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Characterizing emotion dynamics in remitted depression: A network approach using ecological momentary assessment

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ABSTRACT

Background: Maladaptive emotion dynamics and processes, such as emotional inflexibility and dominance of negative emotions, are characteristic of depression. The extent to which these abnormalities persist following depressive episodes, and represent a vulnerability factor for recurrent depressive episodes, remains unknown. The current study investigated whether emotion dynamics predict depressive symptoms in individuals with remitted depression (rMDD) and healthy controls (HC).

Methods: 98 adults (HC: n = 50; rMDD: n = 48) completed a three-week ecological momentary assessment protocol, in which they responded to two items probing positive emotions and three items probing negative emotions six times daily. Contemporaneous and temporal networks were constructed using multilevel vector autoregressive models. Density was calculated as a measure of emotional inflexibility and In- and Out-Expected Influence were calculated as measures of centrality. Linear regression models examined if density predicted clinical outcomes at the 6-month follow-up assessment.

Results: Individuals with rMDD had significantly denser temporal emotional networks than HC participants. Groups also showed differential patterns of the most influential nodes in temporal and contemporaneous networks. Greater temporal density and contemporaneous density was significantly associated with increased depressive symptoms at the 6-month follow-up, assessed through both clinician-rated and self-report measures.

Conclusions: Abnormalities in emotion dynamics persist following remission from depression and can be used to predict future depressive symptoms, suggesting these may represent a vulnerability factor for depression. Future research should study if interventions based on emotion networks targeting maladaptive emotional processes are able to prevent future depression.

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1. Introduction

Major depressive disorder (MDD) is a prevalent and disabling mental condition characterized by abnormalities in emotion processing and emotion regulation (ER), with increased negative affect (e.g., sadness) and decreased positive affect (e.g., anhedonia) reflecting key symptoms [1]. Individuals with depression often show difficulties in flexibly adjusting their emotions to external circumstances, as manifested in a reduced ability to inhibit negative emotions [2] alongside a tendency to dampen positive emotions [3–5]. Further, this emotional inflexibility has been theorized to contribute to the development of depression [4,6].

Disturbances in emotion are thought to persist following remission and may increase vulnerability to future episodes [7,8]. Negative affect may be particularly important in remitted MDD (rMDD), with research identifying increased variability and intensity in negative, but not positive, affect in individuals with rMDD [9–12]. Additionally, emotion dynamics may predict treatment response and remission, with negative affect levels predictive of both outcomes [13]. The abnormalities identified in rMDD may represent a "scar" of depression, referring to a lasting mark of or change due to the depressive episode [14], as evidenced by changes in emotion dynamics following episodes. However, such disturbances may also represent a vulnerability factor, as evidenced by research suggesting that affect dynamics can predict the course and onset of future depressive episodes [15,16]. Given these two putative roles, abnormalities in emotional dynamics should be addressed to reduce depression recurrence.

Previous research has often assessed emotional inflexibility using emotional inertia, the extent to which an emotion predicts itself from one timepoint to the next [17]; such studies generally conceptualize inertia as a form of emotional rigidity, with higher levels reflecting a tendency to become "stuck" in a particular emotional state. Studies have shown that emotional inertia, especially in negative emotions such as sadness, is elevated in currently depressed individuals compared to healthy controls (HC) [10], predicts depression severity [18], and forecasts the onset of depression in adolescents [15]. While this approach has advanced our understanding of maladaptive emotion dynamics in depression, the interpretation and applications of emotional inertia and other traditional indices of emotion dynamics are limited and fall short in capturing the complexity of emotional processes and their relationship to psychopathology [19]. Specifically, traditional metrics overlook the fact that emotions interact dynamically, with each emotion capable of predicting shifts in a range of other emotional states (e.g., anxiety giving rise to anger).

One promising approach to overcome such limitations is network analysis, which maps the dynamic relationships among emotions. In a network, variables such as emotions or symptoms are presented as "nodes" with lines between nodes, or "edges" representing associations between variables. Within-subject networks can be used to demonstrate associations between emotions both at a singular point in time (contemporaneous networks) as well as over multiple consecutive timepoints (temporal networks), enabling examination of emotional processes on multiple timescales. Once networks are constructed, various features can be extracted to aid in the interpretation of the results. Two network features, *density* and *centrality*, have received particular attention in psychological research.

Network density (or connectivity) assesses the strength of the connections between different emotions and can be used within network analysis to reflect emotional inflexibility, capturing the persistence of inter-emotional patterns through consistent emotional responses or cyclical emotional states. In a temporal network, density measures how each emotion at time t is predicted by that emotion as well as all other emotions in the network at time t–1 [20]. In a contemporaneous model, network density represents the strength of the connections between emotions at any given point in time, which may be indicative of the nuance of an emotional network and the participant's general ability to differentiate between specific facets of positive and negative emotion [21].

Network analysis becomes even more powerful when combined with ecologically valid mood assessment methods, such as ecological momentary assessment (EMA). Prior research using these methods has found that density in emotion networks of MDD and HC participants differed primarily on negative affect density, with increased density in participants with MDD, and no difference in positive affect density [20]. Investigating the predictive utility of density, Shin and colleagues [22] found that the densities of both positive and negative affect networks predicted clinical group status above and beyond mean affect levels and affective variability. Moreover, in single networks including both positive and negative affect, density was found to predict self-reported depressive symptoms [21]. Thus far, most research that has been conducted using network density has focused on identifying differences between clinical and HC samples [20,23], or on associating network-derived affective characteristics with momentary mood fluctuations [22,24], with little research conducted on the utility of emotion density in predicting future mood [21]. Further, no research has examined emotional inflexibility in samples with rMDD, which might provide key insights into whether increased inflexibility persists following depression and represents a vulnerability factor for future psychopathology. In light of evidence that individuals with rMDD tend to experience greater emotional inertia [9] and exhibit maladaptive emotional patterns following a depressive episode [11,12], it is likely that such individuals also exhibit greater emotional rigidity.

