The X-Factor: Investigating Turning Points in Psychotherapy for Depression

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Abstract

This study investigates the dynamics of network activation in Exposure-Based Cognitive Therapy (EBCT) for depression, informed by Adele Hayes' Network Destabilization and Transition (NDT) model. Using a dataset of 75 participants, we examined whether observed network dynamics followed those predicted by the NDT model. Multiple regression analyses focused on the relationship between cognitive-emotional processing peaks during phase 2 of therapy and improvements in depression outcomes. Our findings reinforce that peak cognitive-emotional processing significantly predicts better treatment outcomes, supporting the critical role of these peaks in therapeutic change. However, an interaction analysis did not show a significant synergistic effect between cognitive-emotional processing peaks and network strength shifts. Exploratory analyses further revealed the importance of psychological flexibility, as indicated by the network cross-rate, and the activation of positive emotions early in treatment. Higher network cross-rates and initial positive network activation were linked to greater improvements, underscoring the potential benefit of promoting psychological flexibility and positive network activation through therapeutic intervention strategies. Future research should explore the longitudinal dynamics of network activation with higher temporal resolution and investigate interventions that enhance psychological flexibility and positive emotion activation.

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"there is a light somewhere.

it may not be much light but

it beats the darkness."

- *The Laughing Heart* (Bukowski, 1968)

Complexity in Psychology

The complexity theory of psychopathology offers a transformative lens through which to view mental health and its disturbances, challenging traditional notions of psychopathology as a static or linear phenomenon. This theory posits that psychopathology and mental health are dynamic patterns; not mere disruptions of a healthy state but distinct orders in themselves (Olthof et al., 2022). Complexity science has been relevant in the field of developmental psychology for decades (Ford, 1987; Smith & Thelen, 1994). While some researchers have applied these concepts in clinical psychology for years (Hayes & Strauss, 1998; Schiepek et al., 1992; Tschacher & Scheier, 1997), recent advancements in intensive data-collection methods have led to a surge in new research and discourse investigating psychotherapeutic change processes through a complexity lens (Bos & de Jonge, 2014; Curtiss et al., 2021; van de Leemput et al., 2014; Olthof et al., 2020; Schiepek et al., 2015; Wichers & Groot, 2016, to name but a few).

The current study will take a close look at a specific model for applying these ideas to psychotherapy: Adele Hayes' Network Destabilization and Transition (NDT) model (Hayes & Andrews, 2020b). Two essential concepts from complexity science in the model are attractor states and phase transitions. Attractor states represent stable patterns of thought, emotion, and behavior that individuals gravitate towards. In the context of psychotherapy, these states can manifest as either desirable or undesirable patterns (for example, a streak of uninterrupted productivity, or a bout of sadness). An undesirable attractor state becomes pathological when it gets so strong that the system can no longer escape its pull. This fits coherently with a growing body of clinical literature that conceptualizes depression as a particular configuration of a complex, interconnected, multi-modal psychological network (Borsboom & Cramer, 2013; Hayes et al., 2015; Teasdale, 1999; Young et al., 2003). It also conforms with Holtzheimer and Mayberg’s definition of depression as being stuck in a rut. They define depression as an individual’s tendency to enter, and the inability to disengage from, a negative mood state; rather than the mood state itself (Holtzheimer & Mayberg, 2011).

Attractor states can be viewed as either two states within a single network or as two distinct sub-networks within the individual's broader psychological network. For the purposes of this study, it is not crucial to distinguish between these conceptualizations. For the analyses of the current study, the healthy state is referred to as the positive network, and the pathological state as the negative network.

Phase transitions refer to the critical periods of change where a system shifts from one attractor state to another. These transitions are often marked by increased instability and fluctuations in psychological states, reflecting a system that is reorganizing itself (Olthof et al., 2022). One major aim of the NDT model is recognizing and facilitating these phase transitions, as they represent the system assuming a configuration that no longer favors the undesirable attractor state. As such, they represent major transformative moments in the therapeutic process (Hayes & Andrews, 2020a).

Exposure Based Cognitive Therapy and The NDT Model

Exposure-Based Cognitive Therapy (EBCT) is a treatment that applies principles of exposure therapy within a cognitive-behavioral framework to address depression. It targets the mechanisms that maintain depression, such as rumination, avoidance, and the inability to sustain positive emotion, aiming to optimize emotional processing and facilitate new learning (Hayes et al., 2022). EBCT involves a three-phase process, grounded in the NDT model: weakening the pathological attractor state, destabilization and transition, and strengthening the healthy state. The following section provides a brief description of EBCT therapy. For a more detailed account, see Hayes et al., 2022.

Phase 1: Lockdown Release

The first phase of EBCT focuses on weakening the pathological attractor state by identifying and targeting the “lockdown mechanisms” that maintain depression. In this phase, therapists help clients recognize when their depressive network is activated and develop adaptive skills to counter these processes. Activities include mapping out the depressive network, teaching mindfulness and distress tolerance techniques, and encouraging engagement in healthy lifestyle habits. The goal is to increase flexibility and energy for change, setting the foundation for further therapeutic work (Hayes et al., 2022).

Phase 2: Destabilization and Emotional Processing

The second phase involves destabilizing the pathological patterns and facilitating emotional processing. This is achieved through exposure exercises designed to activate and destabilize the depressive network. Therapists guide clients in recounting significant depressive experiences; encouraging them to face and process difficult emotions. The focus is on tolerating distress and fostering new, more constructive perspectives (Hayes et al., 2022).

Phase 3: Positive Growth

The final phase aims to strengthen the healthy attractor state by developing and reinforcing healthy patterns of functioning. Therapists work with clients to elaborate on the positive view of self and associated emotions, behaviors, and physiological responses. This involves engaging in exercises that activate positive emotions and integrating these experiences into a coherent positive network. The goal is to make this positive network the new default state, reducing the likelihood of relapse (Hayes et al., 2022).

Research on EBCT highlights its significant benefits in treating major depressive disorder (MDD). Initial trials showed substantial reductions in depressive symptoms and improvements in quality of life, with low dropout rates (Hayes et al., 2005, 2007). A Swiss trial and a subsequent RCT confirmed these findings, showing comparable effectiveness to traditional CBT and sustained symptom relief (Grosse Holtforth et al., 2012, 2017). EBCT effectively decreases unproductive processing and avoidance while increasing mindfulness and self-efficacy, aligning with the NDT model's goals (Gómez Penedo et al., 2020; Kumar et al., 2008). The therapy's emphasis on destabilizing depressive networks and fostering constructive emotional processing leads to lasting improvements in depression and overall well-being (Gómez Penedo et al., 2020; Grosse Holtforth et al., 2012).

