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Understanding outcomes in family-based treatment for adolescent anorexia nervosa: a network approach

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Abstract

Background.—Family-based treatment (FBT) is the first-line treatment for adolescent anorexia nervosa (AN). Yet, remission is not achieved for about half of adolescents with AN receiving FBT. Understanding patient- and parent-level factors that predict FBT response may inform treatment development and improve outcomes.

Methods.—Network analysis was used to identify the most central symptoms of AN in adolescents who completed the Eating Disorder Examination (EDE) prior to FBT (N = 409). Bridge pathways between adolescent AN and parental self-efficacy in facilitating their child's recovery from AN were identified in a subset of participants (n = 184). Central and bridge symptoms were tested as predictors of early response (⩾2.4 kg weight gain by the fourth session of FBT) and end-of-treatment weight restoration [⩾95% expected body weight (EBW)] and full remission (⩾95% EBW and EDE score within 1 standard deviation of norms).

Results.—The most central symptoms of adolescent AN included desiring weight loss, dietary restraint, and feeling fat. These symptoms predicted early response, but not end-of-treatment outcomes. Bridge symptoms were parental beliefs about their responsibility to renourish their child, adolescent discomfort eating in front of others, and adolescent dietary restraint. Bridge symptoms predicted end-of-treatment weight restoration, but not early response nor full remission.

Conclusions.—Findings highlight the prognostic utility of core symptoms of adolescent AN. Parent beliefs about their responsibility to renourish their child may maintain associations between

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parental self-efficacy and AN psychopathology. These findings could inform strategies to adapt FBT and improve outcomes.

Keywords

adolescence; anorexia nervosa; family-based treatment; network analysis; prognosis

Anorexia nervosa (AN) is an eating disorder characterized by objectively low body weight, persistent behaviors that suppress or reduce weight (e.g. caloric restriction), and, commonly, body image disturbance (American Psychiatric Association, 2013). AN is among the most lethal of psychiatric disorders, due to death by suicide and medical complications, and is accompanied by significant morbidity and impairment (Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011; Udo & Grilo, 2018). AN commonly onsets in adolescence (Nagl et al., 2016) and long-term outcomes data suggest that, with treatment, many remit from the disorder, while a sizable fraction (~30%) develop a protracted course of illness (Lindstedt, Kjellin, & Gustafsson, 2017; Strober, Freeman, & Morrell, 1997). Identifying predictors of recovery is critical so that interventions can be adapted for those unlikely to recover. Here, we use network analysis to identify pretreatment patient- and parent-level factors central to adolescent AN and test whether these factors predict family-based treatment (FBT) outcomes.

FBT is the first-line manualized outpatient treatment for adolescent AN and is typically delivered in three phases (Lock & Le Grange, 2019). The first phase of FBT empowers parents to mobilize their strengths and resources to renourish their child. As weight restores and behavioral symptoms of AN remit, less parental oversight is typically required and FBT transitions to phase two, wherein parents gradually restore autonomy over eating to the adolescent. In the third (final) phase of FBT, therapeutic focus shifts to issues related to adolescent development that were interrupted by AN.

Although FBT is considered the treatment of choice for adolescent AN according to several guidelines (e.g. Couturier et al., 2020; National Institute for Health and Care Excellence, 2017), end-of-treatment remission rates are ~40% and few predictors of FBT outcomes have been identified (Lock & Le Grange, 2019). Early response – defined as the adolescent gaining ⩾2.4 kilograms (kg) by the fourth session of FBT – is a robust predictor of FBT outcome and is associated with a ~70% chance of remission (Doyle, Le Grange, Loeb, Doyle, & Crosby, 2010; Hughes, Sawyer, Accurso, Singh, & Le Grange, 2019; Le Grange, Accurso, Lock, Agras, & Bryson, 2014; Madden et al., 2015). To date, we have limited information about *pretreatment* patient- and parent-level factors that predict FBT outcomes. Information about such predictors may guide treatment adaptation and development, ultimately improving outcomes for patients with AN and preventing a protracted course of illness.

