Keywords

Binge-eating disorder · Eating disorder · Cognitive-behavioral therapy · Network analysis · Prediction

Abstract

Introduction: Network approaches to psychopathology posit that mental disorders emerge from interrelated symptoms, and thus connectivity among symptoms are assumed to negatively predict the treatment response and decrease with efficacious treatment. Objective: This study uniquely sought to elucidate the network structure, its change, and its predictive value in cognitive-behavioral therapy (CBT) for binge-eating disorder (BED). Methods: In a multicenter randomized trial of face-to-face and Internet-based guided self-help CBT, 178 individuals with full syndrome and subsyndromal BED, eating disorder and general psychopathology, and body mass index (BMI) were subjected to Gaussian Graphical Network and Exploratory Graph Analyses before and after treatment and at 6-month follow-up. Results: At pretreatment, 3 network communities of: eating disorder psychopathology; general psychopathology; and restraint and BMI were identified, with the latter community included in the first thereafter. Eating disorder-related impairment and self-esteem were the most central symptoms, while BMI and binge eating had the lowest centrality. Network connectivity significantly increased from pre- to posttreatment, with the greatest increases in strength centrality found in binge eating and shape concern, but it did not predict remission from binge eating. Conclusions: With decreasing symptom severity, CBT resulted in a greater integration and connectivity of the psychopathology network in BED, suggesting an increased patient understanding of relations between binge eating and other symptoms. Network connectivity was not a negative prognostic indicator of treatment outcome. These results indicate a need for further research on the predictive value of network variables in the explanation of therapeutic change for patients with BED.
Introduction

Binge-eating disorder (BED) is characterized by recurrent binge eating in the absence of inappropriate compensatory behaviors [1]. This prevalent clinical eating disorder co-occurs with eating disorder and general psychopathology, obesity (BMI ≥ 30.0 kg/m²), and psychosocial impairment. Cognitive-behavioral therapy (CBT) is the most well-established therapy for BED, with similar efficacy in face-to-face and CBT structured self-help treatment formats [2, 3], but pretreatment predictors of treatment outcomes have proven elusive [4]. Using a network approach to psychopathology, this study uniquely sought to elucidate the network structure, its change, and the predictive value in CBT for BED.

Network approaches to psychopathology posit that mental disorders emerge from a dynamic interplay among symptoms, which can be modeled using network analysis [5, 6] and may predict treatment outcome. In psychopathological networks, symptoms represent nodes that are connected with edges, depicting the strength and direction of correlations between symptoms. Symptoms with stronger relations to other symptoms are considered to be central to the network (i.e., have a high strength centrality). Thus, if the relations between symptoms are strong, the network connectivity is high and the network is assumed to be pathological and self-sustaining. Network theory further proposes that treatments may change single symptoms or symptom networks, depending on their efficacy [6]. If a treatment is efficacious, network connectivity was predicted to decrease after treatment [7] – an assumption that has received mixed support in the psychopathology community with a low integration of binge eating, restraint, and BMI; (2) unchanged network connectivity following CBT; and (3) the ability to predict remission from lower pretreatment network connectivity.

Materials and Methods

In a multicenter randomized trial of face-to-face and Internet-based guided self-help CBT [18], 178 individuals with full syndrome and subsyndromal BED (DSM-IV-TR) [19] aged ≥18 years and with a BMI of 27.0–40.0 kg/m² were treated with twenty 50-min individual sessions of manualized CBT [20] or Internet-based guided self-help [21], both delivered over 4 months. For methodological detail, see the online supplementary material (for all online suppl. material, see www.karger.com/doi/19.1159/000509458).

The treatment response at posttreatment (after 4 months of treatment) and at 6-month follow-up was defined as full remission from binge eating (i.e., zero objective binge-eating episodes [OBEs] over the past 28 days), as assessed through the clinical interview Eating Disorder Examination (EDE) [22, 23]. For network analysis, the following interview- and questionnaire-based variables were used: eating disorder psychopathology, operationalized through the number of OBEs over the past 28 days, and restraint and eating, shape, and weight concern (EDE) [22]; emotional eating (Dutch Eating Behavior Questionnaire; DEBQ) [24, 25]; eating disorder-related impairment (Clinical Impairment Assessment; CIA) [26]; and BMI, calculated from measured height and weight (kg/m²). General psychopathology was operationalized through depression (Beck Depression Inventory-II; BDI-II) [27, 28]; eating, shape, and weight concern (EDE) [22, 23]. For network analysis, the following interview- and questionnaire-based variables were used: eating disorder psychopathology, operationalized through the number of OBEs over the past 28 days, and restraint and eating, shape, and weight concern (EDE) [22]; emotional eating (Dutch Eating Behavior Questionnaire; DEBQ) [24, 25]; eating disorder-related impairment (Clinical Impairment Assessment; CIA) [26]; and BMI, calculated from measured height and weight (kg/m²). General psychopathology was operationalized through depression (Beck Depression Inventory-II; BDI-II) [27, 28]; eating, shape, and weight concern (EDE) [22, 23].