Cognitive-Emotional Processing

One of the key mechanisms of EBCT, promoted during the second phase of the treatment, is cognitive-emotional processing. This refers to the integration and restructuring of thoughts and emotions that contribute to depression. This process reflects the client’s ability to engage with and process depressive thoughts and feelings constructively. Research has shown that clients who experience their highest levels of cognitive-emotional processing during the second phase of EBCT show improved treatment outcomes compared to those whose cognitive-emotional processing peaks during the first phase (Hayes et al., 2005, 2007). Furthermore, depression scores tend to drop in the session following peak cognitive-emotional processing that occurs during phase 2 of treatment, indicating the critical role this process plays in facilitating therapeutic change and reducing depressive symptoms (Hayes et al., 2007).

Research Questions

This paper aims to replicate the findings of the initial studies (Hayes et al., 2005, 2007), that a peak in cognitive-emotional processing during phase 2 of therapy predicts better treatment outcomes. The narrative data used in this study consists of participants' journal entries responding to an open-ended prompt about their depression, collected between therapy sessions to capture their subjective experiences and psychological states throughout the therapeutic process. It has been subsequently coded using the CHANGE coding system, developed by Adele Hayes. This is a method used to analyze therapy session narratives by assessing the valence and intensity of various content areas including cognitive-emotional processing.

This thesis will explore the relationship between cognitive-emotional processing and depression outcomes. It will also examine the changing relative strengths of the pathological and healthy attractors states. Looking at clients who showed improvement during therapy, if we were to overlay time-series plots of the strength of the pathological and healthy attractor states, we would expect to see an “X” where the values associated with the strength of each of the two attractor states cross over. This would visually represent the successful weakening of the negative system and simultaneous strengthening of the positive system. While the presence of a cognitive-emotional processing peak during phase 2 of EBCT represents the transition and reorganization of the system to the healthier state. Therefore, if the “X” occurs in temporal proximity to the peak cognitive-emotional processing period, this would represent an ideal treatment process according to the NDT model. As such, we would expect to see greater improvement with lasting effects compared to clients who did not follow this hypothesized ideal trajectory.

Research Questions:

1. Among improvers, does the strength of the positive and negative networks follow the NDT model of change? In other words, does the pathological attractor state weaken during the first phase of treatment and does the healthy attractor state strengthen during the last phase of treatment?
2. Does peak cognitive emotional processing in the second phase of EBCT treatment predict a better outcome for depression?
3. Is there an interaction effect between the occurrence of peak cognitive-emotional processing and the reversal of attractor state strength (when the healthy state out-performs the pathological)?

Methods

Data Collection

The dataset utilized in this study comprises pre-existing data originally collected and coded by Adele Hayes et al. in the United States, involving individuals undergoing psychotherapy for depression. This dataset consists of two subsets: the first subset has been previously analyzed and published in Hayes et al. (2005), and Hayes et al. (2007), while the second subset has not yet been published. Combining both subsets, the current dataset includes

94 participants that underwent EBCT therapy for depression. To capture the subjective experience and psychological states of the participants throughout the therapeutic process, they were prompted to write journals. Between each therapy session, participants responded to an open-ended prompt encouraging them to write about their depression. In addition to the added participants, the current study employs different analysis techniques from those used in the previous publications, providing a novel perspective on the data.

The treatment's three-phase model allows for a systematic examination of changes across the stages of treatment, which are crucial for investigating the dynamics of psychological transitions in depression. Due to the current study's focus on how variables change across treatment phases, only participants who finished the entire treatment or at least advanced to the third phase were included in the final sample. This criterion led to the inclusion of 75 participants in the current analysis.

In order to maintain participant confidentiality and ethical handling of sensitive psychological data, the dataset was anonymized prior to analysis. Consequently, demographic details such as age, gender, and cultural background were not available for this study.

Depression Measures

In this study, data was collected at two separate treatment sites. At the first site, depression severity was assessed using the self-report format of the Inventory of Depressive Symptomatology (IDS-SR) (Rush et al., 2000). At the second site, the Hamilton Depression Rating Scale (HDRS) was used (Hamilton, 1986). The HDRS scores were later converted to IDS scores using conversion tables provided with the IDS (*IDS/QIDS*, n.d.), derived from the work of Rush et al. (2003). This conversion ensures that the measures are comparable and can be analyzed within a unified framework.

Depression was measured before and after treatment for each participant. An improvement score was calculated by subtracting participants' final IDS score from their initial score. This method quantified the magnitude of improvement experienced by each participant over the course of treatment.

IDS is a suitable choice for the primary analysis because of its demonstrated sensitivity to detect changes in symptom severity (Rush et al., 2000). Furthermore, a psychometric analysis by Trivedi et al. (2004) determined that a change of at least 10-points on the IDS-SR is considered minimally clinically significant, providing a clear benchmark for evaluating the impact of EBCT on depressive symptoms.

The CHANGE Coding System

The dataset for the current study employed the CHANGE coding system, developed by Hayes et al., to analyze the psychological narratives collected from participants (Hayes et al., 2007). This system assesses the valence (positive or negative) and intensity (ranging from 0, indicating very low or not present, to 3, indicating high) of various content areas. These areas include Emotion, Behavior, View of Self, Relationship Quality, Hope, and Somatic Functioning. Additionally, four process variables are coded: Avoidance, Cognitive-Emotional Processing, Unproductive Processing, and Overgeneralization. These latter variables are coded for intensity (0 - 3) but not valence (Hayes et al., 2007). Coding of each narrative was done by two clinical psychologists. In the final data set, the mean of both coders' scores were used for each variable.

In the current study, the primary variables derived from this coding were the sums of domain scores for the negative and positive networks. In order to approximate the current strength of the positive network at a given time-point the intensity scores with a positive valence were summed across the affective, behavioral, and cognitive domains. The same was done for the negative network variables resulting in a positive network activation score and a negative network activation score between 0 and 9. The affect and behavior domain scores correspond directly to these areas in the CHANGE coding system. The cognitive domain score is the maximum value for a given session among the variables View of Self, Relationship Quality, and Hope. For instance, if the ratings (0=*very low to absent* to 3=*high*) for a given session on View of Self, Relationship Quality, and Hope are 0, 2, 1, respectively, then the cognitive domain score is 2. These network scores provided a snapshot of the overall positive and negative network activation within participants at each session.

The cognitive emotional processing variable was used to identify peaks in cognitive emotional processing, marked by a score of three, reflecting significant moments of insight in the narratives. A narrative that warrants a score of 3 is described in the CHANGE coding manual as follows: “Engaged and exploring or confronting a problem area with substantial insight and perspective shifts. This can include making new meaning of experience, integrating past experience with current functioning, benefit finding, reframing, reaching a higher level of abstraction, and resolution/acceptance” (Hayes, 2015).