Emerging research suggests that pretreatment severity of eating disorder psychopathology (patient-level factor) and parental self-efficacy (parent-level factor) may predict FBT outcomes. Research has documented that increased pretreatment eating disorder psychopathology is associated with *better* response to FBT relative to other treatments, like adolescent-focused therapy (Hamadi & Holliday, 2020). Parental self-efficacy (i.e. perceived

ability to facilitate their adolescent's recovery from AN) may also be prognostically important in FBT, as parental self-efficacy has been associated with early response (Darcy et al., 2013) and end-of-treatment outcomes (Byrne, Accurso, Arnow, Lock, & Grange, 2015; Robinson, Strahan, Girz, Wilson, & Boachie, 2013). Moreover, parent ability to assume control over adolescent eating was identified as the strongest predictor of FBT outcome, above and beyond other key therapeutic features, such as externalization of AN from the adolescent and agnosticism about the cause of AN (Ellison et al., 2012). Together, these studies suggest that the severity of adolescent AN and parental self-efficacy may be influential over FBT outcomes.

Network analysis: a way to understand FBT outcomes

A novel way in which pretreatment predictors of FBT outcomes may be identified is network analysis, a statistical approach grounded in the network theory of psychopathology whereby clinical syndromes – such as AN – result from a complex, causal system of mutually reinforcing symptoms (Borsboom, 2008, 2017). Through the network lens, activation of an individual symptom (e.g. fear of weight gain) causes other symptoms to emerge (e.g. food rules, caloric restriction), giving rise to a clinical syndrome (e.g. AN). In network theory, *nodes* represent symptoms and are connected by *edges,* which represent statistical relationships among symptoms. Increased number and strength of edges among nodes promotes a 'contagion' effect, whereby symptom activation spreads throughout the network, ultimately contributing to the onset and maintenance of psychopathology (Cramer, Waldorp, van der Maas, & Borsboom, 2010).

Centrality

Network theory proposes that symptoms with the strongest connections to other symptoms are the most influential or central nodes of a network (Cramer et al., 2010). Central symptoms are thought to facilitate the onset and maintenance of a psychopathology. Network theory posits that central symptoms are preferential treatment targets, as decreasing the activation of central symptoms will weaken their associations to, and activations of, other symptoms (McNally, 2016). For instance, if caloric restriction is the most central AN symptom, prioritizing addressing this behavior in treatment may be important. Because central symptoms may predict course and treatment outcome (Borsboom & Cramer, 2013; Cramer et al., 2010; McNally, 2016), network theory provides a useful framework for identifying pretreatment predictors of FBT outcome.

Bridge pathways

Network analysis permits the modeling of key links (bridge pathways) between a psychopathological network and other processes (e.g. patient-level psychiatric comorbidity, parent-level self-efficacy, etc.) that may perpetuate or exacerbate the psychopathology (Cramer et al., 2010). For instance, a bridge pathway between parental self-efficacy and adolescent AN symptoms could provide insight into which features of parental self-efficacy are linked to adolescent AN and vice versa. Clinically, identification of a bridge pathway between parental self-efficacy and adolescent AN may be informative for treatment and provide clues for 'linchpin' parental factors that could be targeted.

The present study

The application of network science to eating disorders, in general (e.g. Levinson, Vanzhula, Brosof, & Forbush, 2018a), and AN, specifically (Calugi, Sartirana, Misconel, Boglioli, & Grave, 2020; Elliott, Jones, & Schmidt, 2020; Forrest, Jones, Ortiz, & Smith, 2018; Goldschmidt et al., 2018; Monteleone et al., 2019), is a burgeoning area of inquiry. Preliminary evidence suggests that central eating disorder symptoms predict treatment outcome in adolescent and adult AN (Brown et al., 2020; Elliott et al., 2020). Testing whether central symptoms (versus all or theoretically-selected symptoms) predict outcome has implications for the treatment of psychopathology in general. Central symptoms have the strongest connections to other symptoms in a psychopathological network and, when elevated, are thought to maintain psychopathology. If predictive models show that pretreatment elevations of central symptoms are associated with worsened outcome, clinical researchers would have reason to prioritize targeting central symptoms first in treatment as a means to reduce elevations of other symptoms. Research has yet to test whether central symptoms of adolescent AN predict FBT outcome. Moreover, no studies have examined bridge pathways between patient- and parent-level factors in adolescent AN and how bridge pathways predict FBT outcomes.

