Emotion network density is a potential clinical marker for anxiety and depression: Comparison of ecological momentary assessment and daily diary

Ki Eun Shin*1, Michelle G. Newman2 and Nicholas C. Jacobson3

1Teachers College, Columbia University, New York, New York, USA
2The Pennsylvania State University, State College, Pennsylvania, USA
3Dartmouth College, Hanover, New Hampshire, USA

Objectives. Using two intensive longitudinal data sets with different timescales (90 minutes, daily), we examined emotion network density, a metric of emotional inflexibility, as a predictor of clinical-level anxiety and depression.

Design. Mobile-based intensive longitudinal assessments.

Methods. 119 participants (61 anxious and depressed, 58 healthy controls) completed ecological momentary assessment (EMA) to rate a variety of negative (NE) and positive (PE) emotions 9 times per day for 8 days using a mobile phone application. 169 participants (97 anxious and depressed and 72 healthy controls) completed an online daily diary on their NE and PE for 50 days. Multilevel vector autoregressive models were run to compute NE and PE network densities in each data set.

Results. In the EMA data set, both NE and PE network densities significantly predicted participants’ diagnostic status above and beyond demographics and the mean and standard deviation of NE and PE. Greater NE network density and lower PE network density were associated with anxiety and depression diagnoses. In the daily diary data set, NE and PE network densities did not significantly predict the diagnostic status.

Conclusions. Greater inflexibility of NE and lower inflexibility of PE, indexed by emotion network density, are potential clinical markers of anxiety and depressive disorders when assessed at intra-daily levels as opposed to daily levels. Considering emotion network density, as well as the mean level and variability of emotions in daily life, may contribute to diagnostic prediction of anxiety and depressive disorders.

Practitioner points

- Emotion network density, or the degree to which prior emotions predict and influence current emotions, indicates an inflexible or change-resistant emotion system.
- Emotional inflexibility or change resistance over a few hours, but not daily, may characterize anxiety and depressive disorders.
Inflexible negative emotion systems are associated with anxiety and depressive disorders, whereas inflexible positive emotion systems may indicate psychological health. Considering emotional inflexibility within days may provide additional information beyond demographics and mean level and variability of emotions in daily life for detecting anxiety and depressive disorders.

Emotions are not static, but constantly fluctuate over time, following the ebb and flow of daily life. Emotions are adaptive insofar as they signal and motivate individuals to respond to shifting contexts and demands. Emotional flexibility, or an ability to modulate one’s emotional responses in line with changing circumstances, has been associated with psychological health (e.g., resilience, low distress; Bonanno, Papa, Lalande, Westphal, & Coifman, 2004). Conversely, emotional inflexibility, or the resistance of emotional states to change, may characterize psychological maladjustment, such as anxiety and depressive disorders.

Based on their diagnostic definitions, anxiety and depressive disorders are characterized by excessive and persistent negative emotions. More importantly, both disorders have been associated with decreased reactivity to emotional stimuli. In both laboratory and daily life, individuals with generalized anxiety disorder sustained heightened levels of negative emotions (NE) following worrying and showed an attenuated response to negative emotional stimuli (Llera & Newman, 2014; Newman et al., 2019). Major depressive disorder also has been associated with a change-resistant emotional tendency, such as a blunted response to negative and positive emotion inductions (meta-analysis; Bylsma, Morris, & Rottenberg, 2008). In addition, both depressive and anxiety disorders were associated with increased NE inertia, operationalized as the autoregression of NE, which indicated the degree to which NE carried over from one moment to another (meta-analysis; Houben, Van Den Noortgate, & Kuppens, 2015). Overall, these findings suggest constricted emotional tendencies in anxiety and depressive disorders, wherein NE tends to persist, leaving less room for flexible responding to external and internal emotional contexts.

Findings for positive emotions (PE) have been more mixed. On the one hand, anxiety and depressive disorders were associated with reduced reactivity to positive events in daily life (e.g., Carl, Fairholme, Gallagher, Thompson-Hollands, & Barlow, 2014). PE inertia, or the degree to which PE predicted itself over time, was also associated with various mental disorders, although to a lesser extent than NE inertia (Houben, Van Den Noortgate, & Kuppens, 2015). On the other hand, change resistance of PE has been shown to be potentially adaptive. Less decrease in PE in response to daily stressors predicted a lower likelihood of depressive and anxiety disorders over seven years (Rackoff & Newman, 2020). Compared to depressed adolescents, healthy controls also showed less change in PE when transitioning from a positive to negative task (Fussner, Luebbe, & Bell, 2015).

One of the limitations with the prior studies is that they examined emotional inflexibility only at the level of an individual emotion (e.g., sadness) or an emotion composite (e.g., NE). Such approach neglects inter-relations between emotions. Individual emotions are not independent, but covarying, with stronger associations between those of the same valence and arousal (e.g., Posner, Russell, & Peterson, 2005). Emotions also elicit each other over time, with secondary emotions emerging as a response to the initial or primary emotions (e.g., Bailen, Wu, & Thompson, 2019). Furthermore, both anxiety and depressive disorders were associated with emotional non-acceptance, or negative beliefs and emotional responses to one’s own NE (e.g., Flynn,
Hollenstein, & Mackey, 2010; Mennin, Holaway, Fresco, Moore, & Heimberg, 2007). This suggests that in anxious and depressed individuals, experiencing NE can activate secondary NE. Such aversive response to NE may ironically prolong and amplify the distressing emotions. Individuals with generalized anxiety disorder also reported greater fear in response to their emotions of anxiety, sadness, and anger than non-anxious controls (Mennin, Heimberg, Turk, & Fresco, 2005). These findings indicate the need to consider interactions between emotions when assessing emotional inflexibility in anxiety and depressive disorders.

Emotion network density is a novel metric of emotional inflexibility that uniquely reflects temporal dependency among individual emotions. It is based on the network model that conceptualizes discrete emotions as inter-connected and mutually influencing each other over time (Bringmann et al., 2013; Trampe, Quoidbach, & Taquet, 2015). The network view complements the traditional view that discrete emotions (e.g., sad, happy) give rise to NE and PE as higher-order, latent constructs (e.g., Watson, Clark, & Tellegen, 1988). In the traditional view, PE and NE are conceptualized as aggregates or composites across individual emotions, and the bi-factor structure is expected to generalize across individuals. In contrast, the network view approaches PE and NE as dynamic constructs, defined by the pattern of temporal inter-relations among individual emotions. This pattern of emotion dynamics can also vary across individuals. For instance, emotion network density is an individual-specific measure of the overall strength of within-person, temporal inter-relations between emotions. Higher emotion network density indicates a more change-resistant emotion system, where prior emotions, rather than ongoing contextual cues or regulation efforts, influence and determine current emotions.

Emerging evidence from within-person, temporal emotion network studies indicates elevated emotion network density in anxiety and depressive disorders. Depression diagnoses and symptoms were associated with higher overall (including both NE and PE) network density in adults (Pe et al., 2015; Wigman et al., 2015) and adolescents (Lydon-Staley, Xia, Mak, & Fosco, 2019). When NE and PE were examined separately, this finding only held for NE, but not PE network density (Pe et al., 2015). Interestingly, higher overall and NE network densities were also associated with neuroticism (Bringmann et al., 2016), a common, higher-order factor across anxiety and depressive disorders (e.g., Muris, Roelofs, Rassin, Franken, & Mayer, 2005). This suggests that high NE network density might be a clinical marker of both anxiety and depressive disorders. However, prior research has focused on depressed samples, and it remains unknown whether the results would extend to a transdiagnostic sample of anxiety and depressive disorders.

In addition, despite the promising nature of the prior findings, there remain several gaps to fill. First, many prior studies focused on overall emotion network density, which aggregated and confounded the strength of inter-relations among NE (i.e., NE network density), among PE (i.e., PE network density), and between NE and PE. Due to such confounding, implications of the findings are less clear and informative than when examining NE and PE separately. Second, distinguishing between NE and PE network densities is important for potential specificity in their effects. For example, associations between dynamic emotional processes (e.g., inertia) and psychopathology were greater for NE than PE (Houben et al., 2015). In addition, in some studies (e.g., Scott, Victor, et al., 2020), the direction of association with psychopathology changed depending on emotion valence. Third, among the few studies examining PE network density, findings have been inconclusive. Whereas one study found a non-significant association with depression (Pe et al., 2015), in another study, there was a significant positive association with neuroticism in only one of the two data sets examined (Bringmann et al., 2016). Therefore, further
investigation is necessary to clarify whether and how PE network density is associated with depressive and anxiety disorders.

Another important extension of prior studies is to consider the time frame. Most prior emotion network studies (Pe et al., 2015; Wigman et al., 2015) used ecological momentary assessment (EMA), assessing emotions multiple times within a day at fixed or quasi-random intervals (e.g., 8 times per day for 7 days). Whereas EMA studies examined the relations between emotions over a few hours within a day, one emotion network study (Lydon-Staley et al., 2019) assessed emotions once each day (i.e., daily diary) to examine the day-to-day relations between emotions. However, because the latter study focused on overall emotion network density, which confounds NE and PE network densities, it remains unclear how daily NE and PE densities each relate to anxiety and depressive disorders. In addition, temporal associations between emotions can vary depending on the timescale of measurement. For instance, NE and PE were more strongly negatively correlated within individuals over a shorter (vs. longer) time frame (Watson, 1988). Moreover, the degree of emotional fluctuation between consecutive time points was high when examined within days, but low when examined between days (Jahng, Wood, & Trull, 2008). Comparing emotion network density using different timescales may help to clarify at which temporal scale emotional inflexibility manifests. Given rapid fluctuations of emotional states (e.g., over a few hours; Trull et al., 2008) and potentially brief timescales for the unfolding of inter-relations among emotions (Bailen et al., 2019), repeated measurements within a day might be more sensitive to capturing emotional inflexibility, as operationalized by emotion network density, than daily measurements.

In addition, complex emotion metrics such as emotion network density have been critiqued as being potentially conflated with mean and variance in emotions (Dejonckheere et al., 2019). Therefore, it is necessary to test whether emotion network density incrementally adds to the prediction of anxiety and depressive disorders beyond the mean and variance in emotions. Only if so, would it be valid to consider emotion network density as an independent and non-redundant clinical marker. Despite these important implications, no prior study has tested the incremental validity of emotion network density in predicting psychopathology.

To fill the gaps in the literature, the current study used two data sets with varying time frames (i.e., 90-min interval EMA vs. daily diary) to examine emotion network density. We hypothesized that NE network density would show incremental validity in predicting diagnostic status beyond demographic variables and covariates (means and standard deviations of NE and PE). Our hypothesis on PE network density was exploratory given mixed prior evidence. We also hypothesized that associations between emotion network density and diagnostic status would be stronger in the EMA data set than in the daily diary data set.

**Methods**

**Data set 1: Ecological momentary assessment**

**Participants**

119 participants (61 with generalized anxiety disorder (GAD) or major depressive disorder (MDD) and 58 healthy controls) were recruited from a university subject pool ($n = 85$) and the general community via flyers and online advertisements ($n = 34$). Participants were screened using the Beck Depression Inventory-II and the Generalized Anxiety Disorder Questionnaire-IV. Those who met the clinical cut-off on at least one
measure or those who scored one standard deviation below the mean on both measures were invited to complete the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998). Highly trained bachelor-level assessors administered the MINI under the supervision of a doctoral-level therapist and a licensed clinical psychologist. The inclusion criterion for the clinical group was current, primary diagnoses of MDD or GAD. Exclusion criteria were current psychosis, mania, and alcohol or substance use disorders. The inclusion criterion for the control group was no current or past psychopathology. Interviews were audio-recorded, and 57% were re-rated by a blinded assessor. Inter-rater reliability was good (percent agreement: 94%-100%, Cohen’s kappa: .66–1). 129 (64 clinical and 65 controls) were recruited. One clinical and three control participants withdrew before starting EMA. Two clinical and four control participants were removed due to poor compliance ($n = 5$; 10 or fewer responses, $n = 1$; zero variability in response, i.e., rating all items the same every time). The total number of dropouts and exclusions did not differ between clinical and control groups, $\chi^2(1, N = 129) = 1.67, p = .200$.

The final sample (81% female, 19% male) had a mean age of 19.70 ($SD = 3.65$, range $= 18–50$). The sample included White (75%), Asian (12%), Black (5%), Latino/a (5%), and multiracial (3%). Clinical and control participants did not differ on age, $F(1,117) = 2.38, p = .126$, race, $\chi^2(4, N = 119) = 3.50, p = .478$, or recruitment source, $\chi^2(1, N = 119) = .34, p = .562$. However, they differed on gender, with more females in the clinical group than controls, $\chi^2(1, N = 119) = 8.80, p = .003$. The clinical group included 28 participants with GAD, 8 with MDD, and 25 with both GAD and MDD. Forty-one clinical participants (67%) had at least one comorbid disorder: social anxiety disorder ($n = 19$), panic disorder ($n = 15$), obsessive-compulsive disorder ($n = 13$), post-traumatic stress disorder ($n = 10$), agoraphobia ($n = 7$), binge eating disorder ($n = 3$), and bulimia nervosa ($n = 2$). Forty-four clinical participants (72%) were either in treatment ($n = 16$) or had an intent to seek treatment for anxiety or depression ($n = 28$).

**Measures**

*Beck depression inventory-II (BDI-II).* The BDI-II is a 21-item instrument assessing presence and severity of MDD symptoms (Beck, Steer, & Brown, 1996). Items are rated on a scale from 0 to 3 severity. The BDI-II has high internal consistency ($\alpha = .93$ in the current sample), retest reliability, and convergent and divergent validity (Beck et al., 1996). We used a cut-off score of 19, which indicated moderate depression (Beck et al., 1996).

*Generalized anxiety disorder questionnaire-IV (GAD-Q-IV).* The GAD-Q-IV is a 14-item self-report diagnostic measure of GAD based on DSM-IV/5 criteria (Newman et al., 2002). Items assess excessive and uncontrollable worry, number of worry topics, and somatic symptoms. The measure has high internal consistency ($\alpha = .86$ in the current sample), retest reliability, and convergent and divergent validity. The current study used a cut-off of 5.7, which optimized sensitivity (83%) and specificity (89%) and had a kappa agreement of .67 with a diagnostic interview (Newman et al., 2002).

*Mini international neuropsychiatric interview (MINI).* The MINI is a 15- to 30-min structured interview (Sheehan et al., 1998). The most recent version (7.0.2.) assesses 17 mental disorders based on the DSM-5 (American Psychiatric Association, 2013) and the
International Classification of Diseases-10th Edition (ICD-10). It was validated against the Composite International Diagnostic Interview (Robins et al., 1988) and the Structured Clinical Interview for DSM (Spitzer, Williams, Gibbon, & First, 1989). Sensitivity was $\geq .70$ for all diagnoses but dysthymia (.62) and obsessive-compulsive disorder (.67). Specificity was $\geq .85$ for all diagnoses (Sheehan et al., 1998).

**Ecological momentary assessment (EMA).** Participants were prompted to complete a brief self-report survey 9 times per day, at 90-minute intervals, for 8 days. They rated on a 9-point Likert scale from 1 (not at all) to 9 (very much), the degree to which they experienced NE (angry, tired, irritable, anxious, and depressed) and PE (relaxed, content, excited, and enthusiastic) since the last prompt. The survey also included items on MDD and GAD symptoms (e.g., worry, difficulty concentrating). The time frame (‘since the last prompt’) was selected to capture emotional inflexibility more completely relative to sampling participants’ emotions only at the moment they completed an EMA survey. For instance, if emotional inflexibility were to occur across a few minutes, sampling emotions only at 90-minute intervals could yield an incomplete and misleading representation of participants’ emotional experiences. The nine emotion items were selected to capture four quadrants of the emotion circumplex (Russell, 1980), encompassing different combinations of valence (positive, negative) and arousal (high, low) (e.g., negative valence and low arousal: depressed, tired). EMA surveys were delivered using PACO (Google, 2017), a mobile application compatible with Android and iOS platforms.

**Procedure**

At baseline, participants completed self-report questionnaires online. Next, they installed the PACO app on their personal mobile phone and received training on how to respond to the EMA surveys. Starting the next day, participants were prompted nine times per day, at 90-minute intervals, between 10 am and 10 pm for eight days. If participants missed a prompt, they received a reminder after 10 minutes. The survey link was closed 30 minutes from the prompt. Experimenters provided motivational feedback via text on the 2nd, 4th, and 6th day. After the 8th day, participants received debriefing and compensation. Subject pool participants received course credits, and community participants received monetary compensation.

**Data set 2: Daily diary**

**Participants**

175 participants (79% female, 19% male, and 2% transgender) were recruited from undergraduate psychology courses in a large public university. Mean age was 20.04 ($SD = 1.52$, range = 18–31). The sample included 64% White, 16% Asian, 9% Black, 6% Latino/a, and 5% others (e.g., Pacific Islander). 169 (97%) completed the PROMIS Anxiety Scale-Short Form and the PROMIS Depression Scale-Short Form (Pilkonis et al., 2011) at baseline. 72 participants scored lower than the clinical cut-off for moderate-to-severe anxiety and depression (T-score of 60 or above; Pilkonis et al., 2011). 97 met the clinical cut-off on at least one measure; 44 met the cut-off for anxiety, 4 met the cut-off for depression, and 47 met the cut-off for both anxiety and depression. The clinical and
control groups did not differ on age, $F(1,166) = .41, p = .521$, gender, $\chi^2(2,168) = 3.95, p = .139$, or race, $\chi^2(4,168) = .81, p = .938$.\footnote{We note that a preprint on a separate and unrelated study based on the daily diary data set is available online (https://psyarxiv.com/gb5up/).

**Measures**

**PROMIS Anxiety Scale – Short Form.** This measure includes 7 items rated on a 1 (never) to 5 (always) Likert scale measuring anxiety pathology in the past week (Pilkonis et al., 2011). It showed high internal consistency ($\alpha = .92$ in the current sample) and convergent and discriminant validity (Pilkonis et al., 2011). Raw scores were converted to T-scores based on the U.S. population norm (Liu et al., 2010). We used a T-score of 60 as the clinical cut-off, which converged with the clinical cut-off on the GAD-7 (Schalet, Cook, Choi, & Cella, 2014). This score showed 89% sensitivity and 82% specificity in detecting GAD (Spitzer, Kroenke, Williams, & Löwe, 2006).

**PROMIS Depression Scale – Short Form.** This measure includes 8 items rated on a 1 (never) to 5 (always) Likert scale measuring depression pathology in the past week. It showed high internal consistency ($\alpha = .95$ in the current sample) and convergent and divergent validity (Pilkonis et al., 2011). Raw scores were converted to T-scores, with $T = 60$ as the clinical cut-off. The score converged with the cut-off on the Patient Health Questionnaire-9 (Choi, Schalet, Cook, & Cella, 2014), which showed 88% sensitivity and specificity in detecting MDD (Kroenke, Spitzer, & Williams, 2001).

**Positive and negative affect schedule (PANAS).** The PANAS is a 20-item scale assessing positive (10 items) and negative feelings (10 items). Participants rated their experience of each emotion daily on a 0 (not at all) to 100 (extremely) scale, using a continuous slider (Watson et al., 1988). When administered in a daily diary format, the PANAS showed high internal consistency at both between-person (NA: .95–.99, PA: .98-.99) and within-person levels (NA: .73–.88, PA: .81–.94) (Scott, Sliwinski, et al., 2020; Zuroff, Sadikaj, Kelly, & Leybman, 2016).

**Procedure**

Participants completed the PROMIS symptom measures at baseline. Then, they were asked to complete an online version of the PANAS at the end of each day for 50 days. After 50 days, participants completed the PROMIS symptom measures again. Then, participants were debriefed and compensated with course credits.

**Statistical analyses**

Using the R package mlVAR version 0.4.4 (Epskamp, Waldorp, Mottus, & Borsboom, 2018), NE and PE temporal networks were estimated based on a multilevel vector autoregressive (VAR) model (Bringmann et al., 2013). We estimated the extent to which an emotion variable was predicted by all emotion variables of the same valence (including
itself) at a previous time point. In the EMA data set, five univariate multilevel analyses were run to estimate the NE network, where each of the five NE variables was regressed on its lagged value (autoregressive effect) and the lagged values of all other NE variables (cross-lagged effects) at the previous time point. The time lag was the interval between consecutive EMA prompts (90 minutes). Overnight lags were excluded by creating lagged predictor variables within days and individuals such that the first measurement of a day was not regressed on the last measurement of the previous day. Taking depressed feeling as an example, the following equation illustrates the model:

\[
\text{Depressed}_{i,t} = \beta_{0i} + \beta_{1i}\text{Depressed}_{i,t-1} + \beta_{2i}\text{Angry}_{i,t-1} + \beta_{3i}\text{Tired}_{i,t-1} + \beta_{4i}\text{Irritable}_{i,t-1} + \beta_{5i}\text{Anxious}_{i,t-1} + \epsilon_{i,t}
\]

The intercept \((\beta_{0i})\) represented the value of depressed feeling at time \(t\) when all NE variables at \(t-1\) were equal to zero (i.e., equal to within-person means because all variables were within-person standardized). The slope coefficients represented the unique influence of each NE variable on depressed feeling over time. \(\beta_{1i}\) indicated an autoregressive effect, and \(\beta_{2i}\) through \(\beta_{5i}\) indicated cross-lagged effects. The cross-lagged slopes represented Granger causality, where one variable predicted another variable over time, above and beyond the autoregressive effect (Granger, 1969). The intercept and slope coefficients were allowed to vary across individuals to estimate both average (fixed effects) and person-specific networks (random effects). Random intercepts and slopes were also allowed to be correlated. The PE network in the EMA data set and the NE and PE networks in the daily diary data set were estimated in a similar manner.

Before running multilevel VAR models, data were preprocessed by applying linear detrending and within-person standardization (see Appendix S1). Due to low within-person variability in emotion variables, NE networks could not be estimated for four control participants in the EMA data set and eight control and two clinical participants in the daily diary data set. PE network could not be estimated for one clinical participant in the daily diary data set for the same reason. Emotion networks were visualized using the R package qgraph version 1.6.5 (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012). Nodes represented emotion variables, and edges represented temporal associations between emotions. The thickness of the edges reflected the magnitude of associations. The Fruchterman–Reingold algorithm (Fruchterman & Reingold, 1991) was used to place emotions with stronger temporal associations closer to each other in the network plots.

Currently, there is no clear guideline on power for a multilevel VAR model. However, a simulation study showed that multilevel VAR estimation performed well (e.g., high correlations with true networks \((r's > .8)\), high sensitivity and specificity \((> .9)\), and low bias, or the mean absolute deviation of true to estimated parameters \(< .1)\)) in estimating the fixed and random effects of an 8-node temporal network including both autoregressive and cross-lagged estimates in 100 participants with 50 measurements (Epskamp et al., 2018). This simulation also accounted for variability across individual networks and correlated random effects. The simulated model is similar to the emotion networks estimated in the current study in terms of the number of nodes (4–10 nodes) and model complexity (estimating both fixed and random effects, allowing random effects to be correlated, including both autoregressive and cross-lagged effects). Thus, current samples (EMA: 119 participants, 57 measurements on average; daily diary: 169 participants, 45 measurements on average) appear to have sufficient power for the conducted analyses.
Diagnostic status prediction based on network density

Binary logistic regressions were run to examine whether NE and PE network densities added to the prediction of a diagnostic status (i.e., clinical vs. control) beyond demographic variables (gender, race, and age) and covariates (mean and standard deviation of NE and PE). Emotion network density was calculated for each participant by taking the average of the absolute values of all individual slopes from the multilevel VAR analyses. NE network density was the mean of the absolute slope values from the multilevel analyses run to estimate an NE network. PE network density was the mean of the absolute slope values from the multilevel analyses run to estimate a PE network. NE network density represented how strongly individual NE at a previous time point influenced current NE over time (e.g., 90 minutes in EMA, a day in daily diary). PE network density represented how strongly PE at a previous time point influenced current PE over time. Variables were entered in a hierarchical fashion, with demographics first, covariates second, and NE and PE network densities last. Emotion density, mean, and standard deviation were standardized. Multicollinearity was not present (tolerance > .3, variance inflation factors < 3).

Results

Preliminary analyses

In the EMA data set, mean compliance rate was 80% (57 responses, SD = 13). Number of responses did not differ by diagnostic group, $F(1,117) = .06, p = .800, d = .06$, or recruitment source, $F(1,117) = .19, p = .667, d = .09$. In the daily diary data set, mean compliance rate was 89.28% (45 responses, SD = 3.79). Compliance did not differ between clinical and control groups, $F(1,167) = .44, p = .506, d = .11$. Within-person means and standard deviations of emotion variables were also compared by diagnostic group. Results warranted within-person standardization to rule out that group differences in emotion network density resulted from the mean and variance differences (see Appendix S1).

Diagnostic status prediction based on network density

Figures 1 and 2 present the estimated NE and PE networks by diagnostic group in the EMA and daily diary data sets. Table 1 presents means and standard deviations of NE and PE network density by diagnostic group in each data set. Results of hierarchical multiple logistic regression analyses are summarized in Table 2. In the EMA data set, the Step 1 model including demographic variables (gender, race, and age) significantly predicted participants’ diagnostic status, $\chi^2(6, N = 115) = 18.82, p = .004$. Overall predictive accuracy was 69.6% (sensitivity: 86.9%, specificity: 50.0%). Gender was the only significant predictor. The odds of belonging to a clinical group for females were 5.67 times as large as that of males, $p = .003$. In Step 2, means and standard deviations of NE and PE were added to the model and increased predictive validity from Step 1, $\chi^2(4, N = 115) = 46.41, p < .001$. Overall predictive accuracy increased to 81.7% (sensitivity: 83.6%, specificity: 79.6%). Gender was no longer significant, $p = .387$. Mean and standard deviation of NE were significant predictors. One standard deviation increase in mean and

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2 We also inspected potential time effects on missingness of observations at within-person level (see online supplement). We did not find evidence for a time effect that would have systematically biased current findings.
The standard deviation of NE increased the odds of a participant being in the clinical group relative to the control group by 3.68 times, \( p < .001 \), and by 2.71 times, \( p = .016 \), respectively. In Step 3, adding NE and PE network densities further improved predictive validity from Step 2, \( \chi^2(2, N = 115) = 12.83, p = .002 \). Overall predictive accuracy reached 87% (sensitivity: 88.5%, specificity: 85.2%). Both NE and PE network densities significantly predicted diagnostic status. One standard deviation increase in NE network density predicted a 2.80 times increase in the odds of a participant being in the clinical group, \( p = .009 \). On the contrary, one standard deviation decrease in PE network density predicted 2.52 times increase in the odds of a participant belonging to the clinical group, \( p = .017 \). Mean NE continued to be a significant predictor, with one standard deviation

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**Figure 1.** Emotion networks for the clinical group (left column) and controls (right column) in Study 1 (ecological momentary assessment). First row presents negative emotion (NE) network, and second row presents positive emotion (PE) network. Only the significant connections between emotions (e.g., one emotion at time \( t-1 \) and another emotion at time \( t \)) are visualized with the corresponding estimated coefficients. Solid arrows represent positive associations, and dotted arrows represent negative associations. Thicker arrows represent stronger associations.
increase in mean NE predicting an increase in the odds of a participant belonging to the clinical group by 4.73 times, \( p < .001 \). The other variables did not predict diagnostic status.

In the daily diary data set, the Step 1 model including demographic variables (gender, race, and age) did not significantly predict diagnostic status, \( \chi^2(7, N = 157) = 4.74, p = .692 \). Overall predictive accuracy was 60.5% (sensitivity: 87.2%, specificity: 20.6%). None of the demographic variables were significant predictors, \( ps > .05 \). In Step 2, adding means and standard deviations of NE and PE to the model significantly increased predictive validity, \( \chi^2(4, N = 157) = 36.06, p < .001 \). Overall predictive accuracy
increased to 72% (sensitivity: 81.9%, specificity: 57.1%). Mean NE and mean PE were the only significant predictors. One standard deviation increase in mean NE increased the odds of a participant being in a clinical group relative to a control group by 3.01 times, \( p = .001 \). One standard deviation decrease in mean PE increased the odds by 1.56 times, \( p = .044 \). In Step 3, the addition of NE and PE network densities did not improve predictive validity from Step 2, \( \chi^2(2, N = 157) = 1.18, p = .556 \). Overall predictive accuracy was 70.7% (sensitivity: 83.0%, specificity: 52.4%). Mean NE and PE continued to be the only significant predictors, with similar odds ratios as in Step 2.3

**Discussion**

The present study examined emotion network density as a potential clinical marker for anxiety and depression. We tested whether emotion density values predicted diagnostic status above and beyond demographics and the mean and standard deviation of emotion. Only in the EMA data set, NE and PE network densities significantly predicted diagnostic status, demonstrating incremental validity over demographics and the mean and standard deviation of emotion. Higher NE density and lower PE density predicted a greater likelihood of a participant belonging to the clinical group than controls. Associations between either type of network density and diagnostic status were not evidenced in the daily diary data set.

The current study extended prior studies by examining emotion network density at two different timescales (intra-daily, daily). We found that anxiety and depression were associated with emotion network density at intra-daily, but not daily levels. Based on supplementary analyses comparing the EMA and daily diary samples, we did not find significant differences on demographics, baseline depression severity, and the proportion of individuals with ‘pure’ anxiety (i.e., anxiety without depression), ‘pure’ depression, comorbid anxiety and depression, and healthy controls, ruling them out as explanations for the current findings (see Appendix S1). There are theoretical and methodological reasons to suspect that the difference in timescale of assessment drove the results. First, emotional inflexibility, captured by emotion network density, might be more fast-acting than a daily process. Autoregressive and cross-lagged effects between emotions, which constitute an emotion network, were significant at time frames ranging from a few

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**Table 1.** Means and standard deviations of NE and PE network density by diagnostic group

<table>
<thead>
<tr>
<th>Emotion network</th>
<th>Study 1: EMA</th>
<th>Study 2: Daily diary</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Clinical</td>
<td>Control</td>
</tr>
<tr>
<td></td>
<td>((n = 61))</td>
<td>((n = 58))</td>
</tr>
<tr>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
</tr>
<tr>
<td>Negative emotion</td>
<td>0.10 (0.02)</td>
<td>0.08 (0.03)</td>
</tr>
<tr>
<td>Positive emotion</td>
<td>0.11 (0.03)</td>
<td>0.12 (0.03)</td>
</tr>
</tbody>
</table>

*Note.* EMA = ecological momentary assessment.

3 As a sensitivity analysis, we used multiple imputation to account for missing data and reran logistic regression analyses (see online supplement). The pattern of results stayed consistent with primary findings. The only deviation was that in the daily diary data set, the gender effect became significant in Step 1 of the logistic regression model. Females were more likely to belong to the clinical group than males, \( \text{aOR} = 2.35, 95\% \text{ CI } [1.02, 5.40], p = .045 \).
### Table 2. Logistic regression analyses results on prediction of diagnostic status

<table>
<thead>
<tr>
<th></th>
<th>Study 1: EMA</th>
<th>Study 2: Daily diary</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Step 1</td>
<td>Step 2</td>
</tr>
<tr>
<td>Gendera</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>5.67</td>
<td>1.99</td>
</tr>
<tr>
<td></td>
<td>.003*</td>
<td>.387</td>
</tr>
<tr>
<td>Others</td>
<td>1.77–18.2</td>
<td>.42–9.43</td>
</tr>
<tr>
<td>Raceb</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>.78</td>
<td>.95</td>
</tr>
<tr>
<td></td>
<td>.14–4.38</td>
<td>.09–10.27</td>
</tr>
<tr>
<td>Asian</td>
<td>3.73</td>
<td>4.65</td>
</tr>
<tr>
<td></td>
<td>.073</td>
<td>.098</td>
</tr>
<tr>
<td>Hispanic</td>
<td>.88–15.72</td>
<td>.75–28.64</td>
</tr>
<tr>
<td></td>
<td>.06–2.21</td>
<td>.03–2.71</td>
</tr>
<tr>
<td>Others</td>
<td>.46</td>
<td>.23</td>
</tr>
<tr>
<td></td>
<td>.560</td>
<td>.357</td>
</tr>
<tr>
<td>Age</td>
<td>.71–1.02</td>
<td>.72–1.08</td>
</tr>
<tr>
<td>Mean NE</td>
<td>.78</td>
<td>.95</td>
</tr>
<tr>
<td></td>
<td>.387</td>
<td>.241</td>
</tr>
<tr>
<td>Mean PEc</td>
<td>.91–5.35</td>
<td>.52–4.19</td>
</tr>
<tr>
<td>NE SD</td>
<td>.78–4.95</td>
<td>1.96</td>
</tr>
<tr>
<td></td>
<td>.82–2.76</td>
<td>.78–2.84</td>
</tr>
<tr>
<td>PE SD</td>
<td>.92</td>
<td>.329</td>
</tr>
<tr>
<td></td>
<td>.71</td>
<td>.103</td>
</tr>
</tbody>
</table>

*Continued*
Table 2. (Continued)

<table>
<thead>
<tr>
<th></th>
<th>Study 1: EMA</th>
<th>Study 2: Daily diary</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Step 1</td>
<td>Step 2</td>
</tr>
<tr>
<td></td>
<td>aOR</td>
<td>95% CI</td>
</tr>
<tr>
<td>NE density</td>
<td>2.80</td>
<td>1.30–6.04</td>
</tr>
<tr>
<td></td>
<td></td>
<td>.009*</td>
</tr>
<tr>
<td>PE densityc</td>
<td>2.52</td>
<td>1.18–5.38</td>
</tr>
</tbody>
</table>

Note. aOR = adjusted odds ratio; NE = negative emotion; PE = positive emotion; SD = standard deviation.

*Male as a reference group.; White as a reference group.; Mean PE and PE density were reverse-coded by multiplying them by −1 to aid interpretation.; \( p \leq .05. \)
seconds to 10 minutes (Bailen et al., 2019; Koval, Sütterlin, & Kuppens, 2016). In addition, studies suggest that as the timescale of assessment increases so does the likelihood of diluting the within-person association between NE and PE (Watson, 1988), perhaps due to greater confounding variables (e.g., more intermediating emotional stimuli). Relative to EMA, daily diary is also more susceptible to retrospective bias, with participant report influenced by the mood around the time of retrospection (Parkinson, Briner, Reynolds, & Totterdell, 1995). The current findings indicate the importance of considering timescale in examining dynamic emotional processes and warrant further research to clarify the optimal timescale for examining emotional inflexibility.

We replicated prior findings of elevated intra-daily NE network density in MDD (e.g., Pe et al., 2015) in a transdiagnostic sample of MDD and GAD. To inspect whether this finding was driven by individuals with MDD, we conducted supplementary analyses comparing those with ‘pure’ GAD and healthy controls. The results stayed consistent (see Appendix S1). The current finding is in line with prior evidence that NE network density was positively associated with neuroticism, a common factor that cuts across anxiety and depressive disorders (Bringmann et al., 2016). Thus, NE inflexibility might characterize a broad dimension of internalizing disorders rather than specific disorders. In addition, in the EMA data set, MDD and GAD diagnoses were associated with a denser NE network, as well as higher means and standard deviations of NE. Elevations in both emotion inflexibility and variability may appear contradictory but are reconcilable. NE standard deviation summarizes the dispersion of NE levels across the assessment period, whereas NE network density captures moment-to-moment NE dynamics. The two metrics provide complimentary information such that standard deviation captures the overall magnitude of change in NE but does not account for temporal dependency, whereas network density captures temporal dependency of NE. High NE standard deviation can arise for different reasons, such as a large and gradual shift in NE, or many frequent and smaller shifts in NE across time. In the case of high NE network density and high NE standard deviation, the former case is more likely, with moment-to-moment inflexibility of NE within days, but a large, gradual shift in NE across days. A similar emotional profile was found in MDD (Pe et al., 2015). In future research, examining whether this profile generalizes to other disorders beyond MDD and GAD would help to elucidate shared and disorder-specific emotional processes.

In the EMA data set, lower PE network density predicted a greater likelihood of a participant belonging to the clinical group than controls, suggesting that PE inflexibility might be adaptive. Given the paucity of relevant research and inconsistent prior findings on PE network density, the current finding awaits replication. Nonetheless, it is of note that the current study had some methodological improvements over prior studies, such as assessing the full range of PE rather than only medium to high-arousal PE (Pe et al., 2015). In addition, the present finding aligns with the prior evidence that greater PE change resistance may characterize psychological health. For instance, less change in PE in response to daily stressors predicted a lower likelihood of depressive and anxiety disorders across seven years in middle-aged adults (Rackoff & Newman, 2020). Alternatively, PE inflexibility might be differentially associated with psychopathology depending on the context (e.g., positively associated with depression in stressful tasks, but not in positive tasks; Kuppens, Allen, & Sheeber, 2010). Given the sparse literature on PE network density, these speculations merit future investigation.

We found that information collected via mobile intensive longitudinal assessment improved diagnostic prediction above and beyond the effects of demographics. In the daily diary data set, mean NE and PE predicted the diagnostic status. In the EMA data set,
mean NE and NE and PE network density were significant predictors. Mean NE predicted diagnostic status whether it was assessed intra-daily or daily, and over 8 or 50 days. This suggests that mean NE may function as a stable, trait-like construct. In the EMA data set, NE and PE network densities showed incremental validity over demographics and the mean and standard deviation of emotions, increasing sensitivity and specificity by about 5%. Considering the scalability of mobile assessments, there is a potential for this seemingly modest incremental prediction to be impactful. For instance, if 1,000 individuals with undiagnosed GAD or MDD were to complete EMA on their emotions, considering emotion network densities in addition to the other predictors could lead to 50 more individuals correctly diagnosed rather than undiagnosed. A more robust and meaningful test of clinical utility of this approach would require examining whether emotion network densities provided incremental validity over traditional self-report symptom measures. Nonetheless, with the increasing evidence for its feasibility (e.g., van der Krieke et al., 2017), combining mobile assessments with automated statistical analyses holds promise for scalable, personalized diagnostic feedback.

It is important to note limitations with the current study. Although the EMA and daily diary samples showed no differences on demographics, baseline depression severity, and the proportion of individuals with anxiety or depressive disorders, it remains speculative if the difference in sampling rate alone accounted for the current findings. For instance, the degree of retrospection also differed between the EMA and daily diary samples. Daily diary reports might have been influenced by the peak (most intense) and end (final) moments of an emotional experience, as well as the average of experiences across the day (Schneider, Stone, Schwartz, & Broderick, 2011). Therefore, it cannot be ruled out that the retrieval process difference accounted for the current findings. Nevertheless, given the burden of EMA, it would be difficult to provide a direct comparison between daily diary and daily averaged EMA because it would require expanding the number of days of EMA to have requisite power for estimating emotion networks. For instance, it would have required participants to respond to 9 prompts per day for 50 days in the current study, which would have been highly cumbersome. Prior EMA studies assessing emotions in clinical populations also found that compliance decreased as the assessment duration increased (Courvoisier, Eid, & Lischetzke, 2012; Hoeppner, Kahler, & Gwaltney, 2014). Therefore, despite the potential for greater retrospective bias, daily diary may still have utility in assessing daily emotions over a relatively long period.

Another limitation is that contextual influences on the emotion ratings were not considered. The clinical group might have encountered fewer positive and more negative emotional stimuli than the control group (e.g., less activity and social interaction), which might have led to lower PE inflexibility and higher NE inflexibility. It is also of note that although the current study extended previous emotion network findings in depressed samples to a mixed sample of individuals with MDD and GAD, it remains to be tested whether MDD and GAD are differentiated based on emotion network density. In addition, the present study focused on cross-sectional associations of emotion network density with MDD and GAD. Using a longitudinal design would allow for testing whether emotion network density predicts the future onset or remission of anxiety and depressive disorders. Last, but not least, both EMA and daily diary samples mainly consisted of young adults and White individuals. Given the evidence for age differences in emotion regulation patterns (e.g., Labouvie-Vief, Diehl, Jain, & Zhang, 2007), the current findings warrant replication in more diverse samples.

Despite these limitations, the current findings represent an important contribution to the literature on emotional dynamics by examining emotion network density in two data
sets of different time frames. We examined how anxious and depressed individuals differed from healthy controls on emotion network density or temporal dependency among emotions. Our findings suggest the importance of considering the timescale of assessment in operationalizing and understanding dynamic emotional processes. Results also highlight how emotion network density might be differentially associated with anxiety and depression depending on the valence of underlying emotions. Finally, the present study provides preliminary support for emotion network density as a clinical marker of anxiety and depression and its potential utility in mobile-based diagnostic assessments.

Conflict of interest
All authors declare no conflict of interest.

Author contributions
Ki Eun Shin (Conceptualization; Formal analysis; Investigation; Methodology; Writing – original draft) Michelle G. Newman (Conceptualization; Methodology; Writing – review & editing) Nicholas C. Jacobson (Conceptualization; Investigation; Methodology; Writing – review & editing)

Data availability statement
Participant consent to data sharing in a public repository or upon request was not obtained for this research, so supporting data are not available.

References


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**Supporting Information**

The following supporting information may be found in the online edition of the article:

**Appendix S1**: Supplemental Materials.