Controlling for Site Variability in Depression Treatment Outcomes

Given that the treatment occurred at two distinct sites, using different depression measures which were subsequently standardized through conversion, it was crucial to rule out the influence of location on treatment outcomes. Regression analyses were conducted to determine any potential correlation between treatment location and both initial depression scores and overall depression improvement. These analyses revealed no significant relationship between treatment location and either starting depression levels or the extent of depression improvement, indicating that treatment location and the conversion of depression scores did not influence the outcomes. Regression coefficients, 95% confidence intervals, and p-values for these analyses are presented in Table 1.

Analysis - Research Question 1

To address the first research question regarding the changes in positive and negative network strength among improvers following the Network Destabilization and Transition (NDT) model, a descriptive analysis was conducted via visual inspection of time-series plots. Participants were ranked based on their IDS improvement scores and divided into quartiles, allowing for the comparative analysis of changes across different levels of improvement. Time series plots were generated for each participant to visually represent the changes in network activation throughout therapy.

The plots for each quartile of improvers were visually inspected. This inspection aimed to determine if there was a discernible pattern indicating that the negative network weakened during the first phase of treatment (as might be indicated by a visual downward trend in activation scores across the phase-one sessions, or a diminishing frequency of high activation sessions) and that the positive network strengthened during the final phase of treatment (as might be indicated by an upward trend in activation scores during phase 3), in alignment with the NDT model. Each participant's plot provided a comprehensive overview of the treatment progression with relation to network activation.

Additionally, a group-level regression analysis was performed to look for statistical support of the titular X characteristic in improvement trajectories. A variable of overall shift in network strength was regressed against depression improvement, with starting depression as a control variable. This analysis aimed to determine if the total change in network strength from negative to positive independently predicted depression improvement.

This overall shift in network strength was operationalized by calculating the within-person average network activation scores for the positive and negative networks separately in phase 1 and phase 3. The measure of change in the strength of the networks was computed by adding the increase in the mean positive network activation from phase 1 to phase 3 to the decrease in the mean negative network activation from phase 1 to phase 3. This calculation produced a single variable representing the total shift in relative network strength from negative to positive across the treatment phases.

Analysis - Research Question 2

To explore whether peak cognitive-emotional processing during the second phase of treatment predicts outcomes for depression, a linear regression analysis was conducted. The dependent variable in this analysis was the IDS improvement score, as calculated above, representing the overall change in depression severity over the treatment period.

The key predictor variable was the presence of a peak in cognitive-emotional processing during the second phase of treatment. This variable was operationalized as binary, where '1' indicated the occurrence of at least one peak cognitive-emotional processing event (score of 3 on the CHANGE coding system) during phase 2, and '0' represented no such peak events. Additionally, to account for baseline differences in depression severity, initial depression, measured by the IDS score at the start of treatment, was included in the model as a covariate.

To broaden the investigation of cognitive-emotional processing's impact on depression outcomes, a supplementary analysis was conducted using a similar linear regression model. In this alternate model, the presence of peak cognitive-emotional processing events at any point during the entire course of EBCT treatment was evaluated as a binary predictor variable. This comparative analysis aimed to discern whether the timing of peak cognitive-emotional processing events—whether restricted to the second phase or occurring at any time during treatment—differentially predicted the improvement in depression symptoms, thus providing a deeper understanding of the dynamics of therapeutic change within the NDT model.

Analysis - Research Question 3

To investigate whether there is an interaction effect between peak cognitive-emotional processing and the reversal of attractor state strength, the study employed a linear regression model similar to that used in the second research question but extended it to include an interaction term. The dependent variable in this model remained the IDS improvement score.

The key addition to this model was the interaction term between peak cognitive-emotional processing during the second phase of treatment and the total shift in network strength from negative to positive, as described above. The interaction variable explored how the relationship between peak cognitive-emotional processing in phase 2 and treatment outcomes was moderated by the magnitude of the shift in network strengths. This analysis aimed to elucidate whether the timing and intensity of cognitive-emotional processing in conjunction with changes in the psychological network states contribute synergistically to the treatment outcomes. As in previous analyses, the initial depression score was included as a control variable to adjust for baseline severity and ensure that the observed effects were not confounded by initial differences in depression levels.

Additional Exploratory Analyses

In addition to the primary analyses addressing the specific research questions, several exploratory analyses were conducted to further investigate the dynamics of network activation and their relationship with treatment outcomes. These exploratory analyses aimed to uncover additional patterns and associations that could inform future hypotheses and therapeutic interventions.

A correlation matrix analysis was conducted to compare the average activation scores for both the positive and negative networks in each phase of treatment against improvement scores and the occurrence of cognitive emotional processing peaks. Logistic regressions were conducted using each of these variables, as well as baseline depression, as predictor variables for the presence of cognitive emotional processing peaks. This was done to explore the factors that contribute to the presence of cognitive emotional processing peaks, which are hypothesized to be critical for therapeutic change.

Additionally, the frequency with which the positive and negative networks swapped dominance (i.e., which network had a higher activation score) was calculated for each participant throughout the treatment. The total number of dominance swaps was divided by the number of sessions per participant to create the cross-rate variable. This was used as a predictor variable in a multiple regression analysis with initial depression and cognitive emotional processing peaks as covariates and improvement in depression scores as the outcome variable.

Results

Descriptives

Table 2 shows descriptive statistics for depression scores (pre-, post-, and improvement), session count, and by-phase network activation among participants included in the study. Of the 75 participants included in the study, 21 participants (28%) experienced a peak in cognitive-emotional processing at some point during their treatment. Specifically, 7 participants (9.3%) had peaks during phase 1, 10 participants (13.3%) during phase 2, and 13 participants (17.3%)

during phase 3. These numbers add up to more than the total number of peaks because some participants experienced peaks in multiple phases of their treatment. For example, 7 of the 13 participants who experienced a peak during phase 3 had already experienced one earlier in the treatment.

A key metric we are using in assessing the effectiveness of depression treatments is the minimally clinically significant change, which has been identified as a 10-point reduction in IDS scores (Trivedi et al., 2004). In this study, the mean improvement score was 22.1, with a standard deviation of 10.5 (Table 2). Thus, even a one standard deviation decrease from the mean improvement score remains above the threshold for clinical significance. Using this metric, over 85% of the study participants experienced clinically significant improvements in their depression scores.

Research Question 1 - Time-Series Plots

The time-series plots of network activation scores were visually inspected to identify trends in positive and negative network strength among participants. Example plots are provided in Figure 1. Changes in background color distinguish the three phases of EBCT treatment. The horizontal axis of each plot represents the session number, ranging from 1 to 33, which varied across participants. The vertical axis depicts the network activation score, ranging from 0 to 9. In these plots, positive network activation is illustrated in blue, and negative network activation in red. Additionally, the plots feature vertical dotted lines at sessions where peak cognitive-emotional processing events occurred, as identified by a score of 3 on this variable in the CHANGE coding system. This visual marker facilitates the investigation of the effect of high-intensity cognitive-emotional processing on network activation scores.