Thus, our first aim was to use network analysis to identify pretreatment central symptoms of adolescent AN and test the ability of these central symptoms to predict FBT outcomes (early response and end-of-treatment remission). Based on previous research, we hypothesized that dietary restraint, fear of weight gain, feeling fat, desiring weight loss, and body image concerns would be central to the pre-FBT adolescent AN network (Calugi et al., 2020; Elliott et al., 2020; Forrest et al., 2018) and predict the likelihood of early response and end-of-treatment remission (Elliott et al., 2020). Our second aim was to identify bridge pathways between adolescent AN and parental self-efficacy and test whether bridge pathways predicted FBT outcomes. Based on prior research, we hypothesized that parental beliefs about assuming control over adolescent eating would bridge adolescent AN psychopathology and parental self-efficacy.

Method

Participants and procedure

Participants were adolescents aged 12–18 years with *DSM-IV* (exclusive of amenorrhea) or *DSM*-5 AN and were treated with FBT in clinical trials (N = 409; NCT02054363, NCT03097874, NCT00149786, NCT00610753) (Agras et al., 2014; Lock, Agras, Biyson, & Kraemer, 2005, 2010, 2015, 2018; L'Insalata et al., 2020). Participants had not been previously treated with FBT. Clinical trials were approved by local Institutional Review Boards. Parents/caregivers and adolescents provided informed consent and assent, respectively.

Measures

Demographic information—Adolescents and their parents self-reported basic demographic information.

Anthropometric data—Adolescent height and body weight were collected throughout treatment. Percent expected body weight (%EBW) was calculated in reference to the 50th body mass index percentile based on age, height, weight, and sex (Le Grange et al., 2012).

Psychiatric comorbidity—Bachelors- or masters-level trained assessors (research interviewers) administered the Schedule for Affective Disorders and Schizophrenia for School-Age Children – Present and Lifetime version (K-SADS-PL) (Kaufman et al., 1997) to assess current psychiatric comorbidity. The K-SADS-PL has demonstrated strong psychometric properties in clinical adolescent samples (Jarbin, Andersson, Råstam, & Ivarsson, 2017; Kaufman et al., 1997).

Eating disorder symptoms—The Eating Disorder Examination (EDE) (Fairburn, Cooper, & O'Connor, 2008) was administered to adolescents at pretreatment and end-of-treatment by research interviewers. The EDE provides eating disorder behavior frequencies and assesses 23 cognitive eating disorder symptoms that comprise four distinct subscales [Restraint (five items), Eating Concern (five items), Shape Concern (eight items), and Weight Concern (five items)] and a global scale. Individual EDE items (behavior frequencies and all 23 cognitive symptoms) were used in network analysis. The current version of the EDE was used in each clinical trial (versions 12 thru 16). Internal consistency across pretreatment EDE subscales and the global score was acceptable to excellent (α's = 0.74–0.91) in this study.

Parental self-efficacy—The Parents Versus Anorexia scale (PVA) (Rhodes, Baillie, Brown, & Madden, 2005) was used to assess parental self-efficacy (i.e. parents' beliefs about their ability to renourish their child from AN). The PVA is a seven-item measure; each item is rated on a five-point Likert scale from 'strongly disagree' to 'strongly agree', with higher total scores corresponding to increased parental self-efficacy. Item content is presented in Table 2. The PVA demonstrated acceptable internal consistency in the validation study (α = 0.78) (Rhodes et al., 2005). Internal consistency of the PVA was low in our sample (α_{mothers} = 0.47, α_{fathers} = 0.41). Though low internal consistency may indicate heterogeneity within the PVA, individual PVA items (versus the total scale score) were used. The PVA was administered in three trials included in this study (n = 184). As the PVA was assessed for each parent participating in their child's FBT, parent responses to individual PVA items were averaged for each child (Byrne et al., 2015).

Early response and remission—Early response was operationalized as gaining ⩾2.4 kg from the first to fourth session of FBT (Le Grange et al., 2014).

End-of-treatment outcomes were: (1) weight restoration: ⩾95% EBW, and (2) full remission, i.e. weight restoration and end-of-treatment EDE global score within 1 standard deviation of norms. Both outcomes are considered benchmarks of remission and are empirically validated (Agras et al., 2014; Couturier & Lock, 2006).

Statistical analysis

Statistical analyses were conducted in R version 3.6.3 (R Development Core Team, 2013).

