Daily Fluctuation of Emotions & Memories Thereof: Design and Methods of an Event Sampling Study of Major Depression, Social Phobia, and Controls

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Abstract

Symptom fluctuations and the dynamic contexts provoking these are poorly understood. This deficit is compounded by people's limited ability to accurately report about such dimensions in retrospect. Utilizing the advantages of event sampling methodology (ESM), this study rigorously describes and tests proximal environmental, neurobiological and psychological factors associated with symptoms and mood states.

Participants were assigned to three diagnostic groups: Major Depressive Disorder (MDD;n=118), Social Phobia (SP;n=47), or a Control Group without SP or MDD (CG;n=119). Laboratory assessments included cognitive abilities, memory, constructs, and BDNF. ESM lasted seven days, with six assessments per day covering symptoms, affect, daily events, social interactions, post-event processing, well-being, etc. Morning cortisol and actigraphy were also assessed during ESM. Thereafter, participants provided subjective retrospective recall estimates of the emotions they reported during ESM.

The multi-level data of >10,000 observations will allow for thorough examination of fluctuations of psychopathology and well-being in two highly prevalent disorders. Using two clinical groups *and* a non-affected control group, the clinical specificity vs. generalizability of processes can be directly tested, thus providing stimulating information about the overlap and differences between anxiety and affective disorders. This research informs about the development, fluctuation, and maintaining factors of emotions and symptoms and examines the accuracy with which participants recall these dimensions.

Keywords: fluctuation, memory-experience gap, ESM, depression, social phobia

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Variability and fluctuation of mental states constitute a fundamental, yet relatively neglected dimension of psychopathology. That emotions fluctuate is a self-evident quality of human nature. In contrast, affected individuals and researchers alike poorly understand the timing, antecedents, consequences, and processes associated with these fluctuations. As a result, much remains to be learned about the factors that maintain and ameliorate symptomatology as well as potentiate positive states and desirable actions. This form of knowledge seems to be crucial for the development of better and more effective preventive or early treatment interventions, with the potential to reduce the incidence as well as the persistence of harmful emotional and behavioural syndromes.

Examination of emotional fluctuations requires longitudinal methods that are temporally sensitive enough to capture the fluctuations. Towards this end, experience sampling methodology (ESM; Csikszentmihalyi & Larson, 1987; Stone & Shiffman, 1994; Stone, Shiffman, Atienza, & Nebeling, 2007) has come to be considered the method of choice. ESM stresses the reporting of phenomena in their natural context and close to the time of occurrence. These data allow for fine-grained temporal examination of the intra- and inter-psychic stimuli that give rise to symptom fluctuation, where both antecedents and consequences can affect expression (Ebner-Priemer, Eid, Kleindienst, Stabenow, & Trull, 2009).

ESM studies have generated unique and important insights into psychopathology not accessible to cross-sectional methods. For example, contrary to prevailing clinical theory, ESM data revealed that patients with depression react *more positively* than healthy controls following positive events (Bylsma, Taylor-Clift, & Rottenberg, 2011; Peeters, Nicolson, Berkhof, Delespaul, & deVries, 2003). This seemingly counterintuitive effect is referred to as

"mood brightening". Likewise, examination of the longitudinal nature of the data revealed that people who have greater emotional consistency across data points (referred to as emotional inertia) are associated with more disability and even subsequent onset of disorders (Kuppens, Allen, & Sheeber, 2010; Kuppens et al., 2012). It has been hypothesized that those who react with more inertia are insensitive to the contextual variables that help make life colourful and thus miss the reinforcing value of a multitude of activities (Rottenberg, Gross, & Gotlib, 2005). These are but a few examples of recent research insights won from ESM (for an overview see Trull & Ebner-Priemer, 2013).

Memory Experience Gap

Research on processes such as mood brightening and emotional inertia represent a methodological challenge, given people's general inability to precisely report about the fluctuations of their daily mental states and experiences. Research suggests that people's memories of experiences and moods differ from the actual occurrences and these discrepancies are believed to reflect different sources of knowledge: a person's experienced emotion vs. a person's belief about emotion which is driven by complex cognitive-affective schemata (Beck & Haigh, 2014; Conner & Barrett, 2012; Robinson & Clore, 2002). This results in a memory-experience gap, defined as the discrepancy between the average of experienced emotions and the overall retrospective evaluation of the experience (Miron-Shatz, Stone, & Kahneman, 2009). This gap can lead to overestimating or underestimating the frequencies or intensities of experiences and behaviours and has been found to be greater for negative mood than positive mood (Miron-Shatz, 2009). Given the prominence of negative affect in people diagnosed with mental disorders, it is probable that the memory-experience gap is especially pronounced in these individuals. To date, however, this has not been directly tested as previous studies worked with analogue samples. Further, it remains unclear whether the memory-experience gap is limited to recall of frequencies and intensities, or if it extends