Further, it is also possible that individuals with rMDD exhibit differences in the structure of their emotional networks. Centrality, which measures the relative importance of a node and its influence [25], can be used to investigate the differential influence of specific emotions on the emotional system. Expected influence (EI) is a centrality measure optimally suited for networks that contain both positively and negatively valanced nodes [26]. In a temporal network, EI is separated into In-EI and Out-EI. In-EI measures the extent to which a node at timepoint t is influenced by other nodes at timepoint t–1, whereas Out-EI measures the extent to which a node at time t influences other nodes at time $t + 1$. As such, EI can be used to highlight which emotions are most impactful and impacted within an individual's emotional system. Much of the current research using centrality measures in depressed populations has focused on examining DSM-derived symptoms of depression rather than affect [27,28]. While this research has sought to identify core symptoms of depression, identifying highly influential emotions may be important in understanding depression trajectories. In addition, little is known about whether or how the influence of emotions changes following remission from depression.

The current study used EMA data to assess facets of positive and negative emotions several times daily for three weeks. Our aims were to (1) use network analysis to characterize the affective networks of individuals with rMDD and HCs, (2) compare emotional density and emotional centrality derived from these networks between rMDD and HCs, and (3) predict future mood states at a 6-month follow-up session using both self-reported and clinician-rated measures of depressive and anhedonic symptoms.

2. Methods

2.1. Participants

137 participants aged 18–45 were recruited, including 56 participants with rMDD and 81 HC participants. Participants were recruited through online advertisements and flyers posted in the community. Participants were screened by a masters or PhD-level clinician using the Structured Clinical Interview for DSM-5 Research Version (SCID-5-RV) [29]. Participants met criteria for rMDD if they had experienced at least one major depressive episode in the past five years and if their depression was in remission for at least two months prior to the screening session. In addition, clinical scores had to be below the following

thresholds: Beck Depression Inventory–II (BDI-II) [30] ≤ 9; Quick Inventory of Depression Symptomatology (QIDS) [31] ≤ 5; Hamilton Depression Rating Scale (HDRS) [32] ≤ 7 as well as no more than two symptoms of depression reported to more than a mild degree [SCID-5-RV rating of 2] in the eight weeks prior to testing. Accordingly, depressed mood and anhedonia symptoms had to be rated 1 on the SCID, excluding even subthreshold level of depressed mood and anhedonia for rMDD. All participants were unmedicated. Participants were classified as HC if they had no current or past psychiatric illness. Exclusion criteria for the full sample were current or past serious medical illness, current comorbid psychiatric disorders, first-degree relatives with a history of a psychotic disorder or psychotic symptoms outside of the context of a mood disorder, current use of psychoactive drugs, and more than 15 alcohol-induced blackouts.

Participants completed an EMA protocol as part of a larger neuroimaging study. EMA data were collected from 119 participants. Participants were excluded from the analysis if they completed less than half of the EMA surveys, leading to a final sample of 98 participants (HC: n = 50, rMDD: n = 48). Demographics and baseline characteristics of the final sample are presented in Table 1.

Table 1
Demographic and Clinical Characteristics of the Sample.

	HC (N = 50)	rMDD (N = 48)	t/χ^2 (df)	P-value
Age	27.52 (6.51)	25.77 (5.96)	1.39 (96)	0.169
Gender				
Female	37 (74.0%)	41 (85.4%)	1.33 (1)	0.250
Male	13 (26.0%)	7 (14.6%)		
Race				
White	29 (58.0%)	36 (75.0%)		0.0714
Asian	14 (28.0%)	10 (20.8%)		
American Indian/Alaskan Native	1 (2.0%)	0 (0%)		
Black or African American	4 (8.0%)	0 (0%)		
More than one	0 (0%)	1 (2.1%)		
Income				
Less than $10,000	3 (6.0%)	3 (6.3%)	1.43 (5)	0.921
$10,000–25,000	2 (4.0%)	2 (4.2%)		
$25,000–50,000	7 (14.0%)	11 (22.9%)		
$50,000–75,000	9 (18.0%)	9 (18.8%)		
$75,000–100,000	10 (20.0%)	9 (18.8%)		
More than $100,000	18 (36.0%)	14 (29.2%)		
Education				
High School	3 (6.0%)	0 (0%)		0.070
Some College	7 (14.0%)	15 (31.3%)		
Junior College	1 (2.0%)	0 (0%)		
Four-Year College	13 (26.0%)	14 (29.2%)		
Graduate or Professional School	26 (52.0%)	19 (39.6%)		
BDI-II Total Baseline	0.82 (1.67)	2.29 (2.87)	–3.11 (96)	0.002
BDI-II Total 6 M	1.58 (3.35)	4.43 (4.09)	–3.57 (85)	< 0.001
SHAPS Total Baseline	19.21 (6.20)	21.54 (6.04)	–1.96 (96)	0.053
SHAPS Total 6 M	19.87 (6.01)	19.76 (4.16)	0.09 (85)	0.925
HDRS Total Baseline	0.52 (0.84)	1.19 (1.38)	–2.91 (96)	0.005
HDRS Total 6 M	0.69 (1.56)	2.24 (2.13)	–3.89 (85)	< 0.001

Note. Chi-square tests were conducted for categorical variables. For categorical variables in which there were cell counts of 0 or 1, Fisher's Exact Test was conducted in place of the Chi-square test. Two Sample T-Tests were conducted for continuous variables. HC: Healthy Controls, rMDD: Euthymic individuals with remitted depression. BDI-II: Beck Depression Inventory, HDRS: Hamilton Depression Rating Scale, SHAPS: Snaith-Hamilton Pleasure Scale.

2.2. Procedure

The study was approved by the Mass General Brigham (MGB) Institutional Review Board (IRB), protocol numbers: 2014P001871 and 2018P000318, and participants provided written, informed consent after the procedures were explained. Participants first completed a screening session, consisting of informed consent, a clinical interview including the SCID-5-RV [29] and HDRS [32], and online questionnaires including the BDI-II [30] and Snaith-Hamilton Pleasure Scale (SHAPS) [33], a self-reported measure of anhedonia. Participants eligible following this screening visit were invited to complete the parent study's neuroimaging visit of the session, which took place within a month after the screening visit. If participants were unable to complete the two sessions within a month of each other, they completed a short reassessment visit (mood module of the SCID-5-RV) prior to the neuroimaging session to confirm their clinical status.