While the analysis aimed to discern patterns aligning with the Network Destabilization and Transition (NDT) model, several challenges emerged. In general, clear trends were difficult to identify due to significant within-person variability during each phase as well as throughout the entire treatment period. Although a few unique cases displayed consistent trends, these were not distinguishable across the quartiles. As such, rather than including plots for all quartiles, Figure 1 shows all plots of participants that experienced cognitive-emotional processing peaks during phase 2 of EBCT treatment along with their depression score improvement rank (1 being the most improved). Notably, linear patterns were rare, with only a small subset, like participant 37 (Figure 1), showing a clear linear increase in positive network activation. Most participants demonstrated non-linear and highly variable network activation dynamics, indicating that a linear model would not effectively capture these patterns. With the exception of a rare clear X pattern (Figure 1, participant 13), most plots exhibited several Xs recurring throughout the treatment period (Figure 1, participants 10, 16, 31, 37, 38). This abundance of Xs inspired the exploratory cross-rate analysis, the results of which are detailed at the end of this section.

Furthermore, regression analysis showed that the total shift in network strength from negative to positive was not a significant predictor of depression improvement in this model or any others tested (Table 4, Model 4). This suggests that the overall change in network activation did not influence depression improvement scores. We subsequently examined mean scores by phase (Table 2), paired t-tests revealed a statistically significant decrease in negative network activation from phase 1 (M = 4.1, SD = 1.9) to phase 3 (M = 2.9, SD = 1.9), $t(74) = 6.60$, $p < .001$. Conversely, a much more modest but also statistically significant increase in positive network activation occurred from phase 1 (M = 2.4, SD = 1.3) to phase 3 (M = 2.8, SD = 1.7), $t(74) = -2.65$, $p < .01$. This provides statistical evidence that, at least at the group level, the lines for positive and negative network activation intersect during phase 2, creating the hypothesized X pattern.

Research Question 2 - Phase 2 Peaks

The first regression analysis explored the impact of cognitive-emotional processing peaks during the second treatment phase on depression improvement scores, controlling for initial depression severity. Results from all regression models are presented in Table 4.

The initial model only included the starting depression score as a predictor (Table 4, Model 1). Starting depression, as expected, was a significant predictor of improvement. Specifically, for each unit increase in the initial depression score, there was an estimated improvement increase of 0.68 in the IDS change score ($p < 0.001$). The model explained approximately 31.8% of the variance in depression improvement scores.

Expanding the model to include the presence of a peak in cognitive-emotional processing during phase 2, alongside the initial depression score, indicated that the presence of a phase 2 peak was significantly associated with greater improvements in depression scores (Table 4, Model 2). Participants who experienced a peak in cognitive-emotional processing during phase 2 exhibited an average increase of 10.1 points in their depression improvement scores compared to those who did not have such a peak ($p < 0.001$). Furthermore, the impact of the initial depression score remained significant, with a slightly reduced coefficient of 0.61 ($p < 0.001$). The addition of the cognitive-emotional processing peak variable improved the model's explanatory power, with the adjusted R-squared increasing by 0.09, accounting for approximately 41% of the variance in the depression improvement scores, indicating a substantial contribution of the phase 2 peak to the overall treatment outcomes. The overall model significance was reinforced by an F-statistic of 26.75 ($p < 0.001$).

Further analysis investigated the effects of having any peak in cognitive-emotional processing during the entire course of treatment, as opposed to specifically during phase 2 (Table 4, Model 3). This model also controlled for initial depression scores. Results indicated that the occurrence of any peak in cognitive-emotional processing was significantly associated with improvements in depression scores. Participants experiencing at least one peak cognitive-emotional processing event during treatment showed an average increase of 5.4 points in their depression improvement scores compared to those without such peaks ($p = 0.019$). This model explained approximately 36% of the variance in depression improvement scores.

The comparative analysis between the impacts of cognitive-emotional processing peaks specifically during phase 2 versus peaks at any time during the treatment revealed distinct outcomes. While both types of peaks were significantly associated with improvements in depression scores, the results indicated a more pronounced effect for peaks occurring specifically in phase 2. The model including only phase 2 peaks explaining 5% more variance in depression improvement scores.

Research Question 3 - Interaction Effect Analysis

This analysis assessed an interaction effect between peak cognitive-emotional processing during the second phase of treatment and the total shift in network strength from negative to positive on depression improvement scores. This model also incorporated the initial depression score as a control variable to adjust for baseline severity.

The regression results showed that the interaction term between the phase 2 peak and the total shift in network strength was not significant (Table 4, Model 5). This indicates that the hypothesis that interaction between the timing of cognitive-emotional peaks and the magnitude of changes in network strength have a synergistic effect on treatment outcomes was not supported.

Ad Hoc Analyses - Average Network Activation By-Phase

Further exploratory analysis was conducted following the unexpected results from the initial interaction model. This analysis focused on the sub-components of the total network strength shift variable: participants' average network activation scores per-phase. A correlation table (Table 3) highlights connections between these phase-specific network activation means and depression scores. Of the phase-specific network activation scores, only phase 1 ($r = 0.31$, $p < 0.001$) and phase 3 ($r = 0.26$, $p < 0.01$) positive network activation were significantly correlated with depression improvement. This finding indicates that higher levels of positive network activation at the beginning and end of treatment are predictive of greater improvements in depression scores.

Additionally, a logistic regression analysis was performed to assess the predictive power of phase 1 positive network activation on the odds of experiencing a cognitive-emotional processing peak during treatment. The results were significant ($z = 3.95$, $p < 0.001$), suggesting that higher initial positive network activation increases the odds of having a peak in cognitive-emotional processing ($OR = 3.27$, *95% CI* [1.93, 6.36]). This relationship underscores the potential role of positive network activation in facilitating critical moments of cognitive and emotional processing that are crucial for effective therapy.

However, when these factors—phase 1 positive network activation and the occurrence of cognitive-emotional processing peaks—were included in a comprehensive model predicting depression outcomes, alongside initial depression severity, only the initial depression score remained a significant predictor. This implies that early positive activation and the occurrence of cognitive-emotional processing peaks may be explaining overlapping variance in depression outcomes, despite being apparently different constructs. Alternatively, it may indicate that both variables are substantially related to initial depression severity.

A key concern in this study was the potential redundancy between network activation and traditional measures of depression symptoms, specifically whether changes in network activation merely mirrored changes in depressive symptoms. However, the correlation matrix analysis revealed that decreases in negative network activation were not significantly correlated with improvements in depression symptoms (Table 3). Similarly, increases in positive network activation over the course of treatment did not correlate negatively with these symptoms. Additionally, positive and negative network activation in the first phase of treatment showed no significant correlation with initial depression symptoms (Table 3). These findings confirm that positive and negative network activations are distinct from standard depression measures, validating their use in our analyses. Moreover, this underscores the importance of further investigating the relationship between early positive network activation and treatment outcomes, highlighting it as a promising area for future research.