Data Analytic Plan

For treatment of missing data by multiple imputation, comparison of completers versus noncompleters, and therapeutic change in the network variables, see the online supplementary material. Each network included the 12 indicators of eating disorder psychopathology, BMI, general psychopathology, and punishment and reward sensitivity at pretreatment, posttreatment, and at 6-month follow-up. First, in order to examine how the network nodes cluster together, an Exploratory Graph Analysis [33, 34] was conducted. This analysis estimates communities in networks via a random walk algorithm (walktrap) in order to evaluate whether nodes that cluster together in communities are part of the same latent variable or dimension. In order to examine network structure at pretreatment, posttreatment, and at 6-month follow-up, Gaussian Graphical Network Analyses with LASSO (least absolute shrinkage and selection operator) regularization were used to limit the number of spurious connections in the network [35]. To explore network stability, the Bootstrapped Difference Test was conducted (bootnet [17]). Based on this initial support for a predictive value of psychopathological network parameters, this study sought to prospectively investigate psychopathology networks in CBT for BED at pre- and posttreatment and at 6-month follow-up, hypothesizing: (1) a distinct eating disorder psychopathology community with a low integration of binge eating, restraint, and BMI; (2) unchanged network connectivity following CBT; and (3) the ability to predict remission from lower pretreatment network connectivity.
package) [36], and the local centrality measures of strength, closeness, and betweenness were estimated to characterize network structure (for definitions, see the online suppl. material).

Second, the change in network connectivity between pretreatment and posttreatment, as well as 6-month follow-up, was analyzed using the Network Comparison Test (NCT) for repeated measurements, including the Global Strength Invariance Test [37]. Third, for predictor analysis, we compared the pretreatment network connectivity of responders with full remission from OBEs at posttreatment and at 6-month follow-up versus that of nonresponders using between-group NCT analyses as described above.

Results

Exploratory Graph Analysis documented the following 3 pretreatment communities (Fig. 1): restraint and BMI, eating disorder psychopathology (binge eating; eating, weight, and shape concern; emotional eating; and eating disorder-related impairment), and general psychopathology (depression, self-esteem, reward and punishment sensitivity). At posttreatment and at follow-up, 2 communities were identified: restraint and BMI were included in the eating disorder psychopathology community, and the general psychopathology community remained the same as at pretreatment, with the exception of eating disorder-related impairment and emotional eating as part of the latter community at follow-up.

Stability analyses for the networks at pretreatment, posttreatment, and at 6-month follow-up revealed adequate correlation stability of strength (0.753, 0.674, and 0.674) and betweenness (0.438, 0.438, and 0.281). Before treatment, the Gaussian Graphical Network Analyses with LASSO regularization (Figure S1) showed the highest node strength for eating disorder-related impairment (1.75) and self-esteem (1.52), followed by shape concern (0.65). As predicted, the lowest strength was found for BMI (–1.43) and binge eating (–1.31). Eating disorder-related impairment had the highest betweenness (2.55) and bridged between the eating disorder psychopathology and general psychopathology communities, and the restraint and BMI community.

According to the NCT’s Global Strength Invariance Test, treatment resulted in a significant increase in network connectivity from pre- to posttreatment (4.10 vs. 4.79, \(S = 0.69, p = 0.02\)) but not from pretreatment to follow-up (4.10 vs. 4.37, \(S = 0.27, p = 0.25\)). The greatest changes in local centrality from pre- to posttreatment (and 6-month follow-up) included an increase in node strength in shape concern (0.65, 1.32, and 1.58) and binge eating (–1.31, –0.51, and –0.77) and a decrease in punishment sensitivity (–0.05, –0.77, and –0.59). The greatest
changes in betweenness from pre- to posttreatment (and 6-month follow-up) were an increase in shape concern (0.52, 1.28, and 1.79) and a decrease in restraint (0.09, –0.90, and –0.83) and eating-disorder-related impairment (2.55, 1.66, and 1.48).