Participants downloaded a mobile application (MetricWire, Inc.) on their smartphone at the end of the neuroimaging session. They completed a brief survey to indicate their typical wake time, an example survey, and were given an opportunity to ask the research team questions about the procedure. The 21-day EMA period started the day following the neuroimaging session. Participants were prompted to complete surveys six times per day, for a maximum total of 126 surveys. Participants received one survey each morning at their designated wake time and five additional surveys randomly throughout the day between 10:30 am and 8 pm, with an interval of at least 1.5 h between times the surveys were sent. Surveys could be completed up until the next survey was sent, at which point it was counted as a missed signal. Each week, participants received an email reporting their compliance rate and compensation for the previous week. Compensation was given on a progressive schedule, where participants earned money for completing at least 70% of weekly surveys, with additional monetary bonuses for completing at least 80% of the surveys. The amount that participants were eligible to earn each week increased over the three-week EMA period.

Participants completed a follow-up assessment at six months following the neuroimaging session, where they filled out the same online questionnaires assessing depressive symptoms and anhedonia completed at baseline and a clinical interview. A total of 87 participants (89% retention rate; HC: n = 45, rMDD: n = 42) who had usable EMA data at baseline also completed the 6-month follow-up session.

2.3. Measures

2.3.1. EMA Items

The EMA protocol included measures of affect, anxiety, depression, and approach and avoidance behaviors. Given the study aims, the current analyses focused on the affect items, which were presented as grouped items: "cheerful/joyful/enthusiastic" (*Cheerful*), "calm/content/relaxed" (*Calm*), "nervous/worried/afraid" (*Nervous*), "annoyed/angry" (*Annoyed*), "sad/hopeless" (*Sad*). These items were grouped based on valence (positive: *Cheerful, Calm*; negative: *Nervous*, *Annoyed*, *Sad*) and arousal (high: *Nervous*, *Annoyed*; low: *Cheerful*, *Calm*, *Sad*) [34]. All items were preceded by the prompt: "For the next 5 questions, please answer them according to how you feel RIGHT BEFORE you started this survey." Participants rated each item on a 0–100 scale, where 0 indicated low levels of the emotion.

2.3.2. Baseline and Follow-Up Measures

2.3.2.1. Hamilton Depression Rating Scale. The HDRS was used as a clinician-administered depression measure. The HDRS is a 17-item scale that assesses depressive symptoms through a structured interview. Items are scored on a scale of either 0–4 or 0–2 then summed to create a total score, with higher scores indicating increased depression severity. The

internal consistency (assessed using Omega Total (ωt)) was relatively low at baseline (ωt = .52) and the 6-month follow-up (ωt = .66). These values are likely influenced by the low range in the sample since the rMDD sample was specifically recruited to be asymptomatic. Such floor effects reduce variability and inter-item correlations, which in turn deflate estimates of internal consistency.

2.3.2.2. Beck Depression Inventory-II. Participants completed the BDI-II, a 21-item questionnaire measuring the prevalence and severity of depressive symptoms over the past two weeks. Items are rated on a 0–3 scale then summed, with higher scores indicating greater depression severity. The internal consistency of the scale was acceptable at baseline (ωt = .79) and at the 6-month follow-up (ωt = .87).

2.3.2.3. Snaith-Hamilton Pleasure Scale. Anhedonia was assessed using the SHAPS, a 14-item questionnaire measuring hedonic pleasure across four domains: interests, social interaction, sensory experience, and food/drink. Items are scored on a scale of 1 (“strongly agree”) to 4 (“strongly disagree”) and summed to create a total score, with higher scores indicating greater anhedonia. The internal consistency was acceptable at baseline (ωt = .85) and at the 6-month follow-up (ωt = .86).

2.4. Statistical Analysis

Network estimates using correlated effects were derived from multilevel vector autoregressive models (mlVAR), implemented through the *mlVAR* package in R [35]. The temporal and contemporaneous networks were extracted from this model. In the *temporal network*, affect ratings at time *t* were regressed on the rating from the previous timepoint, time *t–1*. The value obtained for each represents the lagged association between emotions from one timepoint to the next while accounting for the influence of all other emotions in the network [36]. To ensure associations were captured within the same day, the lags from the last timepoint of the previous day to the first timepoint of the next day were excluded. The *contemporaneous network* was used to capture relationships between nodes at the same timepoint, controlling for the temporal associations and concurrent (same timepoint) associations with the other nodes [36]. To statistically compare the temporal and contemporaneous networks of HC and rMDD participants, permutation testing set to 1000 permutations was implemented through the *mnet* package in R [37].

Following previous research, density was calculated as the sum of the absolute values of each participant’s network effects [20,21]. Absolute value was used because of the focus on how closely related emotions were to each other, regardless of the direction of the association. For each participant, this yielded two values: a temporal network density and a contemporaneous network density. Greater temporal density indicates a more rigid, inflexible, resistant-to-change emotional network, while greater contemporaneous density indicates increased connectivity between emotions in the moment, suggesting that emotions co-occur, fluctuate together, and may be less differentiated at a given moment.

Centrality measures were extracted using the *qgraph* package in R [38]. This package provides estimates of multiple types of centrality measures, including In-EI and Out-EI. To derive these estimates, *qgraph* network objects were computed separately for each group, then centrality measures were extracted for both the temporal and contemporaneous networks.

To predict 6-month follow-up outcomes, linear regression models were used with density as the predictor. Separate models were conducted to predict 6-month BDI, HDRS, and SHAPS scores. Due to low variance in these outcome measures at baseline, initial models did not control for baseline scores. However, additional sensitivity analyses were conducted using hierarchical linear models in two steps to control for baseline scores of the measures before predicting 6-month outcomes. In step 1, a linear model was used to predict the 6-month follow-up score from the baseline symptom score. The residuals of the model were then extracted and used as the outcome variable in step 2, where density (either temporal or contemporaneous) was used as the predictor. For missing items on the baseline and follow-up clinical measures (BDI-II, SHAPS, and HDRS), multivariate imputation was performed using the *mice* package in R [39]. Imputation was not performed for participants who were lost to follow-up. Overall, there were little missing data: for the SHAPS baseline, two participants were each missing one item; for the SHAPS 6-month follow-up, one participant was missing one item, and one participant was missing two items; and for the HDRS baseline, one participant was missing one item.