into how well people diagnosed with mental disorders understand the correlates and determinates of their own behaviour. Stated differently, how well can people accurately report on the covariation of their own behaviour (e.g., I feel better when I sleep more)? Or is the reporting of this type of self-knowledge equally impeded by the memory-experience gap? Evidence from one study in a clinical population suggests that people diagnosed with mental disorders are not very accurate in reporting this type of self-knowledge (Gloster et al., 2008). This represents a potentially systematic bias inherent in clinical interactions. It needs, however, to be emphasized that testing across diagnoses and clinical severity is an imminent research necessity that is lacking to date.

Social Interaction

Social relationships and interactions are powerful factors associated with well-being (Kawachi & Berkman, 2001; Thoits, 2011). Indeed, social interactions are central to human existence. Unfortunately, mental disorders are often associated with problems in this area. This is especially true with Major Depression (MDD) and Social Phobia (SP), which are also two of the most frequent and impairing mental disorders at both the individual and societal levels (Wittchen et al., 2011; World Health Organization, 2001). For example, people high in depression have been observed to have fewer and more negative social interactions than people low in depression (Brown, Strauman, Barrantes-Vidal, Silvia, & Kwapil, 2011). In SP, social interactions are the focal stimuli that elicit the fear response and avoidance behaviours reflected in the diagnostic criteria (American Psychiatric Association [APA], 2000) and are associated with multitude of impairing sequel (Kashdan & Hofmann, 2008). Despite these associations, the mechanisms of action that maintain problems in social domains remain unclear, especially outside the laboratory and in the context of peoples' natural context.

Towards this end, context-sensitive theories may be especially poised to contribute to the understanding of social interactions across groups. Navigating between appetitive and

aversive stimuli is a task everyone engages in every day. One's well-being depends in part on the degree to which one appropriately understands, reads, selects, and responds to their environment. For patients and healthy individuals alike, context sensitivity is thus important. Indeed, it is seen as a fundamental ability that contributes to psychological health (Kashdan & Rottenberg, 2010) and deficits in this skill are associated with depression (Rottenberg et al., 2005). Given the centrality of social interactions in MDD and SP, further research is needed to examine how naturally chosen/avoided social interactions as well as participants' reactions to them as experienced in every day life (Farmer & Kashdan, 2012). Research in SP suggests that post-event processing (or the rumination of predominately negative aspects of a social interaction after it has concluded) may be especially salient in the maintenance of social anxiety and cross-sectional evidence suggests that it is uniquely associated with SP (Fehm, Schneider, & Hoyer, 2007; Hoyer, Braeuer, Crawcour, Klumbies, & Kirschbaum, 2013). We are unaware, however, of a context dependent and temporally sensitive examination directly comparing SP with other groups. This type of study is therefore needed in order to properly test this claim.

Additional Targets

Several other salient variables, both biological and psychological, have been implicated in mental disorders, but have yet to be examined with respect to their influence on the natural fluctuation of symptoms or the memory-experience gap. For example, sleep disturbances are one criterion of MDD (American Psychiatric Association [APA], 2000) and it has been shown that sleep affects neurocognitive and emotional processes (Walker & Stickgold, 2006) and is affected by antecedent daytime experiences (Brand et al., 2010a, 2010b). Sleep is important for processing of declarative and emotional memory traces (van der Helm & Walker, 2011), which may subsequently affect day time functioning and their memories. It remains unknown how sleep contributes to symptom fluctuation and

responsiveness to stimuli, or whether such effects differ between people afflicted by MDD, SP, and non-affected individuals. Similarly, neurobiological processes have been associated with depression but their impact on symptom fluctuation and event-related responses remains unknown. For example the cortisol awakening response (CAR) is a proxy marker of hypothalamic-pituitary adrenal (HPA) axis functioning (Chida & Steptoe, 2009; Fries, Dettenborn, & Kirschbaum, 2009) and it may both depend on and affect symptom fluctuations. Likewise, serum levels of brain derived neurotrophic factors (sBDNF), a marker for neuroplasticity (Duman & Monteggia, 2006), are associated with more severe psychopathology, while higher levels predict flexible neurocognitive functioning (Mikoteit et al., 2014; Mikoteit et al., 2015). Thus, neuroplasticity may play a role in flexible adaptation to psychosocial contexts, but this has yet to be empirically tested.