Additionally, there was a significant negative correlation between the difference in positive network activation from phase 1 to phase 3 and the difference in negative network activation from phase 1 to phase 3 (Table 3: $r = -0.34$, $p < .01$). In other words, the more the positive network activation increased, the more the negative network activation decreased.

Ad Hoc Analyses - Changes In Network Dominance

The final analysis focused on changes in network dominance, as measured by the network cross-rate variable. While controlling for initial depression scores, the cross rate was found to significantly correlate with depression improvement scores, demonstrating an individual effect, within the total explained variable of the model, comparable to that of having a cognitive-emotional processing peak in phase 2 (Table 4, Model 6). However, the interaction between cross rate and phase 2 peaks was found to be non-significant.

When cross rate was included in the primary predictive model alongside phase 2 peaks and starting depression scores, the adjusted R-squared value of the model increased to 0.50 (Table 4, Model 7). The inclusion of cross rate in the model accounted for an additional 9% of the variance in depression improvement scores, beyond the 9% explained by the presence of phase 2 peaks. This suggests that cross rate and phase 2 peaks capture unique variance within the model, offering complementary insights into the underlying dynamics of network changes and their distinct contributions to therapeutic improvement.

Furthermore, the cross rate was not significantly correlated with either the presence of phase 2 peaks or starting depression scores, indicating that network dominance changes occur independently of these variables.

Table 1

Regression Analysis of Treatment Location Impact on Depression Scores

Variable	Coefficient	95% CI	p-value
Initial Depression Score	-1.14	[-5.589, 3.305]	0.610
Depression Improvement	-2.49	[-7.740, 2.753]	0.347
Final Depression Score	1.35	[-3.197, 5.901]	0.555

Table 2

Descriptive Statistics of Study Variables

Variable	Mean	SD	Min	Max	Range
Initial Depression Score	37.3	9.2	18.0	66.0	48.0
Depression Improvement	22.6	10.8	-4.5	58.5	63.0
Final Depression Score	14.6	9.4	0.0	54.5	54.5
Session Count	24.8	4.4	14.0	32.0	18.0
Cross Rate	0.4	0.1	0	0.7	0.7
Positive Network Activation: Phase 1 Mean	2.4	1.3	0.4	6.1	5.7
Positive Network Activation: Phase 2 Mean	2.3	1.3	0.2	6.2	6.0
Positive Network Activation: Phase 3 Mean	2.8	1.7	0.2	8.2	8.0
Positive Network Activation: Phase 1 to Phase 3 Difference	0.4	1.4	-1.8	4.1	5.9
Negative Network Activation: Phase 1 Mean	4.1	1.9	0.7	8.3	7.6
Negative Network Activation: Phase 2 Mean	3.6	1.8	0.0	7.9	7.9
Negative Network Activation: Phase 3 Mean	2.9	1.9	0.0	8.6	8.6
Negative Network Activation: Phase 1 to Phase 3 Difference	-1.2	1.6	-5.2	2.9	8.0
Overall Network Activation Shift	1.6	2.4	-3.4	8.8	12.2

Table 3

Correlation Table: Variables are the same as in Table 2 but their names were shortened for the sake of formatting.

Variable	IDS-Pre	IDS Change	IDS-Post	Session Count	Cross Rate	+Net1	+Net2	+Net3	+Net Diff	-Net1	-Net2	-Net3	-Net Diff	Net Shift
IDS-Pre		0.57***	0.31**	-0.04	-0.11	0.10	-0.01	0.07	0.00	0.13	0.25*	0.19	0.08	-0.05
IDS Change	0.57***		-0.60***	-0.34**	0.26*	0.31**	0.20	0.26*	0.04	-0.13	-0.12	-0.13	0.01	0.02
IDS-Post	0.31**	-0.60***		0.36**	-0.41***	-0.26*	-0.24*	-0.23*	-0.05	0.28*	0.38***	0.34**	0.07	-0.07
Session Count	-0.04	-0.34**	0.36**		-0.21	-0.15	-0.24*	-0.20	-0.11	0.04	0.23*	0.18	0.17	-0.17
Cross Rate	-0.11	0.26*	-0.41***	-0.21		0.20	0.17	0.11	-0.05	-0.49***	-0.55***	-0.43***	0.05	-0.06
+Net1	0.10	0.31**	-0.26*	-0.15	0.20		0.64***	0.63***	-0.14	0.14	0.19	0.14	0.00	-0.08
+Net2	-0.01	0.20	-0.24*	-0.24*	0.17	0.64***		0.72***	0.32**	0.24*	0.06	0.09	-0.17	0.29*
+Net3	0.07	0.26*	-0.23*	-0.20	0.11	0.63***	0.72***		0.68***	0.24*	0.24*	0.01	-0.26*	0.56***
+Net Diff	0.00	0.04	-0.05	-0.11	-0.05	-0.14	0.32**	0.68***		0.17	0.12	-0.12	-0.34**	0.78***
-Net1	0.13	-0.13	0.28*	0.04	-0.49***	0.14	0.24*	0.24*	0.17		0.74***	0.64***	-0.40***	0.36**
-Net2	0.25*	-0.12	0.38***	0.23*	-0.55***	0.19	0.06	0.24*	0.12	0.74***		0.68***	-0.06	0.08
-Net3	0.19	-0.13	0.34**	0.18	-0.43***	0.14	0.09	0.01	0.17	0.64***	0.68***		0.45***	-0.36**
-Net Diff	0.08	0.01	0.07	0.17	0.05	0.00	-0.17	-0.26*	-0.34**	-0.40***	-0.06	0.45***		-0.85***
Net Shift	-0.05	0.02	-0.07	-0.17	-0.06	-0.08	0.29*	0.56***	0.78***	0.36**	0.08	-0.36**	-0.85***	

** = p-value < 0.05, ** = p-value < 0.01, *** = p-value < 0.001*

Table 4

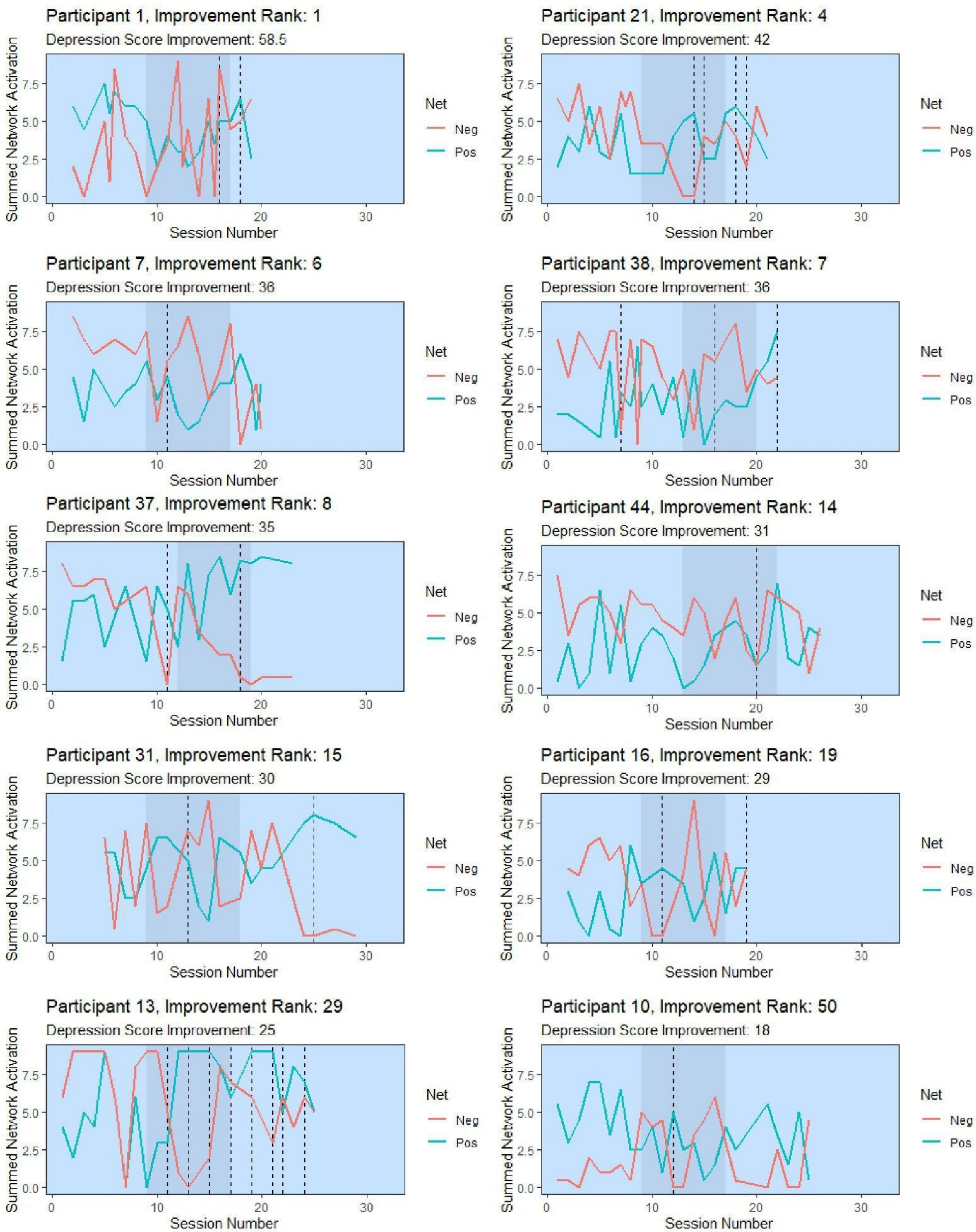
Comparison of Linear Regression Models Predicting Depression Improvement

Model	Adjusted R-squared	Predictor	Estimate	95% CI	T value	p-value
Model 1: Initial Depression	0.32	Initial Depression	0.68	[0.45, 0.90]	5.96	<0.001***
Model 2: Initial Depression + Phase 2 Peaks	0.41	Initial Depression	0.61	[0.39, 0.82]	5.64	<0.001***
		Phase 2 Peaks (TRUE)	10.13	[4.39, 15.87]	3.52	<0.001***
Model 3: Initial Depression + Processing Peaks (Any Phase)	0.36	Initial Depression	0.66	[0.44, 0.88]	6.00	<0.001***
		Any Phase Peaks (TRUE)	5.37	[0.91, 9.82]	2.40	0.019*
Model 4: Network Shift + Initial Depression	0.31	Network Shift	0.23	[-0.64, 1.09]	0.52	0.604
		Initial Depression	0.68	[0.45, 0.91]	5.95	<0.001***
Model 5: Network Shift * Phase 2 Peaks + Initial Depression (Interaction Model)	0.40	Phase 2 Peaks (TRUE)	11.57	[4.32, 18.83]	3.18	0.002**
		Network Shift	0.17	[-0.77, 1.11]	0.36	0.724
		Initial Depression	0.61	[0.39, 0.83]	5.56	<0.001***
		Phase 2 Peaks * Network Shift	-0.64	[-2.53, 1.24]	-0.68	0.499
Model 6: Cross-rate + Initial Depression	0.42	Cross-rate	25.01	[11.38, 38.63]	3.66	<0.001***
		Initial Depression	0.72	[0.51, 0.93]	6.83	<0.001***
Model 7: Cross-rate + Phase 2 Peaks + Initial Depression	0.50	Cross-rate	23.59	[10.93, 36.24]	3.72	<0.001***
		Phase 2 Peaks (TRUE)	9.52	[4.22, 14.81]	3.58	<0.001***
		Initial Depression	0.65	[0.45, 0.85]	6.54	<0.001***

** = p-value < 0.05, ** = p-value < 0.01, *** = p-value < 0.001*

Figure 1

Time-series plots of participants who experienced a phase 2 peak.



Discussion

Research Question 1: Changes in Positive and Negative Network Strength

The first research question examined whether the strength of the negative and positive networks follow the Network Destabilization and Transition (NDT) model of change. However, our visual analysis encountered a significant challenge: the lack of clearly visible trends in the data. The expectation that a clear "X" marking the spot of phase transitions would be observable in the time-series plots was not met. Instead of distinct, temporally localized crossings of network strengths during phase 2, which would indicate a straightforward shift from pathological to healthy attractor states, our data revealed a different pattern. Participant data did not exhibit a clear and unitary "X" in phase 2 but rather numerous “X”s appeared throughout the entire treatment period. This diffuse pattern of network strength changes suggests a more complex and non-linear dynamic in the therapeutic process.

The findings of our analysis of network strength changes based on within-person averages for each phase indicate that the average activation scores for both the positive and negative networks show trends consistent with the NDT model. Specifically, negative network activation tends to decrease, while positive network activation tends to increase as treatment progresses. This pattern aligns with the theoretical framework suggesting that therapy helps individuals transition from a pathological to a healthy attractor state. However, it is important to note that these averages fail to capture the high variability in individual trajectories, highlighting the need for a more nuanced understanding of these dynamics.

Research Question 2: The Effect of Cognitive Emotional Processing Peaks

The second research question explored whether peak cognitive-emotional processing during the second phase of NDT treatment predicts better outcomes for depression. The current study's results support previous research demonstrating that high levels of cognitive-emotional processing during phase 2 of EBCT leads to better outcomes for depression (Hayes et al., 2005, 2007). While this study partly uses the same data from the original studies (n = 29), the inclusion of data from an additional 46 participants provides further evidence for this claim. Furthermore, this study employed a different measurement for peaks in cognitive-emotional processing and utilized different statistical analyses. The original studies looked for a participant's personal peak (their highest score across treatment) in cognitive-emotional processing. In contrast, the current study identified peaks as narratives that received a score of 3 on cognitive-emotional processing from both raters. This was a stricter criterion for peak cognitive-emotional processing; in the original study all participants had a peak as personal peaks were used, in the current study only a subset of participants met this criterion for peak processing experiences. This approach enabled us to use regression analyses to explore between-group differences in treatment response for those who had a peak and those who did not. While the original studies used hierarchical linear models. The consistency in findings across both analytical approaches (showing that peaks in cognitive-emotional processing during phase 2 lead to better outcomes) strengthens the robustness of this conclusion.

The timing of these peaks appears to be important; with phase 2 peaks having the largest effect on depression scores. However, peaks occurring in phase 3 may be confounded by their proximity to the end of treatment, potentially delaying their measurable effects beyond the study period.

It is also important to note the rarity of cognitive-emotional processing peaks by our stricter metric. With only 28% of participants experiencing cognitive emotional processing peaks (and only 13% experiencing them during phase 2 of treatment) it cannot be said that the treatment reliably induces these experiences, nor that the timing can be strictly controlled. Furthermore, in the absence of such experiences, the majority of participants still showed significant improvement in depression scores. This suggests that while cognitive-emotional processing peaks are beneficial, they are not necessary for therapeutic progress. This underscores the idea that multiple pathways to improvement exist (Olthof et al., 2023), and that the presence of these peaks is just one of many factors contributing to successful outcomes in depression treatment.

Visual inspection of the plots (Figure 1) revealed that peak cognitive-emotional processing was often followed by increases in negative network activation. This observation suggests that peak cognitive-emotional processing could be a trigger for destabilization rather than occurring as a consequence of it. However, spikes in negative network activation were fairly common in the absence of peaks in cognitive-emotional processing as well. While this remains a fascinating line of inquiry, data with a higher level of temporal resolution would be required to further investigate the dynamic relationship between cognitive-emotional processing, destabilization, and phase transitions in the context of psychotherapeutic intervention.

Research Question 3: The Combined Effect of Network Strength Change and Phase 2 Processing Peaks

The third research question investigated the interaction effect between peak cognitive-emotional processing occurring during phase 2 of EBCT and the overall shift in positive and negative network strength. Our analysis did not support the hypothesized synergistic effect.

Specifically, the regression results indicated that the interaction term between the phase 2 peak and the total shift in network strength was not significant (Table 4, Model 5). Additionally, the shift in network strength itself was not a significant predictor of depression outcomes. These findings suggest that neither the combined interaction nor the shift in network strength alone significantly influenced treatment efficacy. In retrospect, the variable used for overall network shift was likely an oversimplification of the interdependent, dynamic interactions between the positive and negative networks. It fails to capture highly variable patterns both within and across people over the course of treatment.

Furthermore, a power analysis was conducted for this model using the "InteractionPoweR" package in R, as outlined by Baranger et al. (2023). This revealed a power of only 13%, indicating a significant limitation of the model. This extremely low power suggests a very low probability of correctly detecting an interaction effect if one exists, thereby undermining the efficacy of this hypothesis test. Consequently, it cannot be confidently stated that the hypothesis was effectively tested.

Positive Emotions and Change in Psychotherapy

Logistic regression identified phase 1 positive network activation as a predictor of phase 2 cognitive-emotional processing peaks. Participants who demonstrated higher levels of positive network activation early in treatment were more likely to experience these beneficial peaks in cognitive-emotional processing during phase 2. This finding suggests that a robust positive network may facilitate deeper, transformative cognitive-emotional work in subsequent treatment phases.

This finding aligns with Frederickson's broaden-and-build model which posits that positive emotions broaden an individual's momentary thought-action repertoire. By broadening thought-action repertoires, positive emotions encourage individuals to explore new ideas and take on challenges (Fredrickson, 1998). The resulting increase in psychological flexibility may facilitate the insight and perspective shifts in therapy that are definitive of peak cognitive-emotional processing experiences.

While the current dataset shows an increase in positive network activation over the full course of treatment, it is important to note that our phase-average measurements of network activation do not tell us whether the positive network activation *increases* during the first phase of treatment. The results of the logistic regression may simply indicate that participants who started treatment with more active positive networks were more likely to experience peak cognitive-emotional processing events. Higher initial levels of positive network activation might reflect lower levels of anhedonia. Anhedonia is a core symptom of depression characterized by a reduced ability to experience pleasure. A recent meta-analysis revealed anhedonia to be correlated with heightened symptom severity and worse treatment outcomes (Wong et al., 2024).

Network Cross-Rate and Psychological Flexibility

This study found that the network cross-rate variable, defined as the number of "X" occurrences in a participant's dataset, is positively correlated with depression improvement. The network cross-rate represents the frequency of transitions between positive and negative network dominance, indicating a continuous interplay between these states. This dynamic interaction reflects psychological flexibility, which is the ability to adaptively shift between different emotional and cognitive states. Higher cross-rate values suggest greater flexibility.

Research indicates that depression is often associated with rigidity across cognitive, behavioral, emotional, and biological domains (Hayes et al., 2022). This rigidity is characterized by reduced emotional reactivity to both positive and negative stimuli, which contributes to the persistence of depressive symptoms (Rottenberg, 2017). Conversely, psychological inflexibility has been identified as a significant risk factor for depression (Stange et al., 2017), with some researchers going as far as reconceptualizing Major Depressive Disorder as essentially being “stuck in a rut” (Holtzheimer & Mayberg, 2011). Thus, the ability to adapt to changing circumstances and shift cognitive and emotional responses plays a vital role in recovery from depression. In order to achieve a balanced state in which either the positive or negative networks can be brought to bear, the positive network must be reinforced while simultaneously addressing the negative symptoms of depression (Hayes & Andrews, 2020b).

The negative correlation between the changes in positive and negative network activations from phase 1 to phase 3 further reinforces this push-pull dynamic between the two networks, suggesting a balancing effect. Increases in positive network activation are mirrored by reductions in negative network activation. While this balance highlights the interconnectedness of emotional states, it also suggests that the two scales may be measuring overlapping variances, raising considerations about their distinctiveness in capturing unique aspects of the therapeutic process. This observation warrants careful interpretation of how changes in these networks are understood.

A significant limitation of this analysis was the exclusive focus on the overall cross-rate across the entire course of therapy for each participant. These network dynamics may occur for various reasons throughout therapy, and the current analysis does not clarify whether the treatment induced these fluctuations or if the observed psychological flexibility is an intrinsic trait of the participants, predisposing some to respond more effectively to the treatment. Future research should explore how the cross-rate variable evolves over the course of treatment and its relationship with other critical variables such as positive network activation and cognitive-emotional processing.

Limitations

This study has several limitations that need to be acknowledged. As previously stated, a primary concern is the operationalization of network activation and its potential distinction from network strength. In this dataset, network activation does not appear to represent a "stuck state" as might be expected in the context of pathological and healthy attractor states. The high within-person variation in network activation across the course of treatment, as well as the high variability observed across all levels of improvement suggests that the measure may not effectively capture the stability or rigidity theoretically associated with these states.

Another limitation is the inability to detect trends occurring at different time scales. Network activation, characterized by its high frequency and high amplitude, may mask more gradual changes and trends that develop over longer periods. This variability obfuscated the identification of clear, linear trends and hindered our ability to conduct phase-specific linear modeling. The relatively short duration of each phase of the treatment further exacerbates this issue, as it does not allow for a thorough examination of trends within each phase. Preliminary analyses indicated that linear models did not accurately represent the raw data, underscoring the complexity of the network dynamics involved.

Additionally, the openness of the journaling prompt had both benefits and drawbacks. On the positive side, the open-ended nature of the prompt allowed participants to freely express their thoughts and feelings, potentially capturing a wide range of experiences and insights. However, because the prompt was not directed toward changes in perspective or significant cognitive-emotional processing, it may have led to false negatives. In this context, a false negative means that some instances of meaningful cognitive-emotional processing were not identified or recorded because participants did not explicitly address these changes in their narratives. Consequently, this could have contributed to the rarity of peak processing experiences and biased the results of our analyses related to these peaks.

The absence of a control group in this study presents significant limitations, particularly in interpreting the high proportion of participants experiencing clinically significant improvements. With over 85% of the study participants experiencing clinically significant improvements in depression scores, the potential effectiveness of EBCT treatment is evident. However, this high rate of improvement poses challenges for comparative analysis between participants. Without a control group, attributing improvements directly to the treatment is difficult, as factors like regression to the mean, placebo effects, or natural recovery over time can't be ruled out. Moreover, the absence of comparative data makes it challenging to identify the specific impact of individual dynamics on treatment outcomes, which affects the reliability and generalizability of the findings. This limitation highlights the need to include control groups in future studies to better understand the effects of these individual dynamics throughout treatment.

Furthermore, the multiple regression analysis for models 2, 5, and 7 utilized a binary variable of participants who had phase 2 cognitive-emotional processing peaks (n = 10) and those who did not (n = 65). The disparity in the group sizes may result in heteroscedasticity, where the variance of the error terms differs between these groups. This violation of the assumption of homoscedasticity in multiple linear regression can affect the reliability and validity of the regression coefficients, potentially leading to biased estimates and reduced statistical power. A visual inspection of the residuals versus fitted values plots did not show strong signs of heteroscedasticity, although some visual indication was present. This prompted the use of the Breusch-Pagan test for each of these three models, all of which were statistically significant, confirming the presence of heteroscedasticity. Therefore, it is crucial to interpret the results of these models with caution, as the heteroscedasticity may compromise the robustness of the findings.

For future research, several avenues are suggested. Longitudinal studies incorporating idiographic system modeling techniques (Schiepek et al., 2015) could operationalize idiosyncratic positive and negative networks with a more fine-grained ecological sampling method for higher resolution data on the two networks. This could allow for the investigation of phase transitions during the course of treatment (Olthof et al., 2020). Combining such methods with the journaling techniques used in this dataset to identify the occurrence of cognitive-emotional processing peaks would result in a more comprehensive dataset for analyses using methods from complexity science. This could, in turn, result in a deeper understanding of the contributing factors and outgoing effects of cognitive-emotional processing during treatment. Moreover, exploring the relationship between cognitive-emotional processing peaks and phase transitions could further elucidate the dynamics of therapeutic change.

While this study provides valuable insights into the dynamics of network activation and cognitive-emotional processing, these limitations highlight the potential benefits of tailored measurement approaches, more fine-grained observation techniques, and the use of a control group in future research. Addressing these issues will be crucial for better understanding the intricate processes underlying therapeutic change and the efficacy of treatments based on the Network Destabilization and Transition model such as EBCT.

Conclusion

Theoretical Contributions

This study contributes to the theoretical understanding of depression treatment by reinforcing the importance of cognitive-emotional processing peaks within the Network Destabilization and Transition (NDT) model. Our findings indicate that peak cognitive-emotional processing during phase 2 of Exposure-Based Cognitive Therapy (EBCT) significantly predicts better depression outcomes, aligning with previous research (Hayes et al., 2005, 2007). Furthermore, this study's results suggest that psychological flexibility, characterized by fluid transitions between positive and negative network states (as indicated by the network cross-rate), may be an important factor in therapeutic improvement. This emphasizes the need for a dynamic approach to understanding depression, one that moves beyond static measures of positive and negative affect and considers the adaptability of emotional and cognitive responses (Rottenberg, 2017; Stange et al., 2017). This perspective aligns with complexity science and its application to psychopathology, which views mental health and illness as attributes of complex dynamic systems (Olthof et al., 2022).

Implications for Clinical Practice and Future Research

From a clinical perspective, this study provides further empirical support for the effectiveness of EBCT for treatment of depression. Additionally, it underscores the importance of promoting psychological flexibility and adaptability in therapeutic interventions for depression. Likewise, integrating positive psychology interventions early in treatment may enhance positive network activation and facilitate deeper cognitive-emotional processing. It is important to note that cognitive-emotional processing peaks, although beneficial, are relatively rare, suggesting that while these events can significantly enhance therapeutic outcomes, they are not the only path to improvement.

In conclusion, this study advances our understanding of the mechanisms of therapeutic change in EBCT for depression by emphasizing the critical role of cognitive-emotional processing peaks and psychological flexibility within the Network Destabilization and Transition (NDT) model. The current study reinforces the finding that peak cognitive-emotional processing during phase 2 of Exposure-Based Cognitive Therapy (EBCT) predicts improved depression outcomes, underscoring the importance of such transformative experiences in facilitating therapeutic progress. Additionally, the identification of the network cross-rate as a predictor of depression improvement highlights the value of psychological flexibility—reflecting the dynamic interplay between positive and negative emotional states—as a crucial component of effective therapy. These insights advocate for a nuanced approach to depression treatment, one that incorporates the principles of complexity science to view mental health as a dynamic system. Clinically, this study supports the integration of strategies that enhance positive network activation and promote psychological flexibility, thereby fostering deeper cognitive-emotional processing.

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