Missing data—In total, 2.21% of EDE and PVA items were missing at random and were imputed using the *mice* package (van Buuren & Groothuis-Oudshoorn, 2011). Early response and end-of-treatment outcomes were not imputed, nor were demographic or clinical characteristics.

Node selection—Nodes in the adolescent AN network were selected from the EDE, and parental self-efficacy/AN bridge network nodes were selected from the EDE and PVA. Rational methods were used to select the initial node pool, which was empirically refined. In the rational method, nodes were selected based on clinical judgment and previous network analyses (Elliott et al., 2020). All PVA items were retained in the rational phase, as the PVA has not been used in network analysis.

The 'goldbricker' function in the *networktools* package (Jones, 2017) was then used to empirically refine the node pools for the adolescent AN and parental self-efficacy/AN bridge networks. 'Goldbricker' identifies node pairs that are highly correlated and may represent redundant constructs, which can affect network estimation. The 'best_goldbricker' function determined which item to retain from redundant node pairs.

In the adolescent AN network, two EDE items were removed by 'best_goldbricker': dieting rules and discomfort seeing one's body (e.g. in mirrors). In the parental self-efficacy/AN bridge network, all PVA items were retained and the following EDE items were removed for redundancy: importance of control over eating in relation to self-evaluation, guilt around eating, fear of losing control over eating, dieting rules, and discomfort seeing one's body.

Network estimation—Networks were estimated using the *bootnet* package (Epskamp, Borsboom, & Fried, 2018) and network visualizations were created with the *qgraph* package (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012). Networks were estimated using the graphical least absolute shrinkage and selection operator (GLASSO), which estimates edges between nodes using regularized partial correlations. Networks were estimated using Spearman correlations.

Centrality indices—Centrality was assessed with expected influence, which is the sum of all the edges between one node and all other nodes, accounting for positive and negative edges (Robinaugh, Millner, & McNally, 2016). The 'centralityPlot' and 'centralityTable' functions in the *qgraph* package were used to compute expected influence metrics. We performed a node centrality difference test to statistically compare the centrality of each node against all other nodes in these networks.

Within the parental self-efficacy/AN bridge network, we identified bridge pathways by estimating bridge expected influence, which is the sum of connections between a node in one cluster and all nodes not in the same cluster (i.e. a node in parental self-efficacy cluster and all nodes in adolescent AN), accounting for positive and negative edges. We additionally performed bridge expected influence difference tests to statistically compare the bridge expected influence value of each pathway against all other bridge pathways. The 'bridge' function of the *networktools* package was used to identify bridge pathways between parental self-efficacy and adolescent AN clusters.

Network stability—Network stability was evaluated using the *bootnet* package. For the adolescent AN network, we computed stability coefficients for expected influence (EI-coefficient) and edge (ES-coefficient) parameters. For the parental self-efficacy/AN bridge network, we computed the aforementioned parameters and the bridge expected influence stability coefficient (BEI-coefficient). We used a case-dropping bootstrap, which determines the extent to which centrality indices change when cases are randomly dropped from the sample. The correlation coefficient between the original centrality index and the centrality index of the reduced sample is a metric of network stability. Research suggests that stability coefficients should be ⩾0.25, though ideally ⩾0.50 (Epskamp et al., 2018).

Prognostic utility of central and bridge symptoms—Logistic regression tested whether central symptoms from the pretreatment adolescent AN network and bridge pathways of the parental self-efficacy/adolescent AN network predicted early response and end-of-treatment outcomes. A composite score was created for central and bridge symptoms (Levinson & Williams, 2020). Linear regression tested the ability of central and bridge symptoms to predict continuous outcomes; these results are similar to logistic regression results and are presented in detail in the online Supplementary Materials. Duration of illness and prior treatment history were covariates in analyses, due to significant differences in outcomes as a function of these variables. In additional exploratory analyses, income was tested as a covariate and moderator (on central and bridge symptoms) of outcome (see online Supplementary Tables S5 and S6). Race and ethnicity were not tested as covariates or moderators because 69.4% of the sample identified as Caucasian and the remaining participants identified as one of five other categories (some categories only including <5 participants). We also did not wish to collapse non-Caucasian categories into one group, creating a Caucasian *v.* 'other' comparison. This would ignore the unique attributes of other races and suggest that non-Caucasian races are similar enough to be grouped together.