At the psychological level, psychological flexibility is a salient transdiagnostic variable associated with MDD and SP as well as controls. Psychological flexibility (i.e., inter- and intrapersonal skills that help a person be open to and present with their experiences and surroundings so that they can pursue the things they choose to care about) is considered a basic component of mental health and well-being (Gloster, Klotsche, Chaker, Hummel, & Hoyer, 2011; Huppert, 2009; Kashdan & Rottenberg, 2010; Keyes, 2005) implicated in treatment process and outcome (Gloster et al., 2014; Gloster, Sonntag, et al., 2015; Hayes, Luoma, Bond, Masuda, & Lillis, 2006) as well as genetic analyses (Gloster, Gerlach, et al., 2015). Some context sensitive studies have shown that psychological flexibility moderates the relationship between daily levels of social anxiety and positive events (Kashdan & Steger, 2006). It remains to be tested, however, to what degree psychological flexibility moderates fluctuations in MDD and non-affected individuals.

Study Aim

Taken together, the importance of systematically examining factors associated with finer-grained temporal fluctuations is manifold. This study had two primary aims. First, the understanding of the contexts and correlates that give rise to moment-to-moment expression provides a level of detail that can be directly tied into knowledge about symptom maintenance and the provision of early interventions. Refinements in psychopathological conceptualization require an understanding of the various conditions under which phenotypic expression unfolds. Second, by collecting ESM data we will be able to document the factors that increase the memory-experience gap. This is potentially of large importance for mental disorders with prevalent negative affect such as MDD and SP. Testing and documenting the extent of this effect across disorders will help identify and subsequently refine systematic bias in clinical assessment. This paper describes in detail the methods used in the study.

Beyond the global aims of understanding fluctuations and the memory-experience gap, the study described in this method paper was also designed to test numerous research questions beyond these global aims using the large, multi-level, and multi-trait data set (see Table 1).

--INSERT TABLE 1 ABOUT HERE--

Method

Design

This was a quasi-experimental, intensive, longitudinal study with diagnostic status of group as the quasi-experimental factor. The study was conducted over two weeks in both the laboratory and participants' natural environment. Data collection occurred between May 2014 and August 2016.

The local ethics committee approved the study (Approval # EKBB 236/12) and all participants completed an informed consent procedure.

Recruitment and Selection Criteria

Participants were recruited from treatment centres (university clinics and cooperating local practitioners) in Switzerland and Germany. Participants were additionally recruited through internet advertisements.

All participants recruited for the project underwent a standardized diagnostic clinical assessment (Structured Clinical Interview for DSM-IV Axis I Disorders [SCID]; First, Spitzer, Gibbon, & Williams, 1995). The participant's primary diagnosis determined eligibility and group assignment: a) MDD, b) SP, or c) participants with neither MDD nor SP. The other inclusion criterion was age (18-65). Exclusion criteria were: active current suicidal intent, current substance dependence, inability to understand German, and physical disabilities prohibiting participation such as inability to see text in a smartphone or to hear the smartphone's signal.

Sample Size

We based the power calculation on the hypothesis believed to have the lowest effect size (i.e., between group comparisons involving the SP group) and it thus was the limiting factor in determining the sample size. As a predefined constraint we considered the maximum number of SP patients that could feasibly be recruited within the study period to be no more than 48. Assuming a dropout rate of 5%, this lead to an expected number of patients to complete the study of 45. This number was used for the power analyses which always assumed alpha=.05, power=.8, and a two-sided test.

The limiting model of comparisons between groups included hypotheses regarding retrospective recall, where the dependent variable is a difference score between ESM data and recalled estimates of these data. Based on a t-test for independent samples, a medium effect size (d=0.5), and 45 subjects in the SP group, the sample size necessary to achieve .8 power was 111 subjects in each of the other groups (MDD & controls). Assuming a 5% dropout rate,

117 subjects were targeted in each of these two groups. Thus, comparisons between the SP group and any of the two other groups (MDD or controls) will have enough power for effect sizes of 0.5 or higher, and comparisons between MDD and controls will have enough power for an effect size of 0.38 or higher.

