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Evaluation of the cognitive behavioural theory of eating disorders: A network analysis investigation

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ABSTRACT

Objective: One of the prevailing theories of eating disorders (ED) is the transdiagnostic cognitive behavioural theory of eating disorders, which suggests that certain ED symptoms, such as over-valuation of eating, shape, and weight, may be more central than others. In the present study, network analyses were used to evaluate these assumptions in a patient sample.

Methods: Participants were 336 individuals receiving treatment at an expert center for ED in the Netherlands. Eating disorder symptoms were used to create transdiagnostic and diagnosis-specific networks and assess symptom centrality and density of the networks.

Results: Networks for patients with bulimia nervosa and binge eating disorder confirmed that over-valuation of shape, weight, and eating is the most central symptom in the network. A transdiagnostic network of ED symptoms and separate networks for patients with anorexia nervosa and bulimia nervosa showed that strict dieting was an additional central ED symptom. An exploratory analysis revealed that, although eating disorder symptoms decreased, there were no differences in density of the eating disorder networks before and after treatment with cognitive behavioural therapy.

Discussion: In conclusion, the current study confirmed that over-valuation of shape, weight, and eating is a central symptom across eating disorders, in agreement with the transdiagnostic cognitive behavioural model of eating disorders. Specifically targeting this symptom in treatment could lead to other symptoms improving as a result.

1. Introduction

A prevailing theory of eating disorders is the transdiagnostic cognitive behavioural theory of eating disorders (Fairburn, Cooper, & Shafran, 2003). One of the premises of this theory is that anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED) share the same distinctive psychopathology. A dysfunctional system for evaluating self-worth based mainly on eating habits, shape, or weight is considered to be the core maintaining factor of eating disorders. Other factors, such as extreme dieting, compensatory vomiting, laxative use or excessive exercise, are seen as direct consequences of the core features. The cognitive behavioural theory of eating disorders has led to the

development of Enhanced Cognitive Behaviour Therapy for eating disorders (CBT-E), which has proven to be an effective, transdiagnostic treatment for eating disorders (Byrne, Fursland, Allen, & Watson, 2011; Fairburn et al., 2015; Hay, 2013; Linardon, Wade, de la Piedad Garcia, & Brennan, 2017). In the first evaluations of the theory, confirmatory latent-trait analytic strategies were employed to determine whether it could be confirmed (Hoiles, Egan, & Kane, 2012; Lampard, Tasca, Balfour, & Bissada, 2013). These studies showed that over-valuation of weight and shape is indeed transdiagnostic, and that this was a mediating factor between cognitive factors on the one hand and dietary restraint on the other hand.

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1.1. Network analysis to evaluate the cognitive behavioural theory of eating disorders

An analytical constraint in previous studies is that all symptoms were represented as independent indicators of the eating disorder (DuBois, Rodgers, Franko, Eddy, & Thomas, 2017; Forbush, Siew, & Vitevitch, 2016), whereas cognitive-behavioural theory suggests that certain ED symptoms, such as over-valuation of eating, shape, and weight, are more central than others. Network theory (Borsboom, 2017; Cramer, Waldorp, van der Maas, & Borsboom, 2010) provides a framework that explains such direct relations between symptoms and their dependency structure, in which certain symptoms are more tightly connected than others. In addition, statistical tools for estimating networks from psychological data (i.e., network analysis) have recently been developed (Epskamp, Borsboom, & Fried, 2018; Epskamp & Fried, 2018). The utility of the network approach, especially with regard to the cognitive behavioural theory of eating disorders, has led to several publications in which network analyses have been used to evaluate this theory (Christian et al., 2020; DuBois et al., 2017; Forbush et al., 2016; Forrest, Jones, Ortiz, & Smith, 2018). The most consistent finding from these studies was indeed the centrality of over-valuation of shape and weight across disorders. However, these studies did not distinguish between AN, BN and BED, making it impossible to test whether indeed cognitive-behavioural theory applies similarly for all eating disorders (Christian et al., 2020; Forrest et al., 2018). An additional limitation is that, although using scale scores in network analysis is recommended (Smith et al., 2018), several of these studies used the eating disorder examination questionnaire (EDE-Q; Fairburn & Beglin, 2008) on an item-specific level instead of subscale level to estimate eating disorder networks (Christian et al., 2020; Forrest et al., 2018). Further, a previous network study on the cognitive behavioural theory of eating disorders (DuBois et al., 2017) used the eating pathology symptom inventory (EPSI; Forbush et al., 2013), while the EPSI subscales do not map clearly onto the transdiagnostic theory of eating disorders. In contrast to the EPSI, the EDE-Q does include the central factors of the transdiagnostic theory of eating disorders, and should therefore be included in a critical review of the model.

In the literature of network analysis, the inclusion of items and the resolution at which items should be modelled (sub-scales versus single items), is an ongoing scientific debate (Fried & Cramer, 2017). Currently, there are no generally applicable recommendations, and it has been noted that the choice of items and the resolution of network components should always be driven by the specific research question at hand, and the level at which inferences are ought to be made (Burger, Isvoranu, et al., 2020). In this study, our primary interest was to map the nodes in the network onto components of the transdiagnostic cognitive behavioural theory of eating disorders as much as possible. Furthermore, including single items would have led to a large number of nodes (28 in total), in turn making accurate estimations of the network structure unlikely given the size of the present sample. For these reasons, we opted to include the sub-scales rather than single items, as these better represent the theoretical components, and allow for a reliable estimation.

1.2. Eating disorder networks before and after treatment

According to network theory, mental health is defined by the connectivity of a symptom network, where healthy individuals portray a stable state of a weakly connected network, whereas the stable state of a strongly connected network represents a mental disorder (Borsboom, 2017). Following this line of reasoning, remission of a mental disorder after treatment should be represented in a less dense or less connected network of symptoms. The less dense the network, the less risk there is for relapse, because a single symptom or trigger will not automatically lead to an increase in other symptoms. The few studies that have examined differences in symptom networks between ill and remitted or

healthy patients however, have shown mixed results, ranging from finding denser networks in (treatment-resistant) ill patients compared to remitted patients or healthy controls (Pe et al., 2015; van Borkulo et al., 2015) to a lack of change in density after treatment (Smith et al., 2019), to an increase in network density after treatment compared to before treatment (Bos et al., 2018; Hilbert et al., 2020). It would be very informative to test whether connectivity is a representation of mental health status in a population with eating disorders, or whether, as suggested by Hilbert et al. (2020), connectivity increases after treatment, perhaps due to increased knowledge on relations between eating disorder symptoms. This is important to know, especially given the high relapse rates in patients with low scores on eating disorder symptoms (McFarlane, Olmsted, & Trottier, 2008). Therefore, in the current study, network connectivity before and after treatment was compared.

1.3. Present study

The goal of the current study was twofold. The first goal was to evaluate the cognitive behavioural model of eating disorders in eating disorder patients using the original subscales of the EDE-Q. In accordance with previous findings, it was expected that the most central symptom would be shape-, weight-, and eating concern. This hypothesis was tested transdiagnostically as well as diagnosis specific (AN, BN, BED). The second goal of the current study was to examine whether eating disorder networks changed over the course of treatment. We compared networks before and after treatment in a transdiagnostic patient population treated at a facility where CBT is the treatment-as-usual. It was expected that, compared to baseline, the networks would be less connected/dense after treatment.

2. Methods

2.1. Procedure

Participants were individuals receiving treatment at the GGNet Amaram Expert Centre for Eating Disorders in the Netherlands in the years 2013–2016. Here, individuals aged 12 years and older (but mainly adults) are treated for their eating disorder in both inpatient and outpatient settings. Of the 686 participants that were in care during 2013–2016, there were 336 patients that participated in the routine outcome measure (ROM) and were thus included in the current study. Eating disorder diagnoses according to the DSM-IV (American Psychiatric Association, 2000) were established through a clinical interview with a psychologist as part of routine clinical care. Based on this diagnosis, participants were assigned to one of the following three participant profiles: 1) the AN profile ($n = 134$; those with a full DSM-IV AN diagnosis or a DSM-IV Eating Disorder Not Otherwise Specified (EDNOS) diagnosis falling within the subthreshold AN category); 2) the BN profile ($n = 72$; those with a full DSM-IV BN diagnosis or a DSM-IV EDNOS diagnosis falling within the subthreshold BN category); 3) the BED profile ($n = 107$; those with a DSM-IV EDNOS diagnosis, fulfilling the (subthreshold) DSM-5 BED criteria). Some participants ($n = 49$) could not be assigned to either of these profiles and were omitted from the subgroup analyses. Participants were given the opportunity to object to their anonymous data being used for research purposes. At the start and end of treatment, participants completed online questionnaires regarding eating disorder symptoms as part of ROM.

2.2. Measures

2.2.1. Eating disorder symptoms

The Dutch version of the Eating Disorder Examination Questionnaire version 6.0 (EDE-Q; Fairburn & Beglin, 2008; van Furth, 2000) is a self-report measure that was used to assess eating disorder symptoms. The EDE-Q consists of 28 questions assessing ED symptoms over the past 28 days, with 22 items rated on a scale ranging from 0 to 6. Higher scores

represent greater symptom severity. Additionally, six open-ended items assess the frequency of binge eating and compensatory behaviors such as vomiting/laxative abuse, and excessive exercise. A previous study showed good validity and reliability in a Dutch sample (Aardoom, Dingemans, Op't Landt, & Van Furth, 2012). In the present study, the following subscales were used: 1) shape and weight concern, 2) eating concern, 3) restraint (strict dieting), 4) binge eating (item 14), 5) compensatory vomiting/laxative misuse (average of items 16 and 17), and 6) excessive exercise (item 18).

2.3. Treatment

Treatment was provided in accordance with the standardized treatments advised in the Netherlands. More specifically, for each eating disorder, a CBT protocol adjusted for eating disorders is advised as the standardized treatment, and this protocol was administered to most patients in this study. An exception was a subsample of the patients with BED, who received dialectical behaviour therapy, which is related to CBT, but has a more central focus on emotion (regulation). Dialectical behaviour therapy is advised as the treatment of second choice in the standardized treatment protocol for BED.

2.4. Analyses

SPSS (version 25.0) was used to provide descriptive statistics (EDE-Q global and subscale scores as well as gender and age) for the study sample. A paired samples *t*-test was performed to examine whether participants showed improvement on EDE-Q global and subscale scores after treatment, compared to baseline.

2.4.1. Network estimation

In order to examine the network structures (i.e. the different ways the eating disorder symptoms are connected), networks were estimated using the *bootnet* package in R (Epskamp, Borsboom, & Fried, 2018) for A) the full data set at T0 B) the sub-groups (AN, BN, BED) at T0, and C) the full data set at T1. Model selection *without* regularization, implemented in the *bootnet* package (*ggmModSelect*) was used to estimate networks in the present study. This procedure first estimates a set of regularized networks (e.g., 100), subsequently fits un-regularized networks for each of these models, and finally performs a model selection procedure. The estimation method in this study is based on Spearman's rank partial correlation using pairwise complete observations. To get an estimate of the accuracy of the network structures, accuracy analyses of network estimates were calculated and visualized using the *bootnet* package. This routine provides confidence intervals for each estimated edge, as well as significance tests for each comparison of a pair of edges within a network. Detailed explanations of the accuracy routine have been outlined elsewhere (Epskamp & Fried, 2018).

2.4.2. Centrality of nodes

To evaluate which eating disorder symptoms occupy the most central positions within the network, strength centrality measures have been calculated by summing the total absolute incoming/outgoing edge weights for each node. Further, the stability of these coefficients has been assessed using the *bootnet* package in R. This routine uses a case-drop procedure to demonstrate how highly original centrality estimates correlate with the ones from a sub-set for varying proportions of dropped cases. The correlation stability (CS) coefficient quantifies the proportion of participants that can be dropped such that with 95% confidence a correlation of 0.7 between original and sub-set centrality indices is retained. Finally, the difference between eating disorder symptom centrality was assessed using centrality difference tests.

2.4.3. Network visualization and comparison

The estimated network and centrality coefficients have been visualized using the *qgraph* package in R (Epskamp, Cramer, Waldorp,

Schmittmann, & Borsboom, 2012), and the similarity of their structure compared through correlating adjacency matrices. To examine structural differences between the network at T1 compared to the network at T0, a permutation test was performed using the *NetworkComparisonTest* package in R (Van Borkulo et al., 2016).

3. Results

3.1. Descriptive statistics

Descriptive statistics of the different subsamples used to plot the networks are portrayed in Table 1. The most common diagnosis was AN, followed by BED and BN. The sample consisted of mostly females ranging in age from 12 to 68, averaging around 30. At baseline, the EDE-Q scores were within the extremely high range compared to the norm scores of the general population and within the average range compared to an eating disorder population (Aardoom et al., 2012). The average time between start (T0) and end of treatment (T1) was 37 weeks (SD = 26.46; min = 3, max = 119). As Table 1 shows, post-treatment total and subscale EDE-Q scores were significantly lower compared to pre-treatment scores. The scores were in the low range compared to eating disorder patients and in the high range compared to healthy controls (Aardoom et al., 2012). At T1, 112 (36.7%) of the patients were remitted, defined as having a global EDE-Q score less than 1 standard deviation (SD) above the community mean (Fairburn et al., 2015), while 138 (45.2%) of the patients showed reliable change (i.e. improvement) on the EDE-Q, calculated according to the method described by Jacobson and Truax (1992).

3.2. Networks and stability

Graphical results of the stability and accuracy analyses as well as the figures for the bootstrap difference tests for centrality are available as online supplementary material. Stability analyses resulted in CS coefficients of 0.67 for the before- and 0.75 for the after treatment transdiagnostic networks, and 0.52 for the AN network, which is above the suggested threshold of 0.5 (Epskamp, Borsboom, & Fried, 2018). CS coefficients for the BN and BED network were 0.29 and 0.28. These coefficients suggest that we can have confidence in the centrality measures within the transdiagnostic and the AN networks, while the measures in the BN and BED networks were less stable. Accuracy analyses revealed that the edge weight confidence intervals were rather broad, and some edge weights do not differ significantly from other edge weights. This means we have to be cautious of interpreting the order of edge weights.

3.3. Network structure at baseline

When examining the transdiagnostic and diagnose-specific networks (Figs. 1.1–1.5) and centrality plots (Figs. 2 and 3) at baseline, it appeared that in the transdiagnostic network, over-valuation of eating was the most central symptom, followed by strict dieting, and over-valuation of shape and weight. Strength-centrality differed significantly from most other symptoms in the transdiagnostic network, but not within these symptoms. For BN, over-valuation of eating, dieting, and over-valuation of shape and weight were the most central symptoms in the network. Follow-up bootstrap difference tests revealed that only over-valuation of eating differed significantly in centrality from binge eating. For BED, over-valuation of shape and weight, and over-valuation of eating were the most central symptoms. Follow-up bootstrap difference tests revealed that these differences in centrality were significant for most of the other nodes, except for binge eating. For AN, strict dieting was the most central symptom, followed by over-valuation of shape, weight, and eating. Follow-up bootstrap difference tests indicated that these three symptoms had significantly larger strength-centrality scores than the remaining nodes, but were not differing from one

Table 1

Descriptive statistics (M (SD)) for the full sample, the subsamples at T0, the full sample at T1, and results from t-tests for differences between the full samples at T0 and T1.

	Full sample T0 (n = 336)	Full sample T1 (n = 305)	t (p)	Cohen's d	AN T0 (n = 134)	BN T0 (n = 72)	BED T0 (n = 107)
Female (n, %)	317 (94.3)	290 (95.1)			130 (97.0)	72 (100.0)	93 (86.9)
Age	29.52 (12.23)				24.72 (9.65)	25.69 (9.03)	37.66 (12.40)
EDE-Q total	3.48 (1.45)	2.58 (1.55)	12.31 (<0.001)	0.59	3.53 (1.51)	4.01 (1.25)	3.01 (1.27)
Over-valuation of shape and weight	4.12 (1.57)	3.22 (1.78)	10.90 (<0.001)	0.54	4.05 (1.60)	4.63 (1.37)	3.83 (1.53)
Over-valuation of eating	2.98 (1.50)	2.01 (1.50)	11.66 (<0.001)	0.63	2.90 (1.48)	3.58 (1.38)	2.63 (1.44)
Strict dieting	2.71 (1.81)	1.88 (1.69)	8.64 (<0.001)	0.47	3.14 (1.84)	3.21 (1.53)	1.73 (1.45)
Excessive exercise	6.67 (13.82)	4.36 (7.22)	3.29 (<0.001)	0.23	10.19 (19.67)	6.60 (8.46)	2.41 (4.90)
Binge eating	8.35 (10.91)	3.30 (6.43)	7.43 (<0.001)	0.55	5.03 (12.01)	12.60 (9.69)	10.54 (9.40)
Compensatory vomiting / laxative misuse	3.92 (9.55)	1.68 (5.12)	4.56 (<0.001)	0.30	4.35 (12.32)	7.79 (9.61)	0.69 (2.81)

AN = anorexia nervosa; BN = bulimia nervosa; BED = binge-eating disorder; EDE-Q = eating disorder examination questionnaire.

Note. Given that some participants could not be assigned to a specific diagnostic subgroup (AN, BN, BED), the sample sizes of the subgroups do not add up to the full sample size at T0.

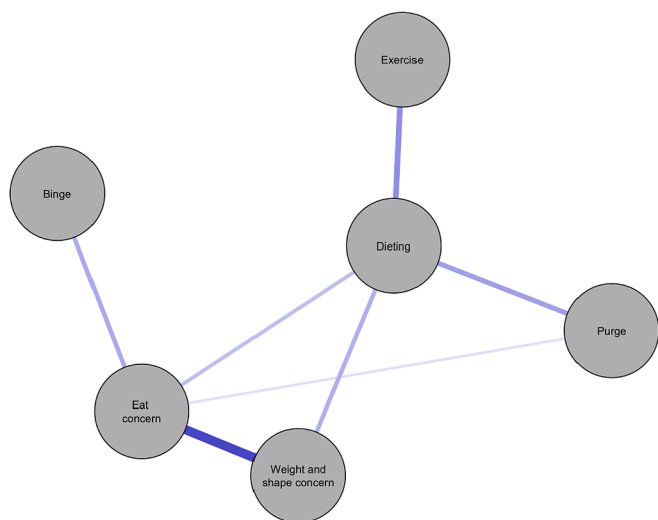


Fig. 1.1. Transdiagnostic eating disorder symptom network T0 (n = 336) Nodes represent eating disorder symptoms. Edges represent Spearman's rank correlations between any two symptoms. Blue edges represent positive relationships and red dashed edges represent negative relationships. The magnitude of the associations is represented by the width and color-intensity of the edges. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

another. In the transdiagnostic network, all symptoms were connected to other symptoms in the network. However, in the AN network, binge eating was not connected to other symptoms in the network, while for the BN and BED networks, compensatory vomiting / laxatives misuse was not connected to other symptoms in the networks. The correlations between the adjacency matrices of the AN, BN, and BED networks ranged between 0.52 and 0.62, indicating that the network structures for these diagnostic groups were rather similar. Also, Network Comparison Tests revealed no significant differences between the AN, BN, and BED networks, with the p-value ranging between 0.42 and 0.76.

3.4. Comparison of networks before and after treatment

After analysing the symptom networks before and after treatment (Figs. 1.1–1.5), it appeared that there were no significant differences in global network strength when comparing the before and after treatment networks (p = .86). The correlation between the adjacency matrices of the before and after treatment networks was 0.90, indicating that both network structures were very similar. Also, the most central symptoms within the networks were the same, being over-valuation of eating, strict dieting, and over-valuation of shape and weight (Fig. 2).

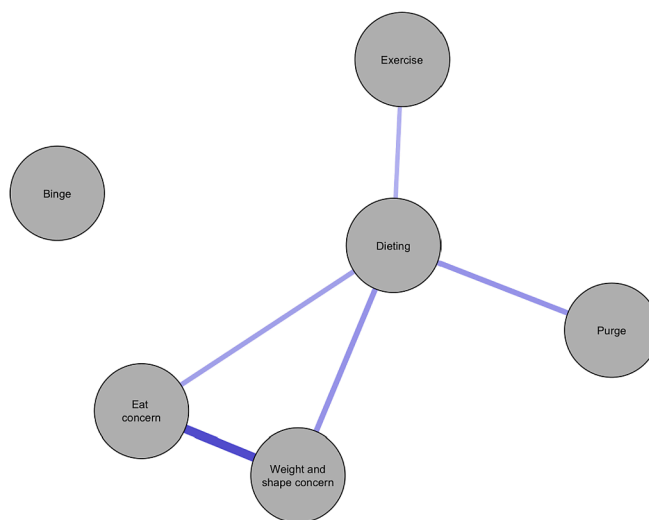


Fig. 1.2. AN symptom network (n = 134) Nodes represent eating disorder symptoms. Edges represent Spearman's rank correlations between any two symptoms. Blue edges represent positive relationships and red dashed edges represent negative relationships. The magnitude of the associations is represented by the width and color-intensity of the edges. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

4. Discussion

In line with our first hypothesis, all networks in the current study showed that over-valuation of shape, weight, and eating was among the most central symptoms in patients with eating disorders. Also, there were no significant differences between networks. This finding was in line with previous findings (Christian et al., 2020; DuBois et al., 2017; Forbush et al., 2016; Forrest et al., 2018). In addition, strict dieting was one of the most central symptoms in the transdiagnostic, AN, and BN networks. This result also corresponds to findings in previous network studies (Christian et al., 2020; Forbush et al., 2016; Forrest et al., 2018), in which restraint, avoiding high-calorie foods, and fasting were found to be highly central symptoms. These results largely comply with the prevailing theory within the eating disorders field, in that over-valuation of shape, weight, and eating is a central, transdiagnostic symptom among patients with eating disorders (Fairburn et al., 2003). Targeting over-valuation of shape, weight, and eating during treatment might result in subsequent changes in other eating disorder symptoms such as bingeing, dieting, and purging. The centrality of strict dieting in the AN and BN network, and not in the BED network is in line with the CBT-E model, in which strict dieting is seen as a shared feature between

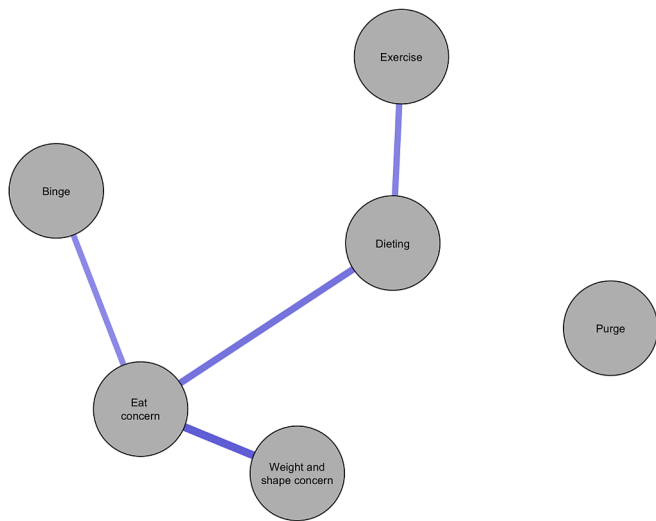


Fig. 1.3. BN symptom network (n = 72)

Nodes represent eating disorder symptoms. Edges represent Spearman's rank correlations between any two symptoms. Blue edges represent positive relationships and red dashed edges represent negative relationships. The magnitude of the associations is represented by the width and color-intensity of the edges. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

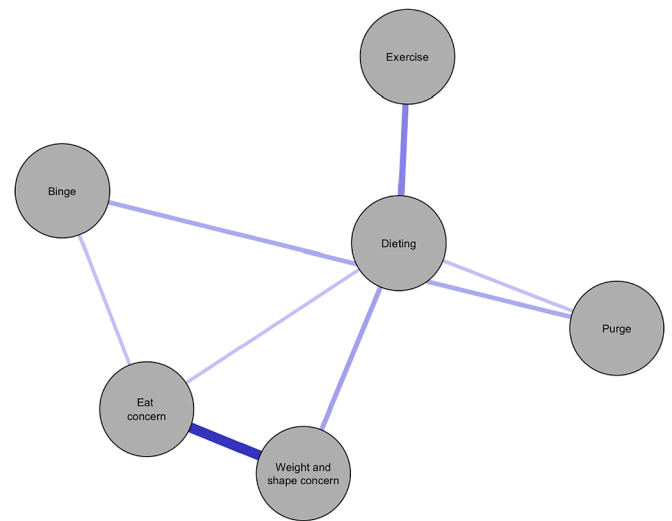


Fig. 1.5. Transdiagnostic eating disorder symptom network T1 (n = 304)

Nodes represent eating disorder symptoms. Edges represent Spearman's rank correlations between any two symptoms. Blue edges represent positive relationships and red dashed edges represent negative relationships. The magnitude of the associations is represented by the width and color-intensity of the edges. The sample size used for the network analyses at T1 were smaller compared to the sample size at T0 due to missingness on the EDE-Q. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

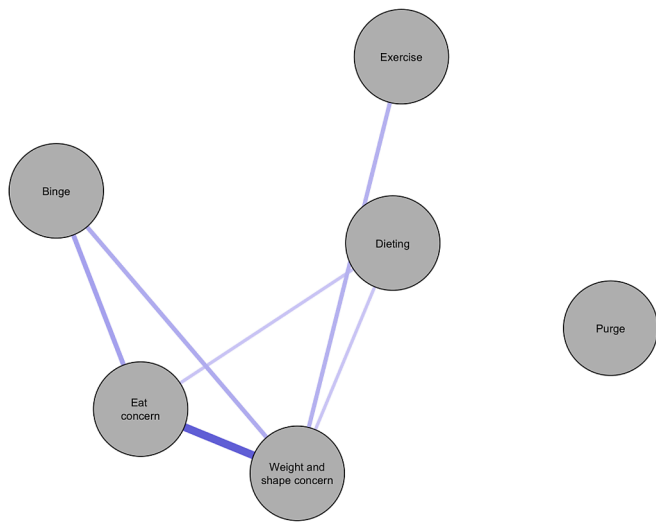


Fig. 1.4. BED symptom network (n = 107)

Nodes represent eating disorder symptoms. Edges represent Spearman's rank correlations between any two symptoms. Blue edges represent positive relationships and red dashed edges represent negative relationships. The magnitude of the associations is represented by the width and color-intensity of the edges. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

AN and BN. Also, findings seem to confirm the idea that undereating or strict dieting is more prominent in AN (Fairburn et al., 2003). The finding of strict dieting as a central aspect of the network justifies the emphasis put on normalization of eating behaviour in AN and BN treatment, and the lack of significant differences when comparing diagnostic networks, it seems sound to keep the same emphasis on eating normalization in BN and BED (Södersten, Bergh, Leon, Brodin, & Zandian, 2017).

A striking result of the current study was the low centrality of compensatory vomiting / laxative misuse in the networks, and the absence of a connection with binge eating in the BN analyses and to a

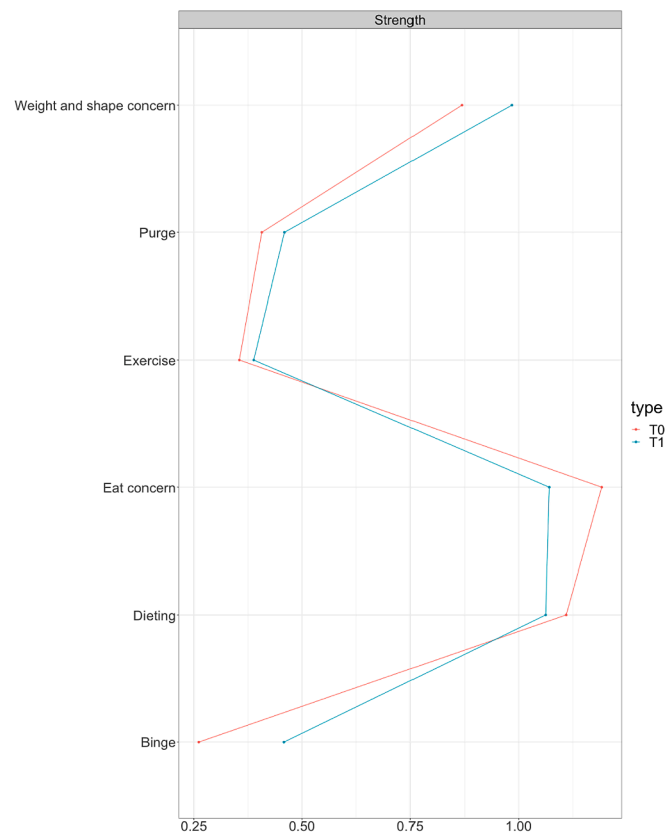


Fig. 2. Symptom strength centrality for the transdiagnostic network models at T0 and T1.

lesser extent also in the AN analyses. In the CBT-E model (Fairburn et al., 2003), a direct feedback loop is hypothesized between binge eating and compensatory vomiting / laxative misuse. Although the present finding

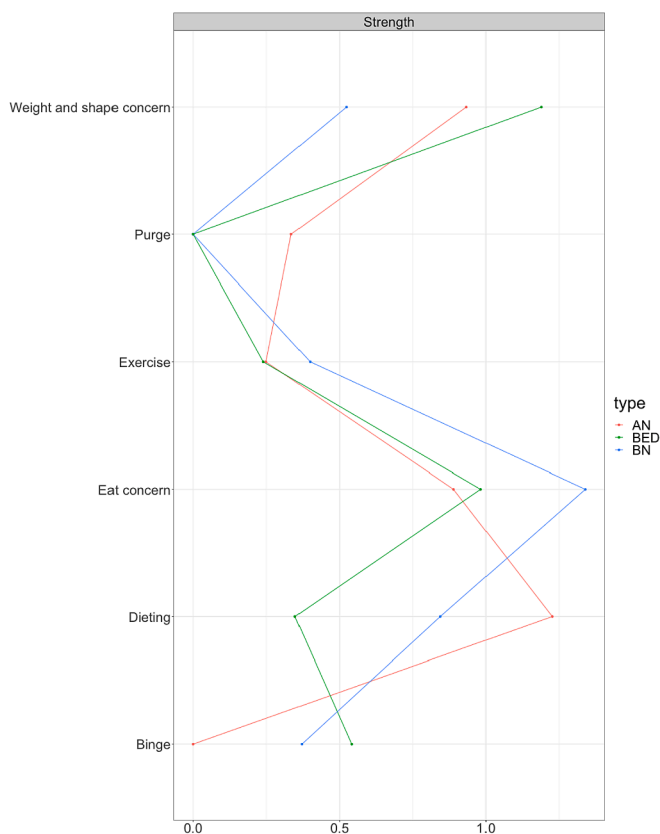


Fig. 3. Symptom strength centrality for the AN, BN, and BED network models.

does not correspond to the theoretical model, the low centrality of compensatory vomiting / laxative misuse is in line with previous findings (Forbush et al., 2016; Hovrud & De Young, 2015). A reason for this finding might be that compensatory vomiting / laxative misuse can be seen mainly as collateral damage that comes with other, more central eating disorder symptoms. It is also important to note, however, that the specific edges/connections within networks show limited replicability (Borsboom, Robinaugh, Group, Rhemtulla, & Cramer, 2018; Forbes, Wright, Markon, & Krueger, 2017), and the stability of the BN network is limited. Therefore, these results should be reviewed with caution. Compensatory vomiting / laxative misuse can result in serious somatic complications, and therefore deserves attention within treatment. Yet, based on present findings regarding centrality, we assume that changing these behaviors will not result in less overall eating disorder symptoms or impairment.

The results of the exploratory analyses comparing the networks before and after treatment revealed that, although there was decreased symptom severity, and some shifts in significant edges within the network, there was no difference in network strength or connectedness. From a theoretical point of view, a less connected network after treatment would have been expected (Borsboom, 2017), yet previous studies in which eating disorder networks before and after treatment were compared also failed to show a decrease in network connectivity (Hilbert et al., 2020; Smith et al., 2019). Perhaps the fact that patients still reported fairly high over-valuation of shape and weight, a central factor in the network, contributed to the continued connectedness of the networks. Another possibility would be that treatment increases knowledge on relations between eating disorder symptoms (Hilbert et al., 2020), which might have interfered with a reduction in density. The addition of significant edges in the network after treatment would also point in this direction, although inferences about specific edges should be made with caution (Borsboom et al., 2018; Forbes et al., 2017). Also, although overall eating disorder symptoms decreased, not all patients in the

current sample showed (full) remission at the end of treatment. Indeed, remission rates for CBT-E range from 22 to 68%, indicating that there is significant room for improvement with regard to treatment effectiveness (de Jong, Schoorl, & Hoek, 2018). Perhaps, when a sample is used that consists of solely remitted patients, and they are followed over a prolonged period of time after treatment termination, a difference in network connectivity can be observed. In the current sample, lack of difference in network strength after treatment compared to before treatment might indicate that, although these patients report decreased eating disorder severity, they are vulnerable for relapse. This corresponds to epidemiological data, which shows that up to 40% of remitted eating disorder patients relapsed within 12 months (McFarlane et al., 2008). When these patients experience an increase in one of the eating disorder symptoms, this might serve as a trigger for the entire eating disorder network to flare up. Future research could provide more clarity on this issue by following eating disorder networks of individual patients for an extended period of time after treatment.

An important strength of the current study is the strong theory-guidance. Theory development is the heart of establishing stable psychological phenomena (Borsboom, van der Maas, Dalege, Kievit, & Haig, 2021). Recently, it has been discussed that clinical psychology often faces an inference gap between data models and theories (Burger, van der Veen, et al., 2020; Fried, 2020; Hasibeck, Ryan, Robinaugh, Waldorp, & Borsboom 2019). The impetus of this study was therefore to align our analyses and measurement scales with the nature of the CBT-model of eating disorders, and evaluate its implications. There are, however, also some limitations that need to be mentioned. A first limitation of the current study was the limited stability of the BN and BED networks. Due to the low stability, results of these networks should be interpreted with great caution. This lack in stability in the BN and BED networks was probably due to the relatively small subsamples of patients with a diagnosis of BN or BED. In addition, the lack of a diagnostic instrument to assess diagnostic categories could have caused diagnostic noise. Future studies should therefore include reliable diagnostic assessment instruments (such as SCID 5) and ensure more equal distributions of different diagnostic samples, thereby optimizing model comparisons. Second, the present study evaluated the core factors of the transdiagnostic cognitive behavioural model of eating disorders. Future studies should also take the maintenance factors (perfectionism, self-esteem, and mood intolerance) into account, since these are additional factors in the transdiagnostic theory thought to play a role in the maintenance of the eating disorder (Fairburn et al., 2003). Finally, we analyzed between-subject data with regard to the networks before treatment, meaning that any conclusions regarding individual dynamics remain putative and should be tested in idiographic designs. However, the aim of this study is to evaluate a theory, which validates the use of between-subject data.

In conclusion, the current study supports that over-valuation of shape, weight, and eating is a central symptom across eating disorders, in agreement with the transdiagnostic cognitive behavioural model of eating disorders. Specifically targeting this symptom in treatment could lead to other symptoms improving as a result. Further, results showed that, given the centrality of strict dieting in especially the AN subsample, but also BN subsamples in previous studies, the emphasis on normalization of eating behaviour in many eating disorder treatments seems justified. The lack of change in network density over time indicates that more research is needed on the evaluation of treatment effects on eating disorder networks over time.

Data availability statement

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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