We additionally tested whether symptoms with higher expected influence or bridge expected influence had greater prognostic value. This procedure is described in the online Supplementary Materials.

Results

Participant demographic and clinical characteristics are presented in Table 1. Node descriptions are provided in Table 2.

Adolescent AN network

The pretreatment adolescent AN network was stable (EI-coefficient = 0.75, ES-coefficient = 0.68). Feeling fat (*feelfat*; EI = 1.20), dietary restraint (*restraint*; EI = 1.20), and desiring weight loss (*desirelose*; EI = 1.20) emerged as the most central symptoms. Feeling fat, dietary restraint, and desiring weight loss had a significantly higher expected influence than 70.37, 59.25, and 66.67% of other nodes, respectively. See Fig. 1 for a visualization of the pretreatment adolescent AN network and centrality plot; see online Supplementary Fig. S1 for the centrality difference test.

Parental self-efficacy/AN bridge network

The pretreatment parental self-efficacy/AN bridge network was stable (EI-coefficient = 0.75; BEI-coefficient = 0.34). Dietary restraint (EI =1.20), feeling fat (EI = 1.20), desiring weight loss (EI = 1.20), discomfort eating in front of others (EI = 1.20), and fear of weight gain (EI = 1.10) were the most central symptoms of the bridge network. Dietary restraint, feeling fat, and fear of weight gain had expected influence values significantly >65.63, 62.5, and 53.13% of all other nodes, respectively. Both discomfort eating in front of others and desiring weight loss had expected influence values >59.38% of all other nodes.

Discomfort eating in front of others (BEI = 0.19), dietary restraint (BEI = 0.12), and parental sense of responsibility to renourish their child (BEI = 0.11) emerged as bridge pathways. The bridge expected influence of discomfort eating in front of others was significantly >56.25% of all other nodes, whereas sense of parental responsibility to restore their child's weight and adolescent dietary restraint both had expected influence values significantly >18.75% of all other nodes. Parental sense of responsibility bridged to adolescent discomfort eating in front of others and weight importance. Adolescent dietary restraint bridged to parents feeling equipped with strategies to treat AN at home and parents feeling like they should understand the cause of AN before treating it. Adolescent discomfort eating in front of others bridged to parental trust of their own instincts in refeeding their child and parental sense of responsibility in renourishing their child. See Fig. 2 for a visualization of the pretreatment parental self-efficacy/AN bridge network and expected influence plots. Difference plots are available in online Supplementary Figs S2 and S3.

Prognostic utility of central and bridge symptoms

Logistic regression results are presented in Table 3 and described below. Supplemental linear regressions testing the ability of central and bridge symptoms to bridge continuous outcomes are presented in online Supplementary Table S1. Results testing the prognostic value of symptoms as a function of their (bridge) expected influence are presented in online Supplementary Tables S2-S4.

Adolescent AN network

Pretreatment central symptoms significantly predicted early response, such that as central symptom severity increased, so did the likelihood of early nonresponse. Results held when covarying for income. Pretreatment central symptoms did not predict remission, and this result was unchanged when covarying for income. Income did not moderate the effect of central symptoms on early response nor remission.

Parental self-efficacy/AN bridge network

Neither pretreatment central nor bridge symptoms of the bridge network predicted early response, and results remained when covarying for income.

Pretreatment central and bridge symptoms did not predict end-of-treatment full remission. However, pretreatment central and bridge symptoms both significantly predicted end-of-

treatment weight restoration, such that increased levels of central and bridge symptoms were associated with more favorable weight outcomes. Results held when covarying for income.

Income did not moderate the effect of central nor bridge symptoms on the outcome (early response, remission).

Discussion

Feeling fat, desiring weight loss, and dietary restraint emerged as the most important symptoms of adolescent AN, replicating previous network analyses in adolescent and adult AN (Calugi et al., 2020; Elliott et al., 2020; Forrest et al., 2018; Goldschmidt et al., 2018). Pretreatment elevations in these symptoms were associated with increased odds of early *nonresponse*. Yet, pretreatment elevation in these symptoms was unrelated to end-of-treatment remission, defined as both weight restoration and full remission (weight and EDE symptom remission). We interpret our findings as an optimistic indication that adolescents with a more severe initial presentation of core AN symptoