Using Network Analysis to Identify Processes of Change in Low-Intensity CBT Interventions for Depression and Anxiety Disorders

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Abstract

This study explores processes of change for individuals who responded to low-intensity Cognitive Behavioural Therapy (CBT) for depression, Generalised Anxiety Disorder (GAD), or panic disorder.

Routinely collected data from NHS Talking Therapies for Anxiety and Depression (TTad) services ($N = 11,396$, 69.2% female) were analysed using network analyses. Nine graphical Gaussian models (GGMs) were conducted: for each disorder, across three time phases (assessment to start-of-treatment; start to mid-point of treatment; mid-point to end-of-treatment). Each GGM included 19 nodes, based on PHQ-9, GAD-7 and NHS TTad phobia scores, using residuals as indices of change for each node.

Networks of symptom change were largely similar. Estimated network matrix similarity ranged between $r = .74$ and $r = .91$ across disorders, with depression and GAD networks more similar to each other than to panic disorder. Networks varied over time within the same disorder, more so for panic disorder ($r = .61$–$.63$) than GAD ($r = .86$–$.90$) or depression ($r = .87$–$.93$). There were close links between changes in worry-related items and feeling nervous or anxious, and between depressed mood and anhedonia across all networks, as well as links between sleep disturbance, appetite, trouble relaxing and irritability.

Findings suggest shared patterns of co-change across anxiety and depression. There is a potential indication that therapy may work by leveraging existing natural change mechanisms rather than by creating entirely new patterns of symptom interaction. Networks also show associations between symptom changes specific to certain disorders at certain points in therapy.

Introduction

Cognitive Behavioural Therapy (CBT) is an effective treatment for a range of mood and anxiety disorders (Cuijpers et al., 2023; NICE, 2011, 2022), demonstrated across numerous randomised controlled trials (e.g. Carpenter et al., 2018; Cuijpers et al., 2023; Fordham et al., 2021).

Compared to the research evidence of the efficacy of full CBT protocols, there is comparatively less evidence of *how* psychological therapy (including CBT) is effective in bringing about recovery (Cuijpers, Reijnders, & Huibers, 2019). However, in terms of transdiagnostic processes in particular, some recent studies have sought to determine overarching features that may promote reliable change during therapy: both features specific to CBT (Salkovskis et al., 2023) and therapies more broadly (Flückiger et al., 2024; Salkovskis et al., 2023; Southward et al., 2024; Wampold & Flückiger, 2023).

Recent years have seen a focus on putative transdiagnostic mechanisms in CBT (Schaeuffele et al., 2021). While transdiagnostic implementations of CBT are efficacious (Almeida & Marinho, 2021; Barlow et al., 2017; Schaeuffele et al., 2024), other approaches, which predominate in the UK National Health Service (NHS), have emphasised the importance of using CBT protocols specific to different diagnoses, such as CBT for generalised anxiety disorder, cognitive therapy for social anxiety disorder, CBT for depression, etc. (Clark, 2018; NHS Digital, 2024).

Most of the current evidence regarding the mechanisms of effective treatment is based on dismantling studies (e.g. Cuijpers, Cristea, et al., 2019; Jacobson et al., 1996; Van Oppen et al., 1995). Dismantling studies such as the ongoing DECOMPOSE project (Cristea, 2023) use specific components of a wider therapy to treat different groups of patients in order to examine the relative effects of each of these components—and as such stand to improve understanding of active ingredients within effective therapy. A useful complement to this work would be empirical research focused on precision in therapeutic mechanisms, and interactions among symptoms over the course of treatment. Symptom-level dynamics can reveal transdiagnostic processes that cut across diagnostic categories. This could help clinicians understand which symptoms to work with within a presenting problem. Improved understanding of the likely dynamic interactions among symptoms could support clinicians and services in offering targeted treatment.

One promising way to examine interactions among symptoms is through the network approach to mental disorders (Borsboom, 2017), which has shown encouraging results in its application to clinical psychology (e.g. Levinson et al., 2023; O'Driscoll et al., 2023; Schumacher et al., 2024). Applying network approaches to clinical psychology has (amongst other developments) found explanatory utility in conceptualising mental disorders as complex systems—either in contrast or complement to prevailing latent-model conceptualisations of these disorders (Borsboom & Cramer, 2013; Cramer et al., 2010a; Ebrahimi, 2023; Epskamp, 2017). In

particular, this approach has encouraged thinking of mental health symptoms interacting and influencing each other in a dynamic fashion (e.g. Borsboom, 2017; Ebrahimi et al., 2024). The dynamic and complex nature of these networks can mean, for instance, that problems can present and progress in different ways for different individuals (e.g. Ebrahimi et al., 2024), certain disorders can show comorbidity and/or bridge into others (e.g. Liu et al., 2023; Skjerdingstad et al., 2021), and that certain symptoms can be more closely linked than others—both within and across disorders (e.g. Cramer et al., 2010b).

The current study aims to build on this work by using network analyses to examine change processes across CBT protocols applied to different disorders. Distinct CBT protocols exist for the treatment of depression (Beck, 2011), generalised anxiety disorder (GAD; e.g. Robichaud and Dugas, 2006) and panic disorder (Clark, 1986; Craske, 1991). All these models comprise psychoeducation, behavioural interventions and cognitive techniques. However, it is unclear whether for instance, behavioural interventions for depression (e.g. behavioural activation) would be expected to bring about similar processes of change to behavioural interventions for panic disorder (e.g. exposure to an avoided situation).

Manualised interventions offer an excellent opportunity to investigate processes of change across depression and anxiety disorders in response to effective CBT. The majority of CBT administered by the NHS in England using stepped care models has taken the form of 'Step 2', 'low-intensity' treatment in NHS Talking Therapies for anxiety and depression (NHS TTad—formerly 'Improving Access to Psychological Therapies'; IAPT) services (Clark, 2018). Low-intensity treatments are highly manualised versions of CBT, typically offered for six treatment sessions (Gyani et al., 2013). They often proceed from psychoeducation in earlier sessions to behavioural interventions in later sessions, though this is not universally the case.

Therefore, the focus of this study is to compare similarities and differences in interrelated symptom changes in networks of three different disorders (depression; GAD; panic disorder), in three different phases of treatment (assessment to start-of-treatment; start-of-treatment to mid-treatment; mid-treatment to end-of-treatment), for people who responded to treatment in a real-world NHS setting using low-intensity CBT.

The resultant networks and their similarities or differences (either across disorders, across time, or both) will provide insight into whether different therapies for different disorders impact the interactions between symptom changes differently. Similar change processes across disorders might support the presence of transdiagnostic processes in response to CBT-informed treatments. Static network patterns across time would suggest that the way symptoms change is homogeneous across treatment. By contrast, distinct network patterns would suggest that the way that symptom relationships change can vary across the course of treatment—for example, because of different techniques used by therapists over time, or because of changes in one symptom leading to changes in others. Improving understanding of therapeutic processes in this way will aid clinicians in decisions about when and how to target symptoms during treatment.

Method

Dataset

Participants were drawn from the North and Central East London IAPT Service Improvement and Research Network (NCEL IAPT SIRN; Saunders et al., 2020). This dataset includes all adult patients who had a therapeutic intervention (two or more treatment sessions) in eight North London NHS TTad services and who had symptoms indicating a probable depressive or anxiety disorder at start of treatment. As part of routine data collection, patients completed patient health questionnaire (PHQ-9), generalised anxiety disorder 7 (GAD-7) and phobia scales at each contact with services, as well as providing gender, age, ethnicity and other demographic information at assessment. Patient information included in the dataset is shared in line with NHS data-sharing agreements for research purposes.

Inclusion Criteria

Participants were retained in the current study if they had completed a course (at least six sessions, including assessment) of individual low-intensity (Step 2) CBT treatment. NHS TTad services assign service users 'problem descriptors', characterising the agreed focus of treatment between clinician and service user in terms of ICD-10 diagnostic categories (NHS Digital, 2024; World Health Organisation, 2019). Participants were retained in the current study if they received treatment for either i) depression (NHS TTad problem descriptors: 'Depressive episode', 'Recurrent depression'), ii) GAD (NHS TTad problem descriptor: 'GAD') or iii) agoraphobia/panic disorder (NHS TTad problem descriptors: 'Agoraphobia', 'Panic Disorder'). In order to explore the characteristics of effective treatment, the current study analysed only those individuals for whom treatment was successful according to NHS TTad recovery criteria (NHS Digital, 2024). Specifically this would mean PHQ-9 and GAD-7 scores moving to below clinical thresholds (PHQ-9$<$ 10, GAD-7$<$ 8) by end of treatment. For panic disorder and agoraphobia, the Panic Disorder Severity Scale (PDSS; Shear et al., 1997) is recommended as an anxiety disorder specific measure and, when available, is used instead of the GAD-7 to determine recovery.

Analyses were conducted on sessions of CBT for individuals who completed at least six treatment sessions, inclusive of an assessment session. Participants could additionally have completed Step 2 group treatments either prior to or following individual CBT. However data was only analysed for those individuals who met clinical threshold for a given problem descriptor at assessment and and did not meet this threshold by end of treatment—indicating they had met recovery criteria following their course of individual CBT.

Measures

All network analyses were conducted on the nine items of the PHQ-9, the seven items of the GAD-7 and the three NHS TTad phobia scale items (Clark, 2018; Gyani et al., 2013).

PHQ-9 and GAD-7

The PHQ-9 and GAD-7 are well-established, reliable measures of depressive and anxious symptoms, respectively (Kroenke et al., 2001; Spitzer et al., 2006). The PHQ-9 comprises nine items evaluating emotional, psychological, and physical symptoms of depression (e.g. Feeling down, depressed, or hopeless; Trouble concentrating on things, such as reading the newspaper or watching television; Poor appetite or overeating). The GAD-7 comprises seven items relating to anxious symptoms and worry (e.g. Feeling nervous, anxious, or on edge; Not being able to stop or control worrying). All items across the two measures are self-rated on Likert-like scales for the frequency they have been experienced over the past 2 weeks: 0—Not at all, 1—Several days, 2—More than half the days, 3—Nearly every day.

NHS TTad Phobia Scales

NHS TTad phobia scales comprise three items asking individuals how much they would avoid certain situations: 1) Social situations due to a fear of being embarrassed or making a fool of myself; 2) Certain situations because of a fear of having a panic attack or other distressing symptoms; 3) Certain situations because of a fear of particular objects or activities. Items are rated on a scale from 0 (*Would not avoid it*) to 8 (*Always avoid it*).

Statistical Analyses

Statistical analyses were performed in R version 4.4.1 (R Core Team, 2024). Full details of all methods and statistical analyses are given in the preregistration at the OSF repository for this study (https://osf.io/5bxvq) and in supplementary materials.

Pre-processing

Data for each of the 19 items (nine PHQ-9 items, seven GAD-7 items and three phobia items) used for network analysis were extracted for four time-points: assessment (session 1), first treatment session, i.e. start-of-treatment (session 2), treatment mid-point (session 4—NB. see Table 1 for average number of treatment sessions in the sample) and treatment end-point (final session: specific to each individual).

Change scores between each pair of successive time-points were calculated as residuals from linear regressions. Change scores enable investigating changes in the interaction between symptoms over treatment, which in mental disorders might present as vicious cycles or self-sustaining symptoms. Previous work has indicated that using residual scores rather than arithmetic change scores produces more consistent resulting networks of change (O'Driscoll et al., 2023).

For each regression the item score at a specific time-point was the dependent variable, and the score for the same item at the immediately preceding time-point was the independent variable. For example, a 'Phase 1' (assessment to start-of-treatment) score for the first item of the GAD-7 ('GAD1') would be the residual of GAD1 at the start of treatment (i.e. the first treatment session), regressing out the GAD1 score at assessment. This was done for all 19 symptom scores to give change scores for three phases of treatment for each item: Phase 1—assessment to start-of-treatment; Phase 2—start to mid-point of treatment; Phase 3—mid-point to end-of-treatment.

All correlation matrices of residual scores (for each phase and subsample) were positive definite. The Goldbricker function in R package networktools v1.5.2 (Jones, 2024) was used to identify redundant variables using the Hittner et al. (2003) method. Some items were found to overlap in some disorders at specific time-points, but no items were found to consistently overlap (i.e. to be redundant) across subsamples. Hence no items were removed from the network analyses.

Network Models

The study estimated graphical Gaussian models (GGMs) using the R package psychonetrics v0.13 (Epskamp, 2024a) to fit network structures to the 19 symptom measures. Networks were visualised using the package qgraph v1.9.8 (Epskamp et al., 2023). Separate models were conducted for each of the three diagnostic subsamples (depression, GAD, panic disorder) at each of the three phases of treatment (assessment to start-of-treatment; start to mid-point of treatment; mid-point to end-of-treatment), giving a total of nine GGMs.

Each of the 19 symptom change scores was represented as a node, based on residual scores capturing change from one time-point to the next. Generating a network using a GGM runs a set of partial rank-order correlations among the individual questionnaire items (nodes), controlling for all other associations within the network. The resultant associations are termed 'edges', and each association between any two nodes can vary in strength ('edge weight') and can be positive or negative (Epskamp et al., 2017, 2022).

The network models were estimated as unregularised GGMs, following recommended guidelines given the large sample sizes in the current study (Isvoranu & Epskamp, 2023). To generate the most parsimonious networks possible given the model-fitting approach used by psychonetrics, all network models were run including the package's prune function (pruning at $\alpha = .01$), followed by its stepup function. Missing data

were addressed by using Full Information Maximum Likelihood (FIML) estimation (Baraldi & Enders, 2010). Measures of the strength and expected influence of specific nodes (Costantini et al., 2015; Valente, 2012) were also estimated for each network, to support the interpretation of network models. Graphs of these centrality measures are given in supplementary material.

A custom function (Epskamp, 2024b) was run to conduct bootstrap tests of robustness in the R package bootnet v1.6 (Epskamp & Fried, 2024). Non-parametric bootstrapping was conducted with 1000 iterations to estimate the stability of edge weights, and casedrop bootstrapping with 1000 iterations to estimate the stability of centrality indices, following Fried et al. (2022).

Pre-registration

This project was pre-registered (https://osf.io/5bxvq). In the pre-registration we had planned to compare the nine resultant networks using the (Individual) Network Invariance Test (INIT; Hoekstra et al., 2024). However, after discussing the applications of the (Individual) Network Invariance Test with its author, we realised that the Network Invariance Test would be inappropriate for our intended use, due to it not yet having been validated outside of comparing idiographic networks. Accordingly, we determined the similarity of our nine GGM networks by reporting correlations between the matrices of each of our networks (see Bjørndal et al., 2023; Ebrahimi et al., 2021; O'Driscoll et al., 2021). Results of the originally planned INIT are reported in supplementary materials.

Ethics

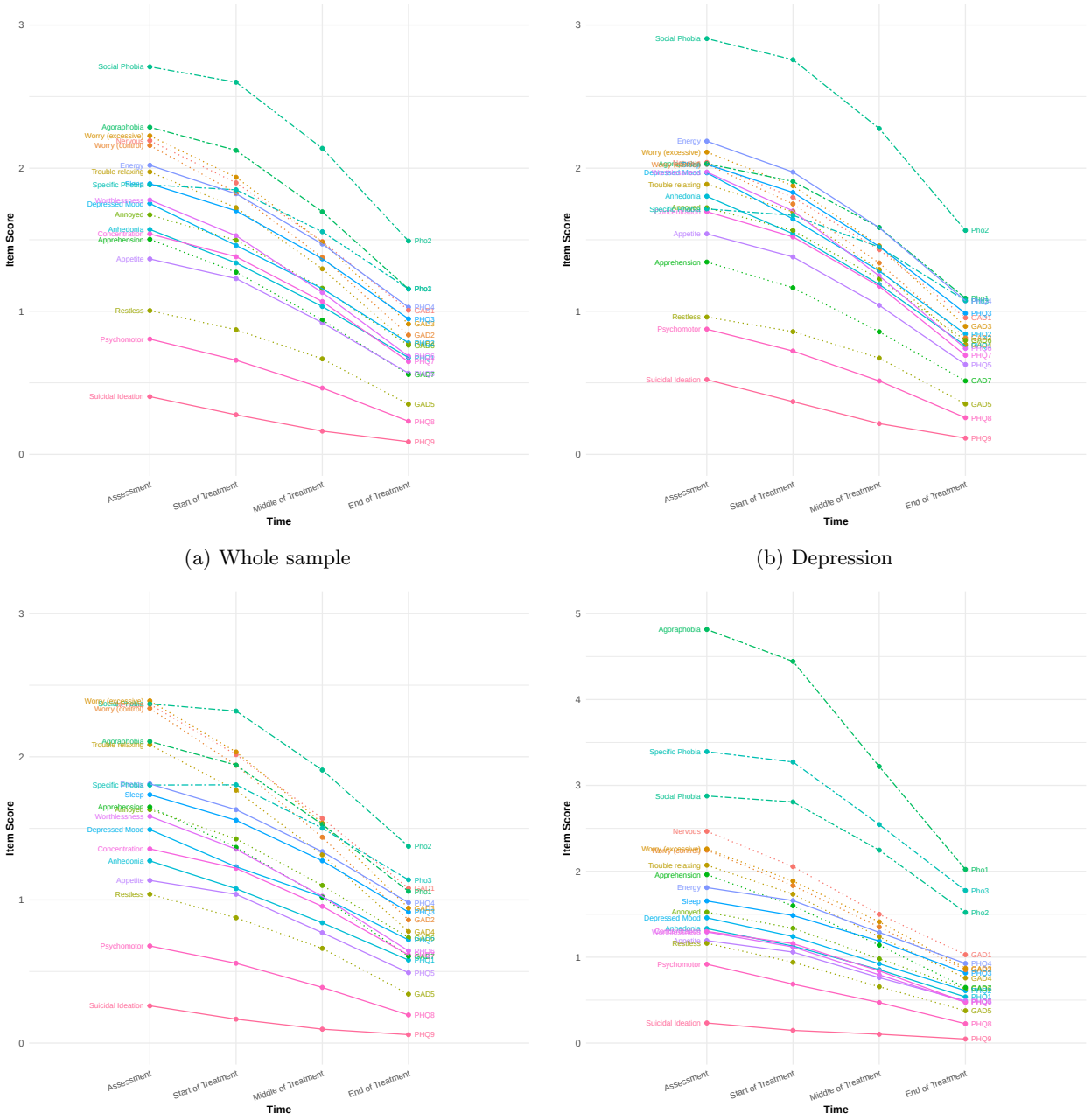
Data were made available for use under license for the purposes of this study, following the terms of the ethical approval granted to the NCEL IAPT SIRN project (Saunders et al., 2020, project reference 00519-IAPT). Raw data are not publicly available due to information that could compromise patient privacy.

Results

Demographic information is given in Table 1, along with missing data rates. The sample was mixed ethnically, with the largest ethnic grouping being 'White British' (44.3%). The sample was predominantly female (69.2%), with a mean age of 36.7 years (ranging 18–91). PHQ-9, GAD-7 and phobia scores from assessment to end-of-treatment are given in Table 2. Raw scores for each item are plotted from assessment to end of treatment in Figure 1. All scores decreased between assessment and end of treatment, with most symptoms showing steeper rates of decline as treatment progressed.

Table 1: Demographic information for the full sample, broken down by NHS TT problem descriptor (Depression, GAD, Panic disorder) and overall. IMD decile refers to the 'Index of Multiple Deprivation' scaled across the population of England, with lower deciles indicating greater deprivation.

	Depression ($n = 6,577$)	GAD ($n = 4,003$)	Panic Disorder ($n = 816$)	Overall ($N = 11,396$)
Number of Sessions				
Mean (SD)	7.09 (1.37)	6.86 (1.10)	7.05 (1.23)	7.01 (1.28)
Median [Min, Max]	7 [6, 20]	7 [6, 17]	7 [6, 16]	7 [6, 20]
No. Group Sessions				
Mean (SD)	0.09 (0.60)	0.13 (0.83)	0.12 (0.75)	0.11 (0.70)
Median [Min, Max]	0.0 [0, 11]	0.0 [0, 19]	0.0 [0, 8]	0.0 [0, 19]
Diagnosis				
Depressive episode	6046 (91.9%)	0 (0%)	0 (0%)	6046 (53.1%)
Recurrent depression	531 (8.1%)	0 (0%)	0 (0%)	531 (4.7%)
GAD	0 (0%)	4003 (100%)	0 (0%)	4003 (35.1%)
Agoraphobia	0 (0%)	0 (0%)	54 (6.6%)	54 (0.5%)
Panic disorder	0 (0%)	0 (0%)	762 (93.4%)	762 (6.7%)
Age				
Mean (SD)	37.3 (13.4)	35.9 (12.7)	35.9 (13.0)	36.7 (13.1)
Median [Min, Max]	34.0 [18.0, 91.8]	32.0 [18.0, 88.0]	32.0 [18.0, 85.0]	33.0 [18.0, 91.8]
Gender				
Not known/stated	16 (0.2%)	10 (0.2%)	5 (0.6%)	31 (0.3%)
Female	4390 (66.7%)	2963 (74.0%)	533 (65.3%)	7886 (69.2%)
Indeterminate	6 (0.1%)	1 (0.0%)	0 (0%)	7 (0.1%)
Male	2159 (32.8%)	1028 (25.7%)	278 (34.1%)	3465 (30.4%)
Nonbinary	6 (0.1%)	1 (0.0%)	0 (0%)	7 (0.1%)
IMD decile				
Mean (SD)	4.13 (2.22)	4.35 (2.28)	3.80 (2.22)	4.18 (2.24)
Median [Min, Max]	3.0 [1, 10]	4.0 [1, 10]	3.0 [1, 10]	4 [1, 10]
Missing	2712 (41.2%)	1363 (34.0%)	240 (29.4%)	4315 (37.9%)
Prescribed mental health medication				
Not known	351 (5.3%)	181 (4.5%)	45 (5.5%)	577 (5.1%)
No	4265 (64.8%)	2837 (70.9%)	441 (54.0%)	7543 (66.2%)
Yes	1961 (29.8%)	985 (24.6%)	330 (40.4%)	3276 (28.7%)
Taking mental health medication				
Not known	351 (5.3%)	181 (4.5%)	45 (5.5%)	577 (5.1%)
No	4509 (68.6%)	3010 (75.2%)	496 (60.8%)	8015 (70.3%)
Yes	1717 (26.1%)	812 (20.3%)	275 (33.7%)	2804 (24.6%)
Long-term physical health condition				
Yes	1703 (25.9%)	1053 (26.3%)	199 (24.4%)	2952 (25.9%)
Missing	1498 (22.8%)	1137 (28.4%)	201 (24.6%)	2836 (24.9%)
Ethnicity				
Not known	56 (0.9%)	45 (1.1%)	7 (0.9%)	108 (0.9%)
White British	2716 (41.3%)	1974 (49.3%)	357 (43.8%)	5047 (44.3%)
White Irish	171 (2.6%)	128 (3.2%)	22 (2.7%)	321 (2.8%)
Any other white background	1255 (19.1%)	885 (22.1%)	204 (25.0%)	2344 (20.6%)
Mixed: White and Black Caribbean	114 (1.7%)	40 (1.0%)	14 (1.7%)	168 (1.5%)
Mixed: White and Black African	57 (0.9%)	20 (0.5%)	6 (0.7%)	83 (0.7%)
Mixed: White and Asian	84 (1.3%)	51 (1.3%)	5 (0.6%)	140 (1.2%)
Any other mixed background	153 (2.3%)	95 (2.4%)	21 (2.6%)	269 (2.4%)
Indian	291 (4.4%)	132 (3.3%)	24 (2.9%)	447 (3.9%)
Pakistani	129 (2.0%)	36 (0.9%)	11 (1.3%)	176 (1.5%)
Bangladeshi	126 (1.9%)	33 (0.8%)	10 (1.2%)	169 (1.5%)
Any other Asian background	170 (2.6%)	91 (2.3%)	20 (2.5%)	281 (2.5%)
Black/Black British: Caribbean	376 (5.7%)	103 (2.6%)	33 (4.0%)	512 (4.5%)
Black/Black British: African	365 (5.5%)	117 (2.9%)	34 (4.2%)	516 (4.5%)
Any other Black background	104 (1.6%)	30 (0.7%)	4 (0.5%)	138 (1.2%)
Chinese	88 (1.3%)	49 (1.2%)	6 (0.7%)	143 (1.3%)
Any other ethnic group	218 (3.3%)	115 (2.9%)	31 (3.8%)	364 (3.2%)
Not stated	104 (1.6%)	59 (1.5%)	7 (0.9%)	170 (1.5%)



(a) Whole sample

(b) Depression

(c) GAD

(d) Panic Disorder

Figure 1: Change in symptom scores over time, given as individual items from the PHQ-9, GAD-7 and NHS TT phobia scales. Questionnaire and item number (e.g. PHQ-9 item 1, GAD-7 item 1, Phobia item 1) is given on the right of each graph, and symptom designation (e.g. low mood, anhedonia, specific phobia etc.) is given on the left. PHQ-9 items are plotted as solid lines, GAD-7 items as dotted lines, and phobia items as dotted-and-dashed lines. Separate graphs depict (a) the sample as a whole, and then individuals with a problem descriptor of (b) depression, (c) GAD, (d) panic disorder/agoraphobia.

Table 2: Scores of depressive (PHQ-9), anxious (GAD-7) and phobic symptoms at: i) assessment, ii) start of treatment, iii) mid-point of treatment (session 4), and iv) end of treatment (final recorded session). Scores are given for individuals assigned problem descriptors of depression, GAD, panic disorder, and the sample as a whole, respectively.

	Depression ($n = 6,577$)	GAD ($n = 4,003$)	Panic Disorder ($n = 816$)	Overall ($N = 11,396$)
Number of Sessions				
Mean (SD)	7.09 (1.37)	6.86 (1.10)	7.05 (1.23)	7.01 (1.28)
Median [Min, Max]	7.0 [6, 20]	7.0 [6, 17]	7.0 [6, 16]	7.0 [6, 20]
PHQ-9 Assessment				
Mean (SD)	14.6 (4.66)	11.3 (4.96)	11.2 (5.68)	13.1 (5.12)
Median [Min, Max]	14.0 [0, 27]	11.0 [0, 27]	11.0 [0, 27]	13.0 [0, 27]
Missing	2131 (32.4%)	1036 (25.9%)	218 (26.7%)	3385 (29.7%)
PHQ-9 Start of Treatment				
Mean (SD)	12.7 (5.08)	9.84 (4.93)	9.68 (5.37)	11.4 (5.25)
Median [Min, Max]	12.0 [0, 27]	9.0 [0, 27]	9.0 [0, 25]	11.0 [0, 27]
Missing	1736 (26.4%)	718 (17.9%)	159 (19.5%)	2613 (22.9%)
PHQ-9 Middle of Treatment				
Mean (SD)	9.69 (4.92)	7.71 (4.48)	7.23 (4.95)	8.77 (4.87)
Median [Min, Max]	9.0 [0, 27]	7.0 [0, 27]	6.0 [0, 25]	8.0 [0, 27]
Missing	1531 (23.3%)	594 (14.8%)	137 (16.8%)	2262 (19.8%)
PHQ-9 End of Treatment				
Mean (SD)	6.07 (4.10)	5.20 (3.58)	4.61 (3.95)	5.65 (3.94)
Median [Min, Max]	6.0 [0, 27]	5.0 [0, 24]	4.0 [0, 24]	5.0 [0, 27]
Missing	1613 (24.5%)	695 (17.4%)	160 (19.6%)	2468 (21.7%)
GAD-7 Assessment				
Mean (SD)	12.10 (4.46)	13.5 (3.74)	13.7 (3.89)	12.7 (4.22)
Median [Min, Max]	12.0 [0, 21]	13.0 [1, 21]	14.0 [0, 21]	13.0 [0, 21]
Missing	2187 (33.3%)	1079 (27.0%)	223 (27.3%)	3489 (30.6%)
GAD-7 Start of Treatment				
Mean (SD)	10.7 (4.80)	11.4 (4.58)	11.4 (4.58)	11.0 (4.72)
Median [Min, Max]	10.0 [0, 21]	11.0 [0, 21]	11.00 [0, 21]	11.0 [0, 21]
Missing	1756 (26.7%)	727 (18.2%)	155 (19.0%)	2638 (23.1%)
GAD-7 Middle of Treatment				
Mean (SD)	8.27 (4.51)	8.64 (4.32)	8.28 (4.60)	8.41 (4.45)
Median [Min, Max]	8.0 [0, 21]	8.0 [0, 21]	8.0 [0, 21]	8.0 [0, 21]
Missing	1555 (23.6%)	605 (15.1%)	138 (16.9%)	2298 (20.2%)
GAD-7 End of Treatment				
Mean (SD)	5.09 (3.55)	5.35 (3.46)	5.17 (3.82)	5.19 (3.54)
Median [Min, Max]	5.0 [0, 21]	5.0 [0, 21]	5.0 [0, 21]	5.0 [0, 21]
Missing	1633 (24.8%)	699 (17.5%)	155 (19.0%)	2487 (21.8%)
Agoraphobia Assessment				
Mean (SD)	2.03 (2.41)	2.11 (2.35)	4.82 (2.46)	2.29 (2.51)
Median [Min, Max]	1.0 [0, 8]	2.0 [0, 8.00]	5.0 [0, 8]	2.0 [0, 8]
Missing	1811 (27.5%)	881 (22.0%)	115 (14.0%)	2807 (24.6%)
Agoraphobia Start of Treatment				
Mean (SD)	1.91 (2.31)	1.94 (2.22)	4.44 (2.45)	2.12 (2.39)
Median [Min, Max]	1.0 [0, 8]	1.0 [0, 8]	4.0 [0, 8]	2.0 [0, 8]
Missing	1780 (27.1%)	812 (20.3%)	115 (14.1%)	2707 (23.8%)
Agoraphobia Middle of Treatment				
Mean (SD)	1.59 (2.05)	1.53 (1.93)	3.22 (2.33)	1.70 (2.08)
Median [Min, Max]	1.0 [0, 8]	1.0 [0, 8]	3.0 [0, 8]	1.0 [0, 8]
Missing	1813 (27.6%)	845 (21.1%)	127 (15.6%)	2785 (24.4%)
Agoraphobia End of Treatment				
Mean (SD)	1.09 (1.69)	1.06 (1.59)	2.02 (2.04)	1.16 (1.70)
Median [Min, Max]	0.0 [0, 8]	0.0 [0, 8]	2.0 [0, 8]	0.0 [0, 8]
Missing	1905 (29.0%)	885 (22.1%)	128 (15.7%)	2918 (25.6%)
Social Phobia Assessment				
Mean (SD)	2.90 (2.39)	2.37 (2.15)	2.88 (2.42)	2.71 (2.32)
Median [Min, Max]	2.0 [0, 8]	2.0 [0, 8]	2.0 [0, 8]	2.0 [0, 8]
Missing	1810 (27.5%)	882 (22.0%)	116 (14.2%)	2808 (24.6%)
Social Phobia Start of Treatment				
Mean (SD)	2.76 (2.25)	2.32 (2.08)	2.81 (2.40)	2.60 (2.21)
Median [Min, Max]	2.0 [0, 8]	2.0 [0, 8]	2.0 [0, 8]	2.0 [0, 8]
Missing	1778 (27.0%)	813 (20.3%)	115 (14.1%)	2706 (23.7%)
Social Phobia Middle of Treatment				

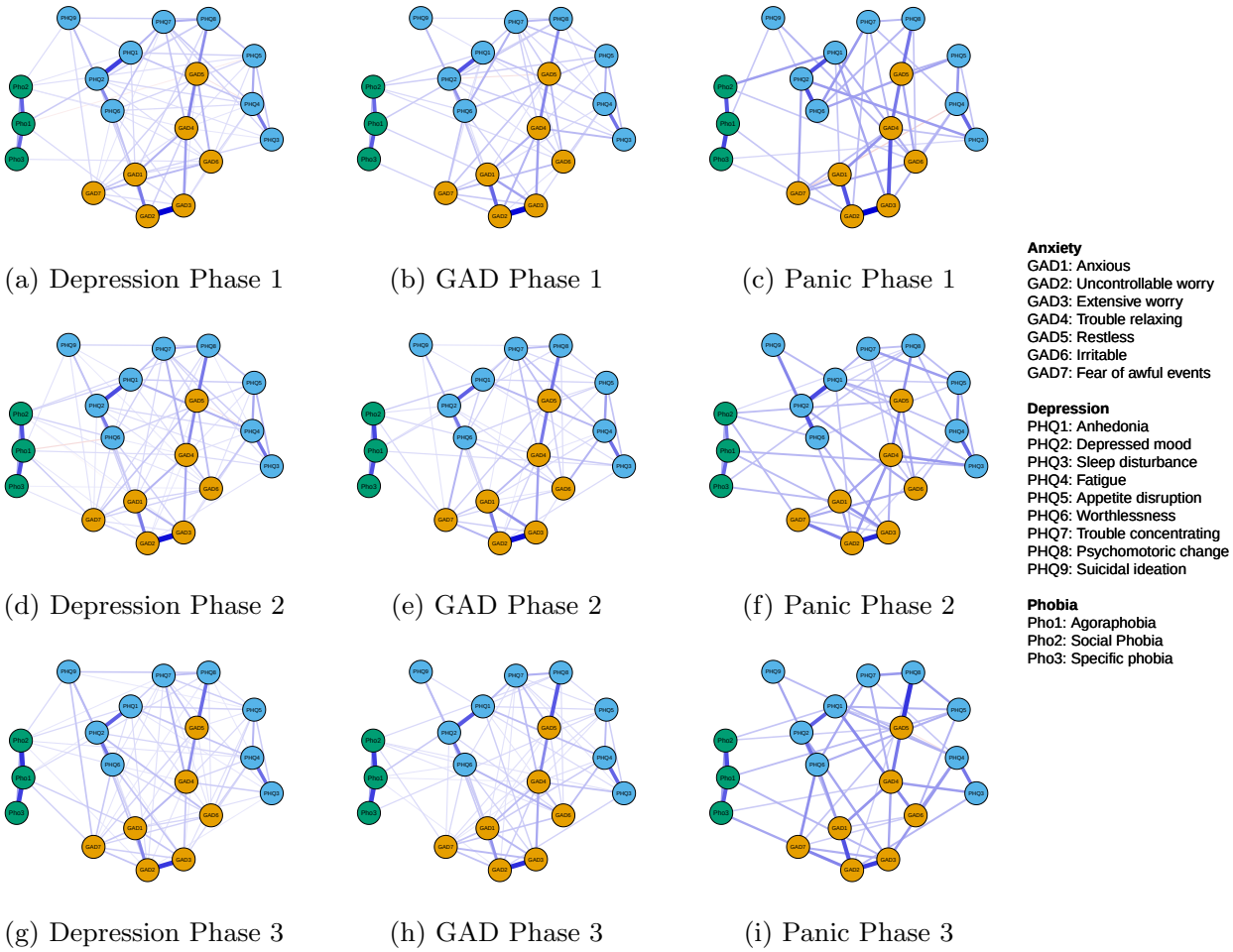
Mean (SD)	2.28 (2.05)	1.91 (1.84)	2.25 (2.03)	2.14 (1.98)
Median [Min, Max]	2.0 [0, 8]	2.0 [0, 8]	2.0 [0, 8]	2.0 [0, 8]
Missing	1810 (27.5%)	842 (21.0%)	125 (15.3%)	2777 (24.4%)
Social Phobia End of Treatment				
Mean (SD)	1.57 (1.75)	1.37 (1.58)	1.52 (1.71)	1.49 (1.69)
Median [Min, Max]	1.0 [0, 8]	1.0 [0, 8]	1.0 [0, 8]	1.0 [0, 8]
Missing	1905 (29.0%)	884 (22.1%)	128 (15.7%)	2917 (25.6%)
Specific Phobia Assessment				
Mean (SD)	1.72 (2.39)	1.80 (2.35)	3.39 (2.89)	1.88 (2.46)
Median [Min, Max]	0 [0, 8.0]	1.0 [0, 8]	3.0 [0, 8]	1.0 [0, 8]
Missing	1810 (27.5%)	882 (22.0%)	116 (14.2%)	2808 (24.6%)
Specific Phobia Start of Treatment				
Mean (SD)	1.67 (2.28)	1.80 (2.32)	3.27 (2.76)	1.85 (2.37)
Median [Min, Max]	0.0 [0, 8]	1.0 [0, 8]	3.0 [0, 8]	1.0 [0, 8]
Missing	1782 (27.1%)	812 (20.3%)	116 (14.2%)	2710 (23.8%)
Specific Phobia Middle of Treatment				
Mean (SD)	1.45 (2.10)	1.50 (2.10)	2.54 (2.48)	1.56 (2.15)
Median [Min, Max]	0.0 [0, 8]	0.0 [0, 8]	2.0 [0, 8]	0.0 [0, 8]
Missing	1814 (27.6%)	844 (21.1%)	125 (15.3%)	2783 (24.4%)
Specific Phobia End of Treatment				
Mean (SD)	1.07 (1.82)	1.14 (1.78)	1.78 (2.17)	1.16 (1.85)
Median [Min, Max]	0.0 [0, 8]	0.0 [0, 8]	1.0 [0, 8]	0.0 [0, 8]
Missing	1905 (29.0%)	885 (22.1%)	128 (15.7%)	2918 (25.6%)

Nine GGMs were estimated and plotted in Figure 2. The smallest and largest edge weights for each network are given in Table 3, and mean edge weights and network densities in Table 4. As all reported networks were estimated on change scores (rather than raw symptom scores, as in more common in network analyses), it is worth noting that edge weights do not simply represent how strongly one symptom (e.g. feelings of anxiety) is related to another (e.g. worrying); in each case they show how strongly changes in one symptom (e.g. feelings of anxiety) are related to changes in another (e.g. worrying).

Table 3: Smallest and largest (absolute) edge weights for each of the GGM networks

		Smallest Edge Weight	Largest Edge Weight
Phase 1	Depression	-.032	.458
	GAD	.053	.443
	Panic Disorder	.075	.465
Phase 2	Depression	-.012	.436
	GAD	.042	.437
	Panic Disorder	.070	.384
Phase 3	Depression	.038	.367
	GAD	.041	.379
	Panic Disorder	-.053	.364

All networks showed stable connections as indicated by correlation-stability (CS) coefficients (Epskamp et al., 2018), as shown in Table 4. The CS coefficients for both edges and node centrality strength reached .75 in the depression and GAD networks, indicating excellent stability. CS coefficients were lower in the three panic disorder networks for both edges (Phase 1 = .59, Phase 2 = .36, Phase 3 = .44) and node centrality strength (Phase 1 = .36, Phase 2 = .53, Phase 3 = .44), but in all cases remained above the recommended .25 threshold (Fried et al., 2022).



(a) Depression Phase 1 (b) GAD Phase 1 (c) Panic Phase 1

(d) Depression Phase 2 (e) GAD Phase 2 (f) Panic Phase 2

(g) Depression Phase 3 (h) GAD Phase 3 (i) Panic Phase 3

Figure 2: Network graphs of GGMs run in the psychonetrics R package, with pruning at $\alpha = .01$ and stepup. Graphs depict change scores (residuals resulting from regressing out the same score a the previous time point) separately for groups of individuals with each disorder, at three different phases: Phase 1 (from assessment to start of treatment); Phase 2 (from start of treatment to mid-point of treatment); Phase 3 (from mid-point of treatment to end of treatment). (a) Depression at Phase 1; (b) GAD at Phase 1; (c) Panic disorder at Phase 1; (d) Depression at Phase 2; (e) GAD at Phase 2; (f) Panic disorder at Phase 2; (g) Depression at Phase 3; (h) GAD at Phase 3; (i) Panic disorder at Phase 3.

Table 4: Bootstrapped network output for the nine network graphs

		Non-parametric Bootstrapping (1000 iterations)		Case-drop bootstrapping (1000 iterations)	
		Mean Edge Weight	Network Density (non-zero edges)	CS(core = 0.7) Edge	Strength
Phase 1	Depression	.045	74/171 (.43)	.75	.75
	GAD	.045	66/171 (.39)	.75	.75
	Panic Disorder	.044	44/171 (.26)	.59	.36
Phase 2	Depression	.047	81/171 (.47)	.75	.75
	GAD	.046	70/171 (.41)	.75	.75
	Panic Disorder	.047	51/171 (.30)	.36	.53
Phase 3	Depression	.048	83/171 (.49)	.75	.75
	GAD	.046	72/171 (.41)	.75	.75
	Panic Disorder	.047	48/171 (.28)	.44	.44

Table 5: Correlations between network matrices of different disorders, separated by different time-points: Phase 1 (Assessment–Start of Treatment); Phase 2 (Start of Treatment–Middle of Treatment); Phase 3 (Middle of Treatment–End of Treatment). 95% confidence intervals for correlation coefficients are given in brackets.

	Triage-SoT			SoT-MoT			MoT-EoT		
	Depression	GAD	Panic	Depression	GAD	Panic	Depression	GAD	Panic
Depression	1			1			1		
GAD	.88 [.86, .90]	1		.91 [.89, .92]	1		.88 [.85, .90]	1	
Panic	.76 [.71, .80]	.76 [.72, .80]	1	.74 [.69, .78]	.76 [.71, .80]	1	.73 [.68, .77]	.74 [.69, .78]	1

Network correlation coefficients are given in Tables 5 and 6. Networks of change scores were generally similar across disorders (particularly depression and GAD), and across time within each disorder.

Characterisations of the similarities and differences between networks reported below are made on the joint basis of: network correlations (Tables 5 and 6), network graphs (Figure 2), bootstrapped edge weights, and indicators of centrality of individual nodes within each network (see supplementary material).

Similarities

All networks showed strong connections between feelings of anxiety and worry (GAD2–GAD3), and between trouble relaxing and restlessness (GAD4–GAD6), with a strong edge between psychomotor change and restlessness (PHQ8–GAD5). Fear of awful events and irritability were less strongly connected to other anxious symptoms than were worry-related items and feeling anxious.

Table 6: Correlations between network matrices of different time-points, separately for different disorders. 95% confidence intervals for correlation coefficients are given in brackets.

	Depression			GAD			Panic		
	Phase 1	Phase 2	Phase 3	Phase 1	Phase 2	Phase 3	Phase 1	Phase 2	Phase 3
Phase 1	1			1			1		
Phase 2	.93 [.91, .94]	1		.89 [.87, .91]	1		.63 [.56, .68]	1	
Phase 3	.87 [.85, .94]	.89 [.87, .91]	1	.86 [.83, .88]	.90 [.88, .92]	1	.67 [.61, .73]	.61 [.54, .67]	1

Across all networks, agoraphobia, social phobia and specific phobia were strongly interrelated. All networks also showed strong associations between fatigue, appetite disruption and psychomotor change. Among depressive symptoms there were close links between anhedonia and depressed mood, and between depressed mood and worthlessness. These common edges were the strongest edges across networks (Figure 2).

Differences

The number of edges differed between networks (Table 4), with depression networks denser (.43 to .49) and panic networks notably sparser (.26 to .30), likely reflecting differences in sample sizes between disorders. The link between anhedonia and fatigue in depression and GAD was initially absent in panic disorder, but emerged over treatment. The link between anxiety and restlessness differed across disorders: absent from panic disorder, appearing in depression, and disappearing then re-emerging in GAD. There were also disorder-specific changes over the course of therapy. The most salient differential elements between disorders are described in more detail below.

Depression

Anhedonia initially had a significant edge with suicidal ideation (Phase 1 = .071), which weakened (Phase 2 = .050) and disappeared (Phase 3). Edges weakened between irritability and fear of awful events once treatment began (Phase 1 = .126, Phase 2 = .082, Phase 3 = .084). Edges also weakened between extensive worry and trouble relaxing (Phase 1 = .178, Phase 2 = .158, Phase 3 = .107) and between restlessness and irritability (Phase 1 = .135, Phase 2 = .120, Phase 3 = .052)—particularly later in treatment in the case of the latter. A weak link emerged between anxiety and restlessness between the middle and end of treatment (Phase 1 = .000; Phase 2 = .000; Phase 3 = .052).

GAD

Trouble concentrating strengthened connections over time with anhedonia (Phase 1 = .099, Phase 2 = .102, Phase 3 = .125), psychomotor change (Phase 1 = .125, Phase 2 = .123, Phase 3 = .142) and trouble relaxing (Phase 1 = .089, Phase 2 = .116, Phase 3 = .120).

Panic disorder

Specific phobia symptoms showed an edge with fear of awful events pre-treatment (.094), which then disappeared in the first phase of treatment, before re-emerging more strongly later in treatment (from .000 to .161). The edge between irritability and fear of awful events weakened latterly in treatment (Phase 1 = .149,

Phase 2 = .151, Phase 3 = .092). Other edges strengthened between pre-treatment and active treatment: between psychomotor change and appetite disruption (Phase 1 = .000, Phase 2 = .120, Phase 3 = .169), and between trouble relaxing and sleep disturbance (Phase 1 = .114, Phase 2 = .187, Phase 3 = .177).

Discussion

This study used network analyses to map interrelations between symptom change scores over the course of effective CBT. Networks were broadly similar across groups receiving CBT for depression, GAD and panic disorder—particularly depression and GAD. This would seem to indicate support for substantive overlap between processes of change in successful low-intensity CBT for depression and anxiety disorders, as argued for by proponents of transdiagnostic CBT mechanisms (Ellard et al., 2010; Salkovskis et al., 2023; Schaeuffele et al., 2021), and as indicated by systematic reviews (Schaeuffele et al., 2024). Though change networks were also broadly similar from assessment to end of treatment (albeit less so for panic disorder), there was also evidence of disorder-specific change processes over time.

Network Structures

Similarities

In all change score networks, we observed strong connections between anxiety-related symptoms and mechanisms (anxiety, extensive worry, and difficulty controlling worry) and between core depressive symptoms (depressed mood and anhedonia). These findings align with O'Driscoll et al. (2022), but may also be explained in part by the well-documented comorbidity of depression and GAD and the difficulty in differentiating these diagnoses (Noyes, 2001; ter Meulen et al., 2021). The interconnected worry components visible in our networks mirror the 'vicious cycles' described in CBT models of GAD (Robichaud & Dugas, 2006) and theories positioning worry as a key maintaining factor in anxiety disorders (Wells, 1995). Pending support in longitudinal studies, these findings suggest addressing these interconnected processes may be equally important in recovery from depression and panic disorder (Beck, 2011).

Across all disorder networks, we found robust connections between sleep difficulties, appetite changes, trouble relaxing, and irritability, again consistent with O'Driscoll et al. (2022). The particularly strong edge between psychomotor changes and restlessness replicates previous network findings of this link bridging anxious and depressive symptoms (e.g. Chen et al., 2023). These connections highlight the central role of physiological processes in emotional disorders, reinforcing why CBT approaches typically address sleep, routine, and physical needs as foundational elements of treatment (Beck, 2011; Brown & Barlow, 2009;

Craske, 1991).

Our analysis used residuals as change scores, which control for each symptom's autocorrelation. This means that the edges in our networks represent relationships between symptom changes beyond what would be expected from each symptom's tendency to correlate with itself over time. Consequently, what remains in the networks are the underlying dynamic relationships between symptoms that exist independently of each symptom's natural tendency to persist. One possibility is that these fundamental symptom relationships may be more consistent across disorders than the raw symptoms themselves. The counterintuitive similarity between pre-treatment (Phase 1) and during-treatment networks (Phases 2 and 3) suggests that the fundamental ways symptoms interact during change may be relatively stable regardless of whether change is occurring naturally or in response to therapeutic intervention. These findings indicate that therapy may work by leveraging existing natural change mechanisms rather than by creating entirely new patterns of symptom interaction—which should be further investigated in future studies.

Differences

In depression networks, edges between anxious symptoms and mechanisms (e.g. restlessness, trouble relaxing, worry) were significant at first, but became weaker over the course of therapy. This supports previous network analytic findings in high-intensity CBT for depression of change mechanisms from excessive worry to control of worry, and from trouble relaxing to restlessness—effects that were specific to CBT rather than e.g. counselling (O'Driscoll et al., 2023). Our change score networks provide enhanced resolution, suggesting these anxiety-related processes may be more critical early in CBT for depression rather than in later stages, a finding warranting replication.

Our GAD-specific findings indicate strengthening relationships between trouble concentrating and anhedonia, as well as between psychomotor changes and trouble relaxing. These evolving connections (depending on the direction of the effect) could represent several clinically meaningful processes: improvements in subjective mood as concentration improves (e.g. Robichaud and Dugas, 2006; Wells, 1995), enhanced cognitive capacity enabling more effective engagement in therapy (e.g. Southward and Sauer-Zavala, 2020), or a combination of these effects. The strengthening of connections between anhedonia, worry, concentration difficulties, and trouble relaxing during therapy suggests the progressive importance of reciprocal relationships between attentional capacity and pleasure experience in successful GAD treatment. If validated, these findings might support greater emphasis on interventions or techniques that explicitly attempt to encourage positive reward sensitivity (e.g. positive affect treatment; Craske et al., 2019) or mindful attention to pleasurable experiences (e.g. mindful savouring practices; Kiken et al., 2017) in CBT for GAD, especially later in therapy.

The generalisation of learning from exposure has been argued to be a fundamental mechanism for the treatment of anxiety disorders (Clark, 1986; Craske, 1991; Parker et al., 2018). In our panic disorder networks specifically, we observed the disappearance of an edge that initially existed between specific phobia and fear of awful events prior to treatment, followed by its re-emergence in the latter part of treatment. Based on the current findings, this is an unclear relationship, and as such warrants further investigation using methods capable of determining directionality (e.g. dynamic networks). Such research could clarify whether this represents the theoretically predicted generalisation from specific fears (targeted through planned exposures) to more general catastrophic fears (Dymond et al., 2015), extinction learning of initial catastrophising (Otto & Deveney, 2005), or if there is a more nuanced, complex or reciprocal relationship between these symptom changes.

Strengths and Limitations

The current study uses a large, comprehensive dataset from primary care psychological therapy services in England (Saunders et al., 2020), using FIML to maximise the amount of analysable data and reduce biases from missing data (Baraldi & Enders, 2010).

Previous studies exploring transdiagnostic CBT interventions have made a priori separations of CBT for depression and anxiety disorders (well-documented as having a great degree of cross-sectional and longitudinal overlap Kendler et al., 1995; Mansell and McEvoy, 2017) from other CBT protocols (Schaeuffele et al., 2024). Our study focused exclusively on CBT for depression and anxiety disorders. While this focus allowed for meaningful exploration of change processes within this domain, it limits the generalisability of our findings to the broader conceptualisation of 'transdiagnostic CBT'. Our network analyses cannot speak to potential mechanisms shared between CBT for emotional disorders and other CBT protocols such as CBT for psychosis (CBTp), CBT for eating disorders (CBT-E), or trauma-focused CBT (tfCBT). Broadening this focus in future work may be of greater relevance to contemporary debates regarding the common factors of effective CBT, and of therapy more generally (Flückiger et al., 2024; Salkovskis et al., 2023; Southward et al., 2024; Wampold & Flückiger, 2023).

The short-term, low-intensity CBT examined in this study is characterised by psychoeducation and behavioural work, which makes the nomothetic approach taken reasonably applicable (e.g. Helmich et al., 2024; O'Driscoll et al., 2022). However, this focus limits our ability to generalise findings to high-intensity, longer-term, or more specialised CBT interventions. Additionally, our study does not account for potential differences in delivery modality (in-person versus remote), which may affect therapeutic processes and outcomes, especially relevant given recent shifts toward digital interventions.

While our group-level network analyses provide valuable insights into shared mechanisms across disorders, they cannot capture the significant individual-level variations in network structures that occur during therapy, as demonstrated by idiographic approaches in clinical psychology (e.g. Ebrahimi et al., 2024; Hoekstra et al., 2024; Molenaar, 2004, 2013). The relative and specific utility of nomothetic versus idiographic approaches to network analysis of symptom change remains an open question. Our findings should be interpreted within this context, recognising that individual patients may experience symptom change patterns that diverge from the group-level networks we identify.

The current study did not disaggregate analyses by gender, ethnicity or socioeconomic status. This was because of the novelty of the analytic techniques used, and the complexity of the resultant models before taking individual or systematic differences into account. However, this is a limitation of the current given the well-established mental health inequalities for gender (Bekker & van Mens-Verhulst, 2007) and ethnicity (Halvorsrud et al., 2019) in regard to both symptom severity and access to care (Cooper et al., 2013).

Future Directions

Our study introduces a novel application of GGMs to model change scores at different therapy time-points. Future studies should use dynamic network models to identify the directionality of symptom dynamics.

Conducting studies using similar methods to the current study with individuals receiving high-intensity CBT would offer deeper insight into putative processes of change over the course of effective CBT. For example, findings might indicate similar network structures of change (implying common recovery processes across different intensities of CBT), or conversely show different network structures, e.g. with high-intensity CBT showing a greater role for cognitive restructuring and reflection (Beck, 2011; Gyani et al., 2013).

The surprising similarity between Phase 1 (assessment to first treatment session) and active-treatment change processes may be partially attributable to our methodology controlling for autocorrelation. If this similarity is reliable, future research should examine whether similar network structures appear among non-responders to treatment. Absence of differences would suggest these networks represent general emotional symptom change patterns regardless of improvement direction. Conversely, Phase 1 network differences between responders and non-responders to treatment might indicate potential early recovery markers. Given the novel methods employed, we strongly recommend replication to rule out statistical artifacts before applying these findings to theory development or clinical practice.

Conclusion

This study provides novel insights into transdiagnostic and disorder-specific change mechanisms in CBT through network analysis of symptom change scores. Our findings reveal both shared patterns across depressive and anxious disorders—particularly connections between worry components and between physical symptoms like sleep and irritability—and disorder-specific relationships that emerge during treatment. These results suggest potential targets for intervention to be investigated in future studies that may be particularly important at specific stages of therapy.

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