Participants

In total, *n* = 290 participants were included in the study. Participants were matched by age and sex across the three groups. Six participants did not complete at least 50% of the ESM time points and were subsequently removed from the data set. The final sample therefore consisted of *n* = 284 participants: MDD, *n*= 118; SP, *n* =47; Control, *n* = 119. On average the sample was 31.75 years old (range 18 – 63), and 66.5% female. Other demographic information can be seen in Table 2.

--INSERT TABLE 2 ABOUT HERE--

Assessors

Assessors were psychology graduate students. Students were trained to competency and supervised weekly by the principle investigator.

Assessments

Assessments for both laboratory-based questionnaires and ESM targeted the same domains, including symptomatology, affect, social interactions, health behaviours, well-being, and prosociality.

--INSERT TABLE 3 ABOUT HERE--

Psychological Variables. Psychological constructs and questionnaires used in this study and the timing of administration can be seen in Table 3.

Neuropsychological Tests. In order to control for possible diminished memory secondary to the disorder of interest, brief neuropsychological measures of executive function were included that have been found to be affected (i.e., shifting & verbal working memory)

and unaffected (i.e., vocabulary) by depression (Snyder, 2013). For this purpose, working memory and shifting was assessed using subscales from the Wechsler Adult Intelligence Scale (WAIS-IV; Wechsler, 2008) (i.e., Working Memory & Backward Digit Span) as well as the Trails Making Test (TMT; Battery Army Individual Test, 1944). Vocabulary was assessed using the Vocabulary subscale from the WAIS-IV (Wechsler, 2008).

Biological Assessments.

Sleep. Parallel to ESM, sleep was objectively assessed with actigraphy over seven nights in combination with a sleep log. Actigraphy was recocorded using a digital movement-measuring instrument the size of a wristwatch (actigraph; Somnowatch^{TM}; Somnomedics, Randersacker, Germany) on the wrist of the non-dominant hand. This tool registers every movement above 0.012 g in a bi-axial direction in 30-s intervals. Following Sadeh, Raviv, & Gruber (2000), the following sleep continuity parameters were computed: sleep onset latency (SOL); morning awakening time defined as the last minute asleep; the number and the times of awakenings after sleep onset (WASO); sleep period time (SPT; i.e., sleep time (minutes) from SOL to morning awakening). The following dependent variables were derived from these parameters: total sleep time (TST; i.e., number of minutes of sleep time excluding all time awake); SOL, SPT, time and number of awakenings after sleep onset (WASO), and the sleep efficiency (SE), i.e., the ratio of TST to SPT.

HPA-system. The cortisol awakening response (CAR) has been shown to be a reliable index of basal HPA axis activity (Pruessner et al., 1997). On two days of the week (one workday and one weekend day), four saliva cortisol samples were taken (0,10, 20, and 30 min immediately after awakening; for more details see (Hatzinger et al., 2013). Participants were instructed to start saliva sampling immediately after awakening without first rinsing their mouth with water. Further, participants were instructed not to eat breakfast or to brush their teeth before sampling was completed. To facilitate verifying the exact timing of the saliva

sampling after awakening, we instructed participants to immediately record each of the four time points of saliva collection in a special log that we provided together with the collection tubes/swabs. Using actigraphy data we will be able to objectively determine the time of awakening. The objective actigraphy data will be compared to the sleep log and related salivary collection times. Using this information, we are in the position to follow recommendations of Smyth et al. (2016) during later analyses, such as excluding cases with a sampling delay exceeding 15min, or – if delay was by less than 15min – mapping the cortisol growth curve on real verified sampling times applying multilevel linear modelling techniques.

Saliva samples were obtained using the "Salivette" device for quick and hygienic sampling (Sarstedt, Nümbrecht/Germany). This device includes a small cotton swab on which the subject gently chews for 0.5-1 min. Thereafter, the swab was transferred into a small plastic tube, the Salivette container, and stored in the freezer. Saliva samples were returned to the laboratory where they were centrifuged at 4 °C (2000 rpm, 10 min) and stored at -20°C until assay. Free salivary cortisol concentrations were analysed using a time-resolved immunoassay with fluorometric detection "Coat-ACount" Cortisol RIA from DPC (Diagnostics Products Corporation; Biermann GmbH, Bad Nauheim, Germany) as described in detail elsewhere (Dressendorfer, Kirschbaum, Rohde, Stahl, & Strasburger, 1992). Intra- and interassay variability of this assay was less than 2.00% and 2.04%. The hormone concentration of the morning cortisol samples at 0, 10, 20 and 30 min after awakening was computed as the area-under-the-concentration-time-curve (AUC; using the trapezoidal integration [Forsythe, Keenan, Organick, & Sternberg, 1969]). The AUC_{total} refers to the entire amount of cortisol concentration under the time curve, whereas the AUC_{basal} describes the initial and averaged amount of cortisol secretion over time, as if the HPA-axis had not been stimulated. Accordingly, AUC_{net} reflects the difference between AUC_{total} and AUCbasal.