3. Results

3.1. Attrition Analyses

Participants who did not complete at least half of the baseline EMA surveys and were thus excluded did not differ from included participants in terms of their BDI ($t(117) = 0.54$, $p = .589$), HDRS ($t(117) = 1.20$, $p = .235$), or SHAPS ($t(117) = –0.03$, $p = .974$) scores. Additionally, participants who were included at baseline but did not complete the 6-month follow-up did not differ from participants who completed the 6-month baseline based on BDI ($t(96) = –0.01$, $p = .995$), HDRS ($t(96) = 0.36$, $p = .723$), or SHAPS ($t(96) = 0.48$, $p = .632$).

3.2. Descriptives

Demographic and clinical information regarding the groups is summarized in Table 1. Though there were significant between group differences on the HDRS, SHAPS, and BDI, the mean levels for both groups were remarkably low and well within the range of psychologically healthy individuals at both timepoints [40,41]. Groups did not differ on the total number of surveys completed (HC: mean±SD=108.06 ± 16.38, rMDD: 105.12 ± 15.82, $t(96) = 0.90$, $p = .369$). Using multivariate ANOVA, there was a significant group difference in average levels of emotions throughout the EMA period (Wilks’ Λ=0.86, $F(5,92) = 3.05$, $p = .014$). Follow-up *t*-tests revealed, on average, during the EMA period, HC participants reported higher levels of *Cheerful* (HC: 65.70 ± 19.17; rMDD: 56.68 ± 20.48; $t(96) = 2.25$, $p = .027$) and *Calm* (HC: 66.83 ± 18.60; rMDD: 58.10 ± 20.60; $t(96) = 2.20$, $p = .030$) than rMDD participants, whereas rMDD participants reported greater levels of *Nervous* (HC: 8.06 ± 8.94; rMDD: 13.29 ± 11.33; $t(96) = –2.54$, $p = .013$) than HC participants. There were no differences in average reported levels of *Annoyed* (HC: 6.52 ± 8.95; rMDD: 8.47 ± 7.64; $t(96) = –1.16$, $p = .249$) or *Sad* (HC: 5.15 ± 8.78; rMDD: 5.68 ± 7.63; $t(96) = –0.32$, $p = .752$). Positive affect items were endorsed as present (i.e., non-zero) at nearly every timepoint (*Cheerful*: 98.89%; *Calm*: 98.83%). Negative affect items were endorsed as present less frequently (*Nervous*: 56.24%; *Annoyed*: 45.25%; *Sad*: 39.67%).

3.3. Network Analyses

3.3.1. Contemporaneous networks

The contemporaneous networks for rMDD and HC participants are shown in Fig. 1**A** and Fig. 1**B**, respectively. The connections identified as significant between nodes were largely similar across groups. However, the rMDD network included two significant connections that were absent in the HC network: *Cheerful–Nervous* (partial r = –0.074, $p = .002$) and *Sad–Calm* (partial r = –0.064, $p = .026$). However, permutation testing identified a significant difference between groups only in the *Cheerful-Nervous* connection (Δ=0.09, $p = .008$). Full results from the rMDD and HC contemporaneous models are presented in Table 2**A** and Table 2**B**, respectively. Full results from the permutation testing are presented in Table 3**A**.

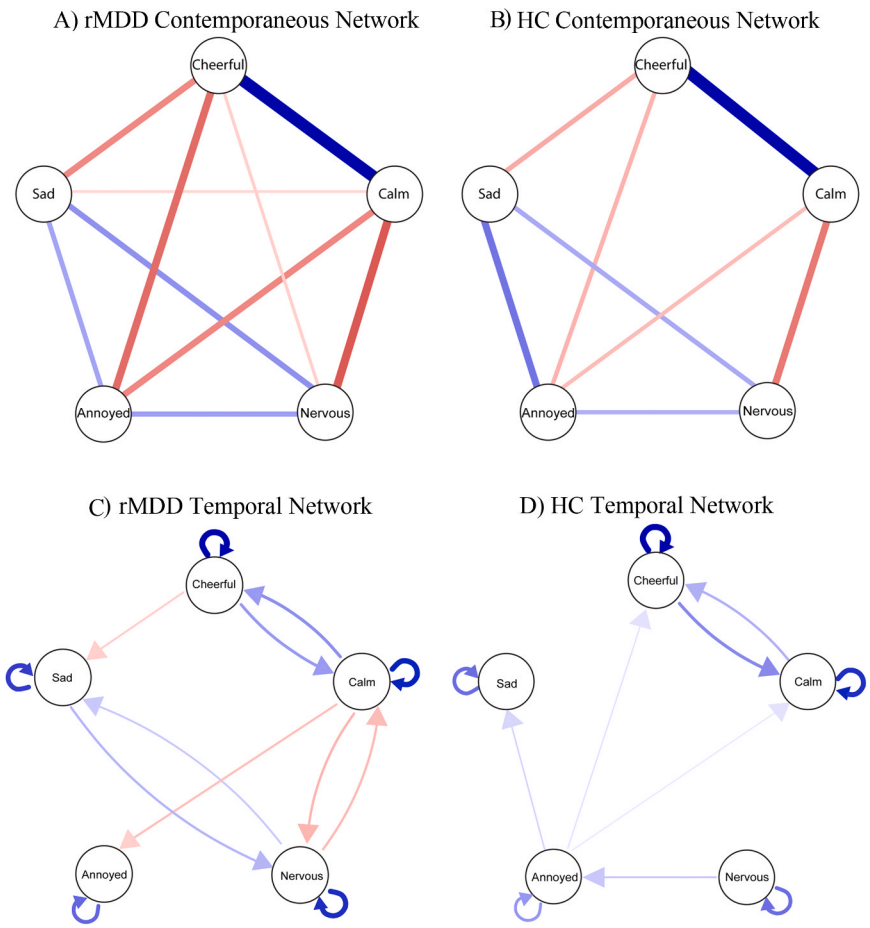

Fig. 1. A) Contemporaneous and C) Temporal networks for rMDD Participants and B) Contemporaneous and D) Temporal networks for HC participants. Only significant edges in each network are plotted on these graphs. Red edges indicate significant negative associations, and blue edges indicate significant positive associations. Greater line boldness indicates a greater strength of the association.