Examining network connectivity as a predictor of treatment response, the NCT’s Global Strength Invariance Test did not reveal any significant differences in pre-treatment network connectivity in responders with remission from binge eating versus nonresponders at post-treatment ($S = 0.12, p = 0.88$) and at 6-month follow-up ($S = 0.36, p = 0.79$). A sensitivity analysis using averaged weight and shape concern showed mostly consistent results, with few deviations, and a further supplementary analysis did not provide evidence for a predictive power of single nodes or local centrality indices (see online suppl. material).

Discussion

This study uniquely explored the network structure and the change and predictive value of network connectivity in a large multicenter randomized trial of CBT for individuals with full syndrome and subsyndromal BED. At pretreatment, 3 distinct network communities of eating disorder psychopathology, general psychopathology (also including dispositional sensitivity to punishment and reward), and restraint and BMI were identified, which is consistent with cross-sectional network research on BED, mixed eating disorders, and obesity [15–17, 38]. In line with most literature on these disorders [15–17, 39–42], eating disorder-related impairment, low self-esteem, and shape concern were among the most central indicators, whereas BMI and binge eating were the least central. The relatively low centrality of punishment and reward sensitivity is consistent with previous evidence on mixed eating disorders [16], likely associated with their general, non-eating-disorder-related formulation. Impairment served as a bridge symptom linking the communities (especially with depression), supporting that eating disorder-related burden was the most prevailing and eradiating symptom with which treatment-seeking individuals with BED presented.

With decreasing symptom severity, CBT for BED resulted in a greater integration of network structure, with the restraint and BMI community included in the eating disorder psychopathology community at posttreatment and at 6-month follow-up. We used Exploratory Graph Analysis [33, 34], linking network analysis with latent variable analysis in order to identify latent variables or dimensions of nodes clustering together, an approach that seeks to reconcile network and “common cause” models [43]. Following CBT, weight pathology did not form an underlying dimension distinct from eating disorder psychopathology anymore, suggesting that the respective symptoms were more closely linked in the maintenance of BED, likely following an integrated developmental pathway [44].

As opposed to the prediction of network theory, assuming that an efficacious treatment should lead to a decrease in network connectivity and its self-sustaining character [4], CBT for BED resulted in a higher connectivity of the psychopathology network, which is consistent with some [8, 9] but not all studies [10] on diverse mental disorders including depression. In mixed eating disorders, Smith et al. [17] did not find a change in network connectivity, presumably related to the lower homogeneity of their sample and treatment approaches. Also opposed to the prediction of network theory [7], and not consistent with Smith et al. [17], network connectivity did not predict remission from binge eating at posttreatment or at 6-month follow-up. Theory, network connectivity was not revealed as an indicator of vulnerability and relapse across 6 months of follow-up. Further research is warranted to address the predictive value of network connectivity in the long term.

Descriptively, changes in network structure were mostly consistent with the therapeutic concept of CBT [20, 45, 46], facilitating connections of core symptoms including binge eating and shape concern with their antecedents and consequences, thereby leading to simultaneous improvements among symptoms. Therefore, our study’s increased network connectivity may not represent a greater psychopathology but rather a therapeutic gain in itself, likely reflecting an enhanced patient understanding of the interrelated nature of psychopathology. These conclusions require confirmation by future research. Of note, a supplementary analysis did not confirm a predictive value of the most central symptoms, as opposed to previous research on mixed eating disorders and obesity [38, 47, 48].

Considering strengths and limitations, our study was based on a large, multicenter RCT [18] using standardized CBT treatment protocols and validated interview- and questionnaire-based assessments through 6 months of follow-up. The cross-sectional network analyses replicated previous findings, lending support to the validity of the results. We had at least 3 times the observations as the number of parameters [36], and thus the sample size sufficed for the analyses and bootstrapping confirmed the ro-
Networks in Treatment for BED

works of BED and the effects of interventions. 
sequences of specific symptoms within dynamic net-
assessments [52], could help to elucidate causes and con-
ment, using intensive longitudinal designs with multiple 
yses on the temporal succession of symptoms over treat-
objectively measured data [12] could represent a viable 
of the interrelations between them. Greater inclusion of 
his/her understanding not only of the symptoms but also 
of the interrelations between them. Greater inclusion of 
objectively measured data [12] could represent a viable 
approach to address this issue. In addition, network anal-
yses on the temporal succession of symptoms over treat-
ment, using intensive longitudinal designs with multiple 
assessments [52], could help to elucidate causes and con-
sequences of specific symptoms within dynamic net-
works of BED and the effects of interventions.

Because of major differences between our study’s re-
results on network connectivity and treatment-related pre-
dictions of network theory, further elucidation of the 
pathological versus healthful nature of networks is neces-
sary. Clarification is warranted regarding to which degree 
networks based on patient-reported symptoms reflect 
his/her understanding not only of the symptoms but also 
of the interrelations between them. Greater inclusion of 
objectively measured data [12] could represent a viable 
approach to address this issue. In addition, network anal-
yses on the temporal succession of symptoms over treat-
ment, using intensive longitudinal designs with multiple 
assessments [52], could help to elucidate causes and con-
sequences of specific symptoms within dynamic net-
works of BED and the effects of interventions.

Acknowledgement

We thank Anne Brauhardt and Frauke Schmidt for their sup-
port in the conduct of this study, and Jamie L. Manwaring, Frederike 
Obereigner, andJosefine Kappe for their editorial support.

Statement of Ethics

This research was conducted ethically in accordance with the 
World Medical Association Declaration of Helsinki. Ethical ap-
proval was granted by the Ethics Committee of the University of 
Erlangen (reference No. 4081), based on which approval was 
granted by site-specific institutional review boards. Informed writ-
ten consent was obtained at the outset of this study.

Conflict of Interest Statement

Dr. Hilbert reports giving lectures and workshops on the psy-
chotherapy of BED and obesity; receiving honoraria as an editor 
with Springer, as a reviewer with Mercator Research Center Ruhr 
and Oxford University Press, and as a consultant with Weight-
Watchers and GlobalData; and publishing books with Hogrefe. Dr. 
Herpertz reports giving lectures and receiving travel grants from 
Berlin Chemie, Lilly, and Sanofi and authoring books and articles 
on diabetes, obesity, and eating disorders published by Springer 
and Thieme. Dr. Zipfel reports authoring books and articles on 
psychotherapy of eating disorders published by Thieme, Springer, 
Routledge, and Elsevier. Dr. Tuschen-Caffier reports giving lec-
tures and workshops on the psychotherapy of eating disorders 
and authoring books and articles on eating disorders published by 
Beltz, DGVT, Huber, Hogrefe, Kohlhammer, Psychosozial-Ver-
lag, and Wiley. Dr. Friederich reports giving lectures and work-
shops on eating disorders and authoring books and articles on eat-
ing disorders published by Springer, Elsevier, Routledge, and Ho-
grefe. Dr. Mayr reports authoring articles on statistical methods 
and eating disorders published by Thieme, Springer, Wiley, and 
Elsevier; authoring books published by Springer; and giving work-
shops on statistics for Siemens Healthcare. Dr. de Zwaan reports 
serving on advisory boards, giving lectures, and receiving travel 
grants from Novo Nordisk and Danone and authoring books and 
articles on psychotherapy of eating disorders published by Thieme, 
Springer, Routledge, and Elsevier. No other disclosures are report-
ed.

Funding Sources

This work was supported by the German Federal Ministry of 
Education and Research (BMBF; 01GV0601). Dr. Hilbert was 
funded by BMBF grant 01EO1501. The sponsors had no involve-
ment in the study design; in the collection, analysis, and interpre-
tation of data; in the writing of this report; or in the decision to 
submit this article for publication.

Author Contributions

Study concept and design: Dr. Hilbert, Dr. Mayr, and Dr. de 
Zwaan. Obtainment of funding: Dr. de Zwaan and Dr. Hilbert. 
Data analysis: Dr. Mayr. Interpretation of data and drafting of this 
paper: Dr. Hilbert, Dr. Mayr, and Dr. de Zwaan. Acquisition of 
data, critical revision of this paper for important intellectual con-
tent: Dr. Hilbert, Dr. Herpertz, Dr. Zipfel, Dr. Tuschen-Caffier, Dr. 
Friederich, Dr. Mayr, and Dr. de Zwaan

References

1 American Psychiatric Association. Diagnos-
tic and statistical manual of mental disorders. 
5th ed. Arlington: American Psychiatric As-
sociation; 2013.
2 Hilbert A, Petroff D, Herpertz S, Pietrowsky 
R, Tuschen-Caffier B, Vocks S, et al. Meta-
analysis of the efficacy of psychological and 
medical treatments for binge-eating disorder. 
J Consult Clin Psychol. 2019 Jan;87(1):91– 
105.
3 Linardon J, Wade TD, de la Piedad Garcia X, 
Brennan L. The efficacy of cognitive-behav-
ioral therapy for eating disorders: A system-
atic review and meta-analysis. J Consult Clin 
Psychol. 2017 Nov;85(11):1080–94.
10 Schweren L, van Borkulo CD, Fried E, Good-

7 van Borkulo C, Boschloo L, Borsboom D, 

13 Levinson CA, Vanzhula IA, Brosof LC, Forb-

15 Wang SB, Jones PJ, Dreier M, Elliott H, Grilo 

13. 

