specifically, patients with higher impairment levels at intake showed more pronounced early positive changes. One potential explanation for this result is rooted in the fact that those patients with more distress simply have more room for improvement. Additionally higher initial scores are accompanied by more regression to the mean, which makes it indispensable to control for initial impairment scores when testing additional predictors of early change patterns. Surprisingly, in the current study, when controlling for initial impairment, none of the other tested intake variables that have shown to be predictive for treatment response in prior research (treatment expectations, prior psychotherapy, chronicity, and GAF) could explain a significant amount of early change variation (e.g., Lutz et al., 2007). This result is in line with the notion that it is hard to find predictor variables that can add something beyond the initial impairment score. Even more it seems to be important to leave the traditional routes of psychotherapy prediction models. If we want to understand more about patient characteristics that influence the course of treatment early in its process, we must assess pieces of individual information that go beyond the information that can be assessed by established diagnostic instruments. Dynamic, intra-individual assessment could help "to parse the shared versus unique variance [...] across individuals" (Fisher, 2015, p. 10). This knowledge could help to individualize treatment approaches early in the therapeutic process in order to make interventions maximally efficient – and thus effective – for the individual patient.

The single predictor models revealed the PA/NA ratio, absolute levels of NA, and fluctuations in NA (MSSD_NA) as EMA variables that explained significant early change variation beyond the initial score whereas absolute levels of PA and fluctuations in PA did not. These results are in line with the findings reported by Forbes et al. (2012) who neither did find a significant association between mean PA levels or fluctuations in PA and change over the course of the treatment. Adding the significant predictors from our single predictor models in a combined model, only fluctuations in NA and the PA/NA ratio significantly predicted early symptom change. This result deviates from the findings of Forbes et al. (2012) who also found a significant influence of absolute levels of NA even when controlling for the PA/NA ratio. However, this difference could be a result from the different time spans which are predicted in the two studies. While the current study is focusing on early change, Forbes et al. (2012) predicted change over the whole course of the treatment.

The strongest EMA predictor in the current study was the PA/NA ratio. If the PA/NA ratio increased by 1 SD from the mean (corresponds to a ratio in which PA outweighs NA by about 2.5 times), while controlling for intake impairment, absolute levels of negative affect, and fluctuations in negative affect, patients showed about twice as much change within the first five sessions compared to patients with an average PA/NA ration (i.e., a ratio in which PA outweighs NA by 1.7 times). These results support our

hypothesis that patients' with higher PA/NA ratios, which reflect higher levels of optimism and emotional well-being, can faster enter the phase of remediation in which, according to the phase model (Howard et al., 1993) symptom reduction takes place. Another explanation is based on the broaden-and-build model by Fredrickson (2000a, 2000b), in which negative affect is assumed to be associated with a limited behavioral repertoire in a given situation and positive affect is assumed to loosen this influence of negative affect. Along these lines, positive affect is associated with approach, whereas negative affect is associated with withdrawal tendencies. Important is the interrelation of PA and NA: PA loosens the influence of NA on the person and broadens the behavioral repertoire by enhancing physical, social, and intellectual resources (Fredrickson and Branigan, 2005). A low ratio can arise within a person who has both strong approach and withdrawal tendencies as well as within a person who has both weak approach and withdrawal tendencies. As a consequence, to overcome the withdrawal tendencies connected to NA, more PA in relation to NA is required as compensation. We found that in order to profit from treatment early, PA must to outweigh NA by about 1.5 times over the EMA period. Lower ratios might represent a clinical state in which patients are not yet ready to approach or implement the change processes offered in therapy due to too few approach compared to withdrawal tendencies and/or a limited behavioral repertoire. With this knowledge, therapists can tailor treatments in accordance to the specific needs of the individual patient. That is, therapists are better informed of whether their patients are ready to profit from the respective, e.g. cognitive, interventions, or if it is first necessary to increase positive or decrease negative affect with interventions especially designed for that purpose.

The study of affect dynamics has focused on different aspects (investigated by different indicators) and their consequences for psychopathology and psychotherapy. The following variables were among the different aspects investigated by previous studies: variability (e.g., Koval et al., 2013; Thompson et al., 2012), instability (e.g., Farmer and Kashdan, 2014; Koval et al., 2013; Trull et al., 2015; Wichers et al., 2010), inertia (e.g., Koval et al., 2012; Kuppens et al., 2010, 2012; Trull et al., 2015), and differentiation (e.g., Kashdan et al., 2014; Trull et al., 2015; Zaki et al., 2013). These accumulated findings have shown that instability in NA – both in daily life and in the lab (Koval et al., 2013) – is associated with psychopathology, as well as symptom and treatment courses. In line with these research findings – and although fluctuation scores for PA were significantly higher than for NA – fluctuation in NA proved to be a more informative indicator with regard to early response in our sample. Our results highlight that fluctuation in NA can not only predict relapse in remitted depressed patients as shown by Wichers et al. (2010), but also early response. Fluctuation in NA does not only differentiate between clinical samples and healthy controls (Thompson et al., 2012) and proves to be a good indicator for relapse in specific diagnostic subgroups, namely depression (Wichers et al., 2010), but additionally proves to be a significant predictor of treatment response with transdiagnostic relevance. The assessment of NA fluctuation before the onset of treatment may help to elucidate an important aspect of psychological functioning in patients, regardless of their diagnoses.

Several limitations of this study should be acknowledged. First, this study's sample size did not allow a comparison between different diagnostic subgroups. The majority of the sample was diagnosed with an affective or anxiety disorder. In separate analyses, we controlled for depressive symptoms to ensure that the relation between PA/NA dynamics and early treatment response was not limited to depressed patients. Although it was not, we cannot ensure that the findings are comparable for every diagnostic subgroup and generalize to other psychological disorders. The small sample size, the accompanied lack of power and lack of full range of scores on the

respective predictors may also be a possible explanation for the result that the intake predictors, which explained slope variation in previous studies, did not turn out to be significant in our analyses. However, the direction of influence of these indicators on the slope factor was as hypothesized.

Second, it should be noted that the EMA assessment and the treatment was temporally very far apart (on average 2.3 month). This large time span might result in a decrease of the predictive power of the EMA predictors. Stronger associations might have been found between these predictors and early treatment response if treatments had start directly after the EMA assessment phase. This might also explain why in comparison to Forbes et al. (2012) we did not find a significant relation between absolute levels of NA and early change if added in a combined model with the PA/NA ratio. In Forbes et al. (2012) the EMA assessment was conducted the weekend before treatment began.

Third, even if the field of electronic devices that can be utilized for EMA develops rapidly, lowering the cost of its application, EMA still appears somewhat resource demanding, not especially with regard to the financial burden associated with the equipment, but with regard to time, personnel, and patient investment. Patients need to be instructed on how to handle the electronic device used for EMA and the generation and interpretation of relevant data outputs requires technical and statistical methods, which may not be available to every clinician. However, there are promising developments in the field of electronic devices that come with integrated routines to generate relevant output that can be used for assessments, monitoring and also for patient feedback (e.g., Wichers et al., 2011a, 2011b). Developments of this kind would enable more clinicians to integrate EMA strategies into their diagnostic routines. Future research should focus on replicating the results with regard to the feasibility of EMA predictors for early treatment response. There are preliminary indications of satisfying feasibility and acceptance (86.6% of the study participants indicated that they were not at all or only slightly burdened by the assessments and 40% even indicated that the assessments had moderate or strong positive effects on their mood), however these results must still be tested in different settings to ensure broad applicability in the field of patient focused psychotherapy research. If our results can be replicated and EMA indicators prove to be good predictors of early treatment response, the next step would be to integrate EMA periods during later treatment phases as additional instruments to assess treatment effects and evaluate treatment courses. Similarly, future studies should test whether the two EMA indicators we applied help to enhance prediction models for ultimate treatment outcome.

In closing and despite the mentioned limitations, our findings suggest that the integration of methods that allow the assessment of patient characteristics, which go beyond regular intake measures, broadens our knowledge of patient variables and helps explain differences in early treatment response. In line with many other authors (e.g., Barret et al., 2007; Greenberg, 2012; Kring, 2010; Rottenberg and Gross, 2007), our results underline the centrality of affect in psychopathology and processes of change within psychotherapy. The assessment of patterns of negative and positive emotions in daily life may help therapists gain insights into the psychopathology of a particular patient, thus possibly improving decision-making regarding treatment choices (Shiffman et al., 2008). In order to ensure the best possible patient-centered treatment, the integration of EMA strategies into the diagnostic process before the onset of treatment seems to be a promising endeavor.

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Conflict of interest

None.

Author's contributions

Authors 1, 3 and 5 designed the study and wrote the protocol, author 2 supported the process with valuable experiences in EMA applications. Author 1 managed the data collection. Authors 1 managed the literature searches and analyses. Authors 1, 3 and 5 undertook the statistical analysis, and author 1 wrote the first draft of the manuscript. All authors contributed to and have approved the final manuscript.

Acknowledgments

Appendix A

EMA ITEMS

Negative affect

1. „At the moment you feel depressed?"
2. „At the moment you feel ashamed?"
3. „At the moment you feel anxious?"
4. „At the moment you feel nervous?"

Positive affect

1. „At the moment you feel excited?"
2. „At the moment you feel determined?"
3. „At the moment you feel alert?"
4. „At the moment you feel active?"

Answers

$1 =$ "slightly or not at all",
$2 =$ "a little",
$3 =$ "to some extend",
$4 =$ "considerably",
$5 =$ "extremely".

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