Table 2
Contemporaneous network results. Partial correlations, correlations, and resulting p-values for edges in the A) rMDD and B) HC contemporaneous network.

A) rMDD participants					
Variable 1	Variable 2	P 1→2	P 2→1	Partial r	r
Calm	Cheerful	< 0.001	< 0.001	0.351	0.502
Nervous	Cheerful	0.002	< 0.001	-0.073	-0.317
Nervous	Calm	< 0.001	< 0.001	-0.235	-0.395
Annoyed	Cheerful	< 0.001	< 0.001	-0.211	-0.404
Annoyed	Calm	< 0.001	< 0.001	-0.176	-0.390
Annoyed	Nervous	< 0.001	< 0.001	0.138	0.318
Sad	Cheerful	< 0.001	< 0.001	-0.175	-0.337
Sad	Calm	0.026	0.073	-0.064	-0.286
Sad	Nervous	< 0.001	< 0.001	0.157	0.295
Sad	Annoyed	0.002	0.001	0.131	0.295
B) HC participants					
Variable 1	Variable 2	P 1→2	P 2→1	Partial r	r
Calm	Cheerful	< 0.001	< 0.001	0.429	0.516
Nervous	Cheerful	0.299	0.457	0.017	-0.213
Nervous	Calm	< 0.001	< 0.001	-0.238	-0.346
Annoyed	Cheerful	< 0.001	< 0.001	-0.141	-0.322
Annoyed	Calm	< 0.001	< 0.001	-0.126	-0.326
Annoyed	Nervous	< 0.001	0.002	0.141	0.280
Sad	Cheerful	< 0.001	0.001	-0.155	-0.305
Sad	Calm	0.140	0.170	-0.049	-0.271
Sad	Nervous	< 0.001	< 0.001	0.151	0.271
Sad	Annoyed	< 0.001	< 0.001	0.235	0.352

Table 3
Empirical differences and resulting p-values from the mlVAR permutation test. Partial correlation differences and corresponding p-values of the A) contemporaneous and B) temporal network permutation-based group comparison test.

A) Contemporaneous network comparison					
	Cheerful	Calm	Nervous	Annoyed	Sad
Cheerful					
Calm	0.081				
	(.102)				
Nervous	0.092	–0.012			
	(.008)	(.787)			
Annoyed	0.070	0.064	0.001		
	(.096)	(.150)	(.976)		
Sad	0.014	0.016	–0.001	0.118	
	(.777)	(.727)	(.996)	(.074)	
B) Temporal network comparison					
	Cheerful	Calm	Nervous	Annoyed	Sad
Cheerful	0.048	–0.018	–0.009	0.044	0.005
	(.356)	(.744)	(.796)	(.131)	(.879)
Calm	0.039	0.026	0.051	0.027	–0.008
	(.365)	(.567)	(.125)	(.316)	(.815)
Nervous	–0.009	0.05	–0.044	0.021	–0.040
	(.853)	(.347)	(.358)	(.500)	(.370)
Annoyed	0.022	0.026	0.026	–0.030	–0.007
	(.625)	(.613)	(.587)	(.543)	(.884)
Sad	0.019	0.033	–0.051	0.024	–0.027
	(.672)	(.517)	(.071)	(.544)	(.625)

3.3.2. *Temporal networks*

The temporal networks for rMDD and HC participants are presented

in Fig. 1**C** and Fig. 1**D**, respectively. Both networks featured a significant, bidirectional relationship between *Calm* and *Cheerful*. However, the rMDD network presented an additional significant bidirectional relationship between *Nervous* and *Sad* ($\beta_{Nervous–Sad}$=0.062, $p_{Nervous–Sad}$=.005; $\beta_{Sad–Nervous}$=0.085, $p_{Sad–Nervous}$=.006) and between *Nervous* and *Calm* ($\beta_{Nervous–Calm}$=–0.083, $p_{Nervous–Calm}$<.001; $\beta_{Calm–Nervous}$=–0.089, $p_{Calm–Nervous}$=.017). Permutation testing identified no statistically significant differences between groups. Full results from the temporal model for rMDD and HC participants are presented Table 4**A** and Table 4**B**, respectively. Full results from the permutation testing are presented in Table 3**B**.

Table 4
Temporal network results. Temporal network results for the A) rMDD participants and B) HC participants.

A) rMDD participants				
From	To	β	SE	p
Cheerful	Cheerful	0.277	0.032	< .001
Cheerful	Calm	0.113	0.027	< .001
Cheerful	Nervous	-0.019	0.029	0.511
Cheerful	Annoyed	0.004	0.030	0.891
Cheerful	Sad	-0.057	0.025	0.024
Calm	Cheerful	0.124	0.033	< .001
Calm	Calm	0.242	0.039	< .001
Calm	Nervous	-0.089	0.038	0.017
Calm	Annoyed	-0.076	0.037	0.039
Calm	Sad	-0.024	0.035	0.487
Nervous	Cheerful	-0.003	0.021	0.874
Nervous	Calm	-0.083	0.019	< .001
Nervous	Nervous	0.230	0.036	< .001
Nervous	Annoyed	0.050	0.033	0.138
Nervous	Sad	0.063	0.022	0.004
Annoyed	Cheerful	0.001	0.017	0.949
Annoyed	Calm	0.011	0.018	0.532
Annoyed	Nervous	-0.013	0.019	0.504
Annoyed	Annoyed	0.167	0.034	< .001
Annoyed	Sad	0.031	0.022	0.159
Sad	Cheerful	-0.030	0.019	0.110
Sad	Calm	0.022	0.021	0.293
Sad	Nervous	0.085	0.031	0.006
Sad	Annoyed	0.029	0.029	0.319
Sad	Sad	0.212	0.033	< .001
B) HC participants				
From	To	β	SE	p
Cheerful	Cheerful	0.325	0.038	< .001
Cheerful	Calm	0.152	0.025	< .001
Cheerful	Nervous	-0.028	0.030	0.354
Cheerful	Annoyed	0.026	0.029	0.353
Cheerful	Sad	-0.038	0.028	0.166
Calm	Cheerful	0.106	0.027	< .001
Calm	Calm	0.268	0.031	< .001
Calm	Nervous	-0.039	0.030	0.190
Calm	Annoyed	-0.050	0.032	0.118
Calm	Sad	0.009	0.029	0.753
Nervous	Cheerful	-0.012	0.023	0.605
Nervous	Calm	-0.032	0.023	0.165
Nervous	Nervous	0.186	0.027	< .001
Nervous	Annoyed	0.076	0.036	0.038
Nervous	Sad	0.012	0.018	0.509
Annoyed	Cheerful	0.045	0.019	0.018
Annoyed	Calm	0.039	0.016	0.016
Annoyed	Nervous	0.008	0.021	0.692
Annoyed	Annoyed	0.137	0.031	< .001
Annoyed	Sad	0.054	0.025	0.027
Sad	Cheerful	-0.025	0.020	0.204
Sad	Calm	0.014	0.021	0.500
Sad	Nervous	0.045	0.024	0.063
Sad	Annoyed	0.022	0.028	0.445
Sad	Sad	0.186	0.028	< .001