20 Hilbert A, Tuschen-Caffier B. Essanfälle und 

5 Borsboom D, Cramer AO. Network analysis: 

19 American Psychiatric Association. Diagnos-

26 Bohn K, Doll HA, Cooper Z, O'Connor M, 

17 Smith KE, Mason TB, Crosby RD, Cao L, 

17. 

31 Carver CS, White TL. Behavioral inhibition, 

35 Epskamp S, Fried EI. A tutorial on regularized 

33 Golino HF, Epskamp S. Exploratory graph analysis: A new approach for estimating the number of dimensions in psychological research. PLoS One. 2017 Jun;12(6):e0174035. 

38569. 

31. 

32 Strobel A, Beauducel A, Debener S, Brocke B. Eine deutschsprachige Version des BIS/BAS- Fragebogens von Carver und White. Z Differ Diagn Psychol. 2001;22(3):216–27. 

30. 

34 Fried EI, Cramer AO. Moving forward: chal-

56 Golino H, Shi D, Christensen AP, Garrido LE, 

28 Hautzinger M, Keller F, Kühner C. Beck Depres-

16. 

39 Forrest LN, Sarfan LD, Ortiz SN, Brown TA, 

25 Grunert SC. Ein Inventar zur Erfassung von 

28. 

24. 

22 Fairburn CG, Cooper Z. The Eating Disorder Examination. In: Fairburn CG, Wilson GT, editors. Binge eating: nature, assessment, and treatment. New York: Guilford Press; 1993. pp. 317–56. 

23. 

27 Beck AT, Steer RA, Brown GK. Manual for the Beck Depression Inventory-II. San Antonio (TX): Psychological Corporation; 1996. 

26. 

20. 

18 de Zwaan M, Herpertz S, Zipfel S, Svaldi J, 

21. 

19. 

13. 

15. 

14. 

13. 

12. 

11. 

10. 

9 Madhoo M, Levine SZ. Network analysis of depression and anxiety symptom relationships in a psychiatric sample. Psychol Med. 2016 Dec;46(16):3359–69. 

8 Madhoo M, Levine SZ. Network analysis of the quick inventory of depressive symptomatology: reanalysis of the STAR*D clinical trial. JAMA Psychiatry. 2018 Jan;75(1):98–100. 

7. 

6. 

5. 

4. 

3. 

2. 

1. 

0
45 Fairburn CG, Cooper Z, Shafran R. Cognitive behaviour therapy for eating disorders: a “transdiagnostic” theory and treatment. Behav Res Ther. 2003 May;41(5):509–28.
46 Murphy R, Strasbele S, Cooper Z, Fairburn CG. Cognitive behavioral therapy for eating disorders. Psychiatr Clin North Am. 2010 Sep;33(3):611–27.
47 Olatunji BO, Levinson C, Calebs B. A network analysis of eating disorder symptoms and characteristics in an inpatient sample. Psychiatry Res. 2018 Apr;262:270–81.
48 Brown TA, Vanzhula IA, Reilly EE, Levinson CA, Berner LA, Krueger A, et al. Body mistrust bridges interoceptive awareness and eating disorder symptoms. J Abnorm Psychol. 2020 Mar. doi: 10.1037/abn0000516.
49 DuBois RH, Rodgers RF, Franko DL, Eddy KT, Thomas JJ. A network analysis investigation of the cognitive-behavioral theory of eating disorders. Behav Res Ther. 2017 Oct;97:213–21.
50 De Paoli T, Fuller-Tyszkiewicz M, Huang C, Krug I. A network analysis of borderline personality disorder symptoms and disordered eating. J Clin Psychol. 2020 Apr;76(4):787–800.
51 Kober H, Boswell RG. Potential psychological & neural mechanisms in binge eating disorder: implications for treatment. Clin Psychol Rev. 2018 Mar;60:32–44.
52 Levinson CA, Vanzhula I, Brosof LC. Longitudinal and personalized networks of eating disorder cognitions and behaviors: targets for precision intervention a proof of concept study. Int J Eat Disord. 2018 Nov;51(11):1233–43.