Serum levels of BDNF. For assessment of BDNF serum concentration, blood samples were collected between Times 1 and 2 in the morning (before 10 a.m.). Laboratory analysis was performed with a BDNF Emax immunoassay kit (Promega, Duebendorf, Switzerland) described elsewhere (Mikoteit et al., 2014). The specificity of sBDNF analysis (i.e. cross-reactivity to related neurotrophins) was <3% and sensitivity was at least 15 pg/ml.

Event Sampling Methods. ESM items were administered via smartphone six times per day queried about emotions, symptoms, and events. Participants responded to 92.3% of the prompted ESM assessments, for a total of n = 10,979 assessments. Of these, 51 were only partially completed. Thus, n = 10,928 assessments (91.9%) were responded to and fully completed. Item stems read, "Since the last beep (about the last three hours)…" followed by the content specific items. The "percentage of time" approach was used to estimate the amount of time a participant engaged in or experienced various emotions (e.g., "…what percent of the time did you …"). This approach is preferable to querying for absolute frequencies or durations when symptoms lack a clear beginning or end (i.e., I felt tired, I lacked energy, etc.) (Schimmack, Oishi, Diener, & Suh, 2000).

Both disorder-specific items and processes common across diagnostic groups were administered. The items were derived from previous ESM studies (Brown et al., 2011; Gloster et al., 2008; Kashdan & Steger, 2006), established questionnaires, and self-developed items. Items from established questionnaires that were modified for the ESM format were only changed to reflect the time frame since the last beep (e.g., "since the last beep…"). In order to limit participant fatigue that can ensue with frequent repeated measurements, some scales were shortened. In these cases, items with the highest item-total correlations were chosen while simultaneously ensuring minimal item overlap. The ESM items assessed the same domains as the questionnaires (see Table 3). The established measures from which the ESM items largely stemmed included measures of depression (Beck, Steer, & Brown, 1996),

affect (Watson, Clark, & Tellegen, 1988), sleep (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), post event processing (Fehm, Hoyer, Schneider, Lindemann, & Klusmann, 2008; Sierra & Berrios, 2000), social anxiety (Rodebaugh et al., 2004), and psychological flexibility (Benoy, Knitter, Doering, Knellwolf, & Gloster, 2017). Additionally, health related items were derived from standard questions in epidemiological surveys (Bundesamt für Statistik [BFS], 2013) and social interactions were assessed using a functional analysis framework (e.g., frequency & quality of interactions; antecedents & consequences).

Study Procedure

The data were collected over 2 weeks with observations in 7-day intervals. Time 1 observations occurred on the first day of participation following the screening. Time 2 observations occurred on the 8th day of the study. ESM assessments were conducted between Time 2 and 3. Time 3 observations occurred on the 15th day of the study (see Figure 1). Each point of contact is described in detail below.

- INSERT FIGURE 1 ABOUT HERE -

Screening. Participants were informed of their research rights prior to data collection. Participants were screened for the presence of SP, MDD, or neither of the two disorders using the screening module from the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First et al., 1995) plus two additional screening questions for core diagnostic symptoms. Participants that screened positive were invited to the Time 1 assessment.

Time 1 (diagnostic and baseline assessment). Participants completed informed consent procedures prior to data collection. Following signing, all participants underwent a standardized diagnostic clinical assessment (i.e., SCID) to determine diagnostic status. All participants also answered demographic questions, brief neuropsychological measures of executive function believed to be clearly affected (i.e., shifting & verbal working memory)

and unaffected (i.e., vocabulary) by depression (Snyder, 2013); and completed diagnostic-specific and transdiagnostic questionnaires (see Table 3).

Participants whose diagnostic status was confirmed (i.e., MDD, SP, or neither of the two disorders (i.e. controls)) were scheduled for ESM training at Time 2.

Time 2 (ESM training). One week later, participants met with an assessor and completed diagnostic-specific and transdiagnostic questionnaires a second time.

Participants then received a smartphone and were instructed in its use. Data was recorded by touching the screen. Training included a demonstration of how to operate the smartphone, initiate data recording, record data, and recognize the signalling tone. Participants practiced recording data, had the opportunity to ask questions, and were informed that the smartphone time stamps all responses. They were instructed on how to record any difficulties or malfunctions with the device.

Ambulatory Monitoring (ESM between Time 1 & 2). The ambulatory assessment took place in the week interval between Times 2 and 3 (days 8 to 15 of the study). Participants completed questionnaires on the smartphone contingent on an audible signal at 6 fixed times throughout the day, every 3 hours (e.g., 8 a.m., 11 a.m., 2 p.m., 5 p.m., 8 p.m., & 11 p.m.) The ESM-survey was separated into a morning, a day, and an evening questionnaire (i.e., last of the day). The morning questionnaire was to be filled out at the first prompt of the day. The day and evening questionnaires were identical except for a set of questions in the evening questionnaire that asked participants to make judgments about their entire day and commitments for the next day. Consistent with previous research (Gloster et al., 2008) participants were contacted during the 1st, 3rd, and 6th days of ESM self-monitoring to address any questions regarding the use of the smartphone and increase adherence.

Time 3 (retrospective recall and correlation estimation). On the last day of the study, participants returned the smartphone and completed the questionnaires a final time.

Participants then estimated the total percentage of time they engaged in the focal symptoms queried by the smartphone over the preceding week. For example, participants were asked, (a) "In the last week (when you used the smartphone), what percentage of time did you…" and (b) "On average, how long did you spend doing …each day?"

After estimating the percentage of time for ESM symptom items, participants completed a graphically supported, interactive, 27-slide PowerPoint didactic on the meaning of a correlation used in previous research (Gloster et al., 2008). The purpose of the didactic was to teach participants the meaning of correlation so they can reliably estimate correlations between symptoms and supplemental variables (e.g., amount of sleep, mood, etc.). Participants learned two major concepts: direction of relationship (i.e., positive, negative, and zero relation) and magnitude of relationship (i.e., strong, mild, weak). To simplify the learning process, participants learned correlations using a -100 (perfect negative correlation) to +100 (perfect positive correlation) scale instead of using decimals. Further, a visual analogue scale with a color-coded, double-pointed arrow was utilized to facilitate learning. Labels reading "positive correlation", "negative correlation", and "no correlation" anchored the arrows. Other descriptive labels further clarified the meaning of a given correlation's direction and magnitude. Embedded in the tutorial were three concept quizzes/ test questions to assess comprehension. Participants were required to successfully answer these questions before continuing. After completing the tutorial, participants estimated correlations between selected symptoms and transdiagnostic/ supplemental ESM items encountered during the past week. Participants estimated correlations using the same color-coded visual analogue scale used in training. For each item, the stem sentences learned during the tutorial were then adapted for the specific content of that question and appeared right below the arrow (e.g., "As

sleep per night went up, depressive thoughts went up" and "As sleep per night went up, depressive thoughts went down"[1]).

In order to explore the memory-experience gap, participants estimated both disorder-specific correlations and non-specific variable pairs. Order of presentation was randomized across item content types and assumed strength of relationship. In addition to the correlation estimations, participants also made judgments about the same item pairs using a Likert scale. Presentation order of format (correlation vs. Likert) was also randomized.

Participants then rated their confidence in their memory, perceived accuracy in symptom recall, and perceived accuracy in correlation judgments. Finally, in a follow-up questionnaire they were asked about their awareness of the study's hypotheses.

Data Cleaning

Data cleaning involves the identification of univariate (z-scores) and multivariate outliers (Cook's distances). Regarding missing values we will first check the extent to which these may bias results by computing absolute and relative frequencies of missing values for all involved variables and test whether a missing completely at random (MCAR) pattern is feasible or not. If MCAR holds across all involved variables and the percentage of missing values is low (<5%), statistical models are applied without special consideration. If MCAR does not hold and/or the percentage of missing values is high (≥5%) either of two methods to deal with missing values will be used: full information maximum likelihood estimation or multiple imputation (Schafer & Graham, 2002). Continuously distributed outcome variables will be checked for normality and homoscedasticity and transformed if necessary prior to any analysis.

Analysis Plan

[1] An example can be seen in the supplementary material.

Research questions involving non-hierarchical/independent data will be analysed using traditional regression analysis techniques such as single or multiple linear regression models and analysis of variance techniques. Dichotomous outcomes will be analysed using logistic regression models. More complex research questions will be dealt with using structural equation models. Care will be taken to avoid over-complex models, which suffer from overfitting, thereby enhancing the risk of chance findings (Harrell, 2015). If necessary, variable selection techniques will be used (Kuhn & Johnson, 2013). For hierarchical/dependent data such as those obtained from ESM we will use linear (continous outcomes) or generalised linear (dichotomous) mixed models and multilevel structural equation models (Preacher, Zyphur, & Zhang, 2010).

Discussion

This study captures the daily experience of a large sample of participants suffering from an affective disorder, an anxiety disorder, and controls. By capturing the fluctuations of symptoms, emotions, social interactions, well-being, and health-behaviours as well as neuropsychological and biological variables, we can contribute to the understanding of antecedents, consequences, and inherent processes that give rise to these fluctuations. These data also allow us to test how well participants can accurately report on their own experiences, thereby increasing understanding of how to better probe for such information in research and the clinic (Gloster, Meyer, Witthauer, Lieb, & Mata, in review). These data are thus relevant for basic clinical theory and clinical care alike.

By using ESM, we were able to capture experiences in participants' naturally chosen context thereby generating fundamentally different type of knowledge than questionnaires (i.e., belief and memory; Conner & Barrett, 2012). Participants' retrospective recalls were limited to three hours during ESM. As a result, any aggregation participants engage in was limited to a time frame for which they have much more accurate memories than with

questionnaires which require participants to aggregate experience across days, weeks, months, or even years. Within an individual, symptoms can appear extremely stable when assessed across large time frames, yet highly variable when examined within a single day or a week. This does not suggest that ESM is better, but rather necessary to supplant information from questionnaires/ belief.

Stability and fluctuation of emotions and symptoms are relatively neglected in the literature. A detailed understanding of naturally occurring stability and variability is one important key to understanding mental health (Kashdan & McKnight, 2011; Kuppens et al., 2010; Thompson et al., 2012; Watson, 2004) and provides a metric against which decisions are made for diagnostics (i.e., indication of frequency and length of symptoms) and treatment outcome. Treatment goals are partially determined by the inherent nature of stability. That is, the malleability of symptoms must be judged against the natural fluctuation of the targeted phenomena with an understanding of comparative phenomena. Treatment outcome measures assume a stability of symptoms whose level is influenced primarily via the intervention. The more strongly the assumption of symptom stability is violated, the less valid are pre-post questionnaires used to determine outcome. Nevertheless, a refined understanding of symptom fluctuation/ stability/ and "emotional inertia" across disorders is lacking (Kuppens et al., 2010; Watson, 2004), and the present study targets this need.

The combination of ESM, neuropsychological tests, and biological measures, as well as questionnaires in a large group of participants with an affective disorder, anxiety disorder, and controls results in a unique and rich data set. These data will help advance understanding of psychopathology, well-being, and clinical assessment by uniquely testing emerging theories in these areas such as psychological flexibility (Kashdan & Rottenberg, 2010), interactions of biological variables and environment (van Winkel et al., 2014), the importance of social interactions (Kawachi & Berkman, 2001; Thoits, 2011), and the interaction of these

areas. Importantly, testing across diagnostic conditions is consistent with current initiatives to identify processes relevant across boundaries of mental health (RDoC; Insel et al., 2010). Data from this study were collected under scientifically stringent conditions so that they are ideally situated to critically test the transfer into clinical care as called for in mobile health developments (Anthes, 2016; Johnson et al., 2009; Steinhubl, Muse, & Topol, 2015). Ultimately, these data may be used to develop a signalling system that will help identify patterns of maladaptive coping in (previously) depressed or socially anxious patients that can (Gloster et al., 2017) be used in clinical care.

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Conflict of Interest

The authors have no conflicts of interest to declare.

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Table 1

Example Research Questions to be Tested

Fluctuation of Symptoms and Emotions	
Example Research Questions	Variables
Does symptom fluctuation differ by diagnostic group?	Affect; Symptoms; Diagnosis
Do the antecedents and consequences of positive events differ by diagnostic group?	Affect; Events; Diagnosis
Does emotion regulation differ by diagnostic group?	Symptoms; Psychological Flexibility
Does the cortisol awakening response differ by diagnostic group and is this response altered by positive events?	Cortisol; Affect
Is sleep associated with subsequent symptom fluctuation?	Objective (physical movement measured via actigraphy) & subjective sleep; Symptoms
Are levels of BDNF associated with symptom fluctuation?	Serum BDNF; Symptoms
How do interpersonal interactions differ by diagnostic group?	Social Interaction Scale; Post-event processing; Diagnosis
Memory-Experience Gap	
Does the Memory-Experience Gap differ by diagnostic group?	ESM & retrospective recall; Diagnosis
Does the Memory-Experience Gap differ by content?	ESM & retrospective recall; Positive & Negative Affect
What psychological and biological factors are associated with the Memory-Experience Gap?	ESM & retrospective recall; Memory; Symptoms; BDNF

Table 2

Demographic Information (%) & Sample Characteristics (Mean & SD)

	MDD N = 118	SP N = 47	Controls N = 119
Age in years			
Mean	32.7	28.3	32.2
Median	29.0	26.0	28.0
SD	12.0	7.8	12.0
Sex			
Male	33.9	34.0	32.8
Female	66.1	66.0	67.2
Years of education			
8-10	21.1	9.3	12.0
11-13	51.4	67.4	53.0
14+	27.5	23.3	35.0
Living arrangement			
Alone	22.9	21.3	30.3
Family/partner	60.2	55.3	49.6
Other	16.9	23.4	20.2
Employment Status			
Employed	52.5	38.3	57.1
Unemployed	46.6	61.7	39.5
Number of diagnoses			
0	0.0	0.0	90.8
1	45.8	44.7	6.7
2	29.7	27.7	1.7
3+	24.6	27.7	0.8
In therapy			
No	41.5	53.2	85.7
Yes	58.5	46.8	14.3
BDI-II	27.0 (8.2)	17.0 (11.7)	3.0 (7.1)
SIAS	31.0 (14.4)	44.0 (12.5)	10.0 (7.3)
PSS	27.0 (5.3)	25.0 (6.0)	13.0 (7.2)
MHC-E	1.67 (0.96)	2.67 (1.12)	4.00 (0.92)
MHC-S	1.20 (0.94)	1.40 (0.96)	2.60 (1.14)
MHC- P	1.75 (0.98)	2.17 (1.19)	3.50 (0.96)

Note. MDD = Major Depressive Disorder; SP = Social Phobia; BDI-II = Beck Depression Inventory; SIAS = Social Interaction Anxiety Scale; PSS = Perceived Stress Scale; MHC- E,S,P = Mental Health Continuum – Emotional, Social, & Psychological Subscales

Table 3

Assessment Strategy

Domain	Instrument	Description	T1	T2	T3
Psychological Variables					
Diagnosis	SKID-I	Structured clinical interview	x		
Executive Functioning	Trails A & B	Attention & task switching	x		
	Backward digit span	Working memory	x		
	Vocabulary	Verbal comprehension	x		
Symptoms	BDI-II	Depression	x	x	x
	SIAS	Social anxiety	x	x	x
Affect	PANAS	Positive/negative affect	x	x	x
	PSS	Perceived stress		x	x
Health	HCU	Health care utilization	x		
	IPAQ	Physical activity		x	x
	ISI	Sleep problems		x	x
Psychological Flexibility	AAQ-II	Psychological flexibility	x	x	x
	F-ACT	ACT-processes	x	x	x
Emotion	ERQ	Emotion regulation	x		
	DERS	Emotion dysregulation	x		
Well-being	MHC-SF	Mental health continuum	x		x
	VLQ	Personal values	x		x
	MLQ	Meaning in life	x	x	x
Biological Variables					
Sleep	Actimeter	Movement during sleep		x	x
Cortisol Awakening Response	Saliva sample	Cortisol in saliva upon awakening		x	x
BDNF	Blood sample	Serum Level of BDNF	x		

Physical Activity Questionnaire, ISI Insomnia Severity Index, AAQ-II Acceptance and Action Questionnaire-II, F-ACT Fragebogen zu Acceptance and Commitment Therapy, ERQ Emotion Regulation Questionnaire, DERS Difficulties in Emotion Regulation Scale, MHC-SF Mental Health Continuum-Short Form, VLQ Valued Living Questionnaire, MLQ Meaning in Life Questionnaire, BDNF Brain-derived neurotrophic factor, T1 Day 1, T2 Day 7, T3 Day 15

Figure 1

Study design and procedure