Beta values represent the strength of the connection between nodes. Significant values indicate that the edge significantly differs from 0.

3.4. Density

Density was computed for the full baseline sample as well as for the subsample of participants who completed both the baseline and 6-month follow-up measures. For the baseline sample, groups significantly differed on temporal density (HC: 2.16 ± 0.43; rMDD: 2.37 ± 0.47; t(96) = –2.33, p = .022), such that participants in the rMDD group had denser networks than the HC group. Groups did not differ in contemporaneous density (HC: 3.62 ± 0.68; rMDD: 3.83 ± 0.82; t(96) = –1.39, p = .167). Similarly, when restricting the sample to only participants with follow-up data, the temporal density was significantly higher in the rMDD sample (HC: 2.16 ± 0.45; rMDD: 2.38 ± 0.49; t(85) = –2.28, p = .025) compared to the HC sample, and there was no significant difference in the contemporaneous density (HC: 3.63 ± 0.70; rMDD: 3.92 ± 0.82; t(85) = –1.77, p = .081).

3.5. Emotional Density Predicting 6-Month Follow-Up Outcomes

3.5.1. Clinician-Rated Depression Symptoms (HDRS)

Greater temporal (β=1.13, p = .011) and contemporaneous (β=0.712, p = .010) densities significantly predicted higher HDRS scores. Critically, in hierarchical regression models controlling for baseline HDRS scores, the effect of temporal (β=1.03, p = .019) and contemporaneous (β=0.58, p = .035) density remained significant.

3.5.2. Self-Reported Depression Symptoms (BDI-II)

Greater temporal (β=2.42, p = .006) and contemporaneous (β=1.30, p = .018) densities significantly predicted higher BDI-II scores. In hierarchical regression models controlling for baseline BDI-II scores, the effects of temporal and contemporaneous density became insignificant (ps >.096).

3.5.3. Self-Reported Anhedonia Symptoms (SHAPS)

Greater temporal (β=2.73, p = .018), but not contemporaneous (β=0.920, p = .225) densities significantly predicted increased SHAPS scores. Controlling for baseline SHAPS scores in the hierarchical regression model, the effects of temporal density and contemporaneous density were insignificant (ps > .070).

3.6. Centrality Measures

Centrality measures (i.e., In-EI and Out-EI) were calculated separately for each group for both temporal and contemporaneous networks. In-EI and Out-EI measures for the temporal network for rMDD and HC participants are shown in Fig. 2**A** and Fig. 2**B**, respectively. EI values for the contemporaneous network for rMDD and HC participants are shown in Fig. 2**C**.

In the rMDD temporal network, *Cheerful* had the greatest In-EI (EI=0.12), indicating that cheerfulness was the most influenced by emotions at the previous timepoint, and increased when these emotions increased, while *Sad* had the greatest Out-EI (EI=0.08), indicating that sadness most strongly influenced emotions at the following timepoint. In the rMDD contemporaneous network, *Calm* was the most influential emotion (EI=–0.12), indicating that increases in other emotions in the network suppressed feelings of calmness, while increases in calmness suppressed other emotions in the network.

In the HC temporal network, *Calm* had the highest In-EI (EI=0.19), indicating that it was the most influenced by other emotions in the network. *Cheerful* had the greatest Out-EI (EI=0.15), followed closely by *Annoyed* (EI=0.14), indicating that increases in these emotions led to increases in other emotions in the network at the next timepoints. Notably, the In-EI for *Nervous* was 0, indicating that nervousness was not impacted by other emotions in the network, and the Out-EI for *Sad* was 0, such that sadness at one timepoint did not have an impact on other emotions at the next timepoint. In the HC contemporaneous network, *Sad* was the most influential emotion (EI=0.23), suggesting that when

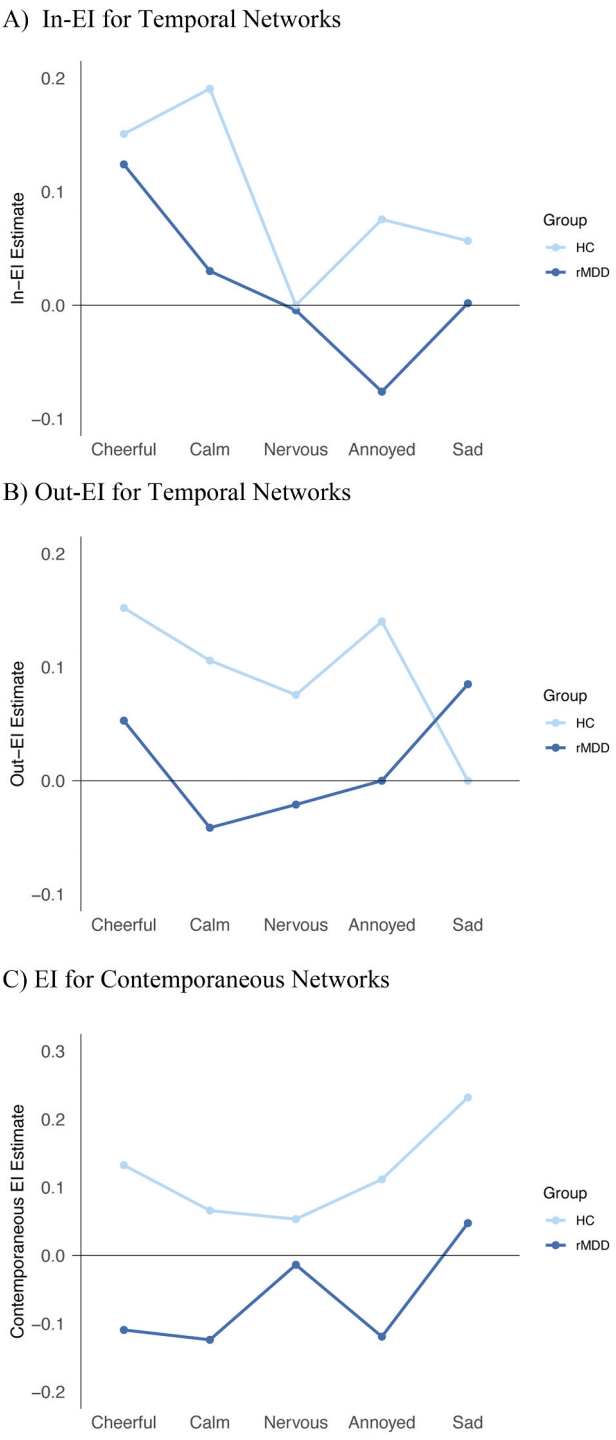

Fig. 2. A) In-Expected Influence, B) Out-Expected Influence, and C) Expected Influence values for temporal and contemporaneous networks for rMDD and HC participants. In-EI represents the amount to which a node is influenced by the other nodes at timepoint $t - 1$. Positive values mean that as the values of other nodes increase, the node's values also increase, whereas negative values suggest that increases in other nodes cause a decrease in that node. B) Out-EI represents the extent to which a node influences other nodes in the network at timepoint $t + 1$. Positive values mean that the node causes increases in the other nodes, whereas negative values mean that the node causes decreases in the other nodes. C) In contemporaneous networks, EI measures the amount of influence a node has on other nodes, with positive values suggesting that it is related to increases in other nodes and negative values suggesting it is related to decreases in other nodes.

sadness is experienced, it dominates the other emotions at that timepoint.

4. Discussion

In the current study, we used network analysis to characterize emotion networks in rMDD and HC participants. Using density and expected influence, we found that rMDD participants were characterized by a denser temporal emotional network than HC participants. Moreover, both temporal and contemporaneous emotional densities predicted future depressive and anhedonic symptoms, and the two groups diverged in which emotions were most influential over concurrent and future emotions. These findings have several important implications.

First, exploring the individual networks of the rMDD and HC participants provided additional insight into the emotional dynamics of these groups. Although there was no overall difference between the rMDD and HC groups in contemporaneous density, there was a significant group difference in the *Cheerful–Nervous* edge in the contemporaneous networks, such that rMDD participants had a stronger inverse association between these emotions. Additionally, trends in other edges between positive and negative emotions (for example, *Cheerful–Annoyed* in the contemporaneous network) may suggest increased emotional polarization, the experience of positive and negative emotions as mutually exclusive [42]. Participants with rMDD also had an increased number of inverse correlations between positive and negative emotions, for example, *Cheerful* being negatively associated with *Sad* at the following timepoint in the temporal network. While network edges should be interpreted with caution given their instability [43], this results may point toward an additional possibility for how emotion dynamics are related to or could lead to future depression. In line with this conjecture, polarization of emotion has previously been associated with increased depressive symptomatology [42], and its presence in the rMDD network may suggest maladaptive emotional processes that could increase the likelihood of recurrence. The number of significant differences between the contemporaneous and temporal networks was relatively low. This may be expected given that the rMDD sample was not currently experiencing an active depressive episode, with participants showing very low levels of depressive symptoms and negative affect at the time of the study. Individuals in remission function closer to a normative emotional baseline, and their momentary affective dynamics would therefore be expected to more closely resemble those of healthy controls than during an active episode.

Second, rMDD participants had a significantly denser temporal emotional network than HC participants. Denser emotional networks are thought to represent maladaptive ER patterns, particularly, a reduced ability to update emotional states based on changes in the external environment, and therefore less flexible responding [20]. Increased emotional density has been shown to predict diagnostic status [22], and our findings extend previous studies by investigating this construct in individuals with rMDD, highlighting a persistence of this state following remission. Although prospective studies in at-risk samples will be needed for conclusive evidence, these findings raise the possibility that increased emotional density may be a "scar" of depression, such that an individual's emotional experience is changed as a result of experiencing depression [44]. These findings suggest that even though individuals who remit from depression may no longer experience clinical symptoms of depression, underlying maladaptive emotional processes may persist, potentially increasing risk for future episodes. However, because the sample consisted of participants with rMDD, we cannot rule out the possibility that these emotional processes were a vulnerability factor, preceding the first episode of depression. The difference in density in the temporal network but not in the contemporaneous network may be driven by the relative importance of the interaction of emotions and their inertia over time having more relative influence on symptomatology than within-moment emotional experiences. This is supported by previous research that emphasizes the importance of time-varying

metrics, such as inertia and variability, over traditional metrics [12,45]. By moving beyond metrics such as inertia that focus on a singular emotion, the use of network analysis allowed us to dissect important connections between emotions that would not have been captured by traditional metrics. Overall, these results underscore the importance of investigating both temporal and contemporaneous emotional dynamics in rMDD.

Third, emotional density at baseline was associated with both clinician-rated and self-report depressive symptoms at the 6-month follow-up, though only clinician-rated depressive symptoms survived controlling for baseline symptoms, suggesting emotional density may be a vulnerability factor for future depressive symptoms. The increased strength of the association with the HDRS relative to the BDI and SHAPS aligns with previous research reporting higher effect sizes when using clinician-reported outcomes than self-report outcomes [46]. These findings build on prior research in adolescents showing that greater emotional inertia predicts future depressive episodes [15] and extend it by investigating an adult, unmedicated, fully remitted sample and quantifying the connectivity of the entire emotional network as a predictor rather than inertia of specific emotions. To our knowledge, this is the first study to use emotional density to prospectively predict depressive symptoms in adults, although one study has found similar results in an adolescent sample [21]. These findings suggest that targeting maladaptive emotional processes may be a key area of intervention to prevent future depressive episodes, although further research is needed to clarify whether emotional density is a scar of depressive episodes, a vulnerability factor, or both.

Lastly, the centrality of emotions – as measured through EI – showed differing patterns for rMDD and HC participants, especially for the influence of *Sad*. Specifically, in the rMDD temporal network, *Sad* had the strongest Out-EI, highlighting a pervasive impact on other emotions in the network at future timepoints and suggesting that following remission from depression, sadness remains a core emotion. Additionally, since this association was positive, it shows that as sadness increases, the intensities of other emotions in the network also increase, potentially creating a negative feedback loop. However, it is also important to consider that these effects may be due to external events or stressors, such that *Sad* is an initial emotion in response to an event, but that a situational factor may be responsible for the changing dynamics following this emotion. Importantly, the impact of *Sad* as a temporally influential emotion was unique to the rMDD participants. In the HC temporal network, the Out-EI of *Sad* was 0, indicating that it had no impact on future emotional ratings. However, *Sad* remained an important node in HC participants in the contemporaneous network, where *Sad* had the greatest EI, suggesting it has a transient impact on the network. One possible explanation for these results is that although *Sad* may dominate the emotional network immediately, HCs may more effectively cope with negative emotions and readily disengage from them. While these results provide some insight into the relative importance of certain emotions, it is important to note that centrality measures tend to be unstable except in very large sample sizes [43], so these results should be interpreted with caution.

While this study provides novel insights into emotion dynamics in rMDD and the predictive power of these dynamics, several limitations should be noted. First, because our sample focused on individuals with rMDD and did not include currently depressed participants, there was little variability in baseline and follow-up levels of depressive symptoms, limiting our ability to detect significant differences when controlling for baseline levels. Similarly, because very few individuals in our sample (6.90%) experienced either a first episode (HC participants) or a recurrence (rMDD participants) of MDD between baseline and the 6-month follow-up, we lacked the power to predict depressive episode onset and instead focused on dimensional measures of symptoms. Accordingly, future studies should replicate and extend our findings in currently depressed individuals or individuals with subclinical depressive symptoms and use longer longitudinal timeframes to provide additional insight into the trajectories of emotion networks throughout the course of MDD. Second, since the data for this study were drawn from a broader study utilizing neuroimaging techniques, participants had to meet strict eligibility requirements regarding medical history and comorbidities. Such exclusions may limit the generalizability of the presented results. Relatedly, data for the current analyses were drawn from a larger neuroimaging study, which precluded the ability to conduct prospective power analyses or modify sample size. Whereas calculating exact power for network models is highly complex and depends heavily on specific edge weights [47], recent extensive simulation studies by Ávalos-Tejeda & Calderón [48] established the sample-to-variable ratio (n/k) as a practical heuristic for network recovery. Specifically, their simulations demonstrated that a minimum ratio of 10:1 serves as a robust threshold for obtaining reliable edge estimates and centrality indices with low bias. The current study included $N = 98$ participants and $k = 5$ emotion variables, yielding a ratio of 19.6:1, which safely exceeds this recommended threshold.

Third, although the emotions assessed covered both positive and negative valences and high and low arousal, emotions were presented as grouped categories rather than individually, limiting our ability to detect more subtle nuances in emotional relationships. While this grouping did capture both positively and negatively valanced emotions as well as high and low intensities [34], future studies should incorporate more emotional nodes with larger sample sizes to detect such differences. Fourth, though no edges in the temporal network reached significance, it is important to note that the permutation test used reaches peak performance with larger sample sizes and larger empirical differences than those collected and observed in the present analysis. Therefore, future studies incorporating larger sample sizes may help better elucidate potential differences between networks. Fifth, temporal network models assume equal spacing between observations. Although intervals varied slightly, assessments were administered within relatively constrained windows. Thus, estimates reflect average lagged associations across these intervals. Future work could apply continuous-time models (e.g., ctsem) to explicitly account for timing variability. Lastly, because our EMA protocol did not include any contextual variables, it is unknown whether outside factors such as stressful events contributed to the emotion dynamics observed in the study. Including questions related to life events and stressors may be of interest in interpreting emotion dynamics and understanding how external events impact the persistence of emotional states.

Taken together, our results highlight the importance of investigating emotion dynamics as both a consequence of and potential precursor to psychopathology by demonstrating persistent alterations in emotion networks following remission from depression. Considering these alterations and continuing to investigate emotion dynamics is important for understanding how to prevent depression onset and recurrence. Importantly, our results highlight underlying affective mechanisms of depression, and understanding these mechanisms can help us to choose more effective, efficient interventions and inform prevention efforts. For example, individuals with denser emotional networks may benefit from interventions that target emotional rigidity and aim to increase flexibility. Techniques drawn from dialectical behavior therapy, such as acceptance and ER skills, may be useful in increasing emotional flexibility, helping individuals modulate their emotional responses. In future work, it will be important to explore whether targeting emotional dynamics prior to the onset of depression prevents the occurrence of depressive episodes and decreases the chance of relapse.

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Declaration of Competing Interest

Over the past three years, Dr. Pizzagalli has received consulting fees from Abbvie, Arrowhead Pharmaceuticals, Boehringer Ingelheim, Circular Genomics, Compass Pathways, Engrail Therapeutics, Magnetic Tides, N1 Biocorp, Neumora Therapeutics, Neurocrine Biosciences, Neuroscience Software, Syntropic Medical, and Xenon Pharmaceuticals; he has received honoraria from the American Psychological Association, Psychonomic Society and Springer (for editorial work) and from Alkermes; he has received research funding from the BIRD Foundation, Brain and Behavior Research Foundation, Circular Genomics, DARPA, Millennium Pharmaceuticals, NIMH and Wellcome Leap MCPsych; he has received stock options from Ceretypes Neuromedicine, Compass Pathways, Engrail Therapeutics, Neumora Therapeutics, and Neuroscience Software. Dr. Webb receives consulting fees from King & Spalding law firm. No funding from these entities was used to support the current work, and all views expressed are solely those of the authors. The other authors declare no competing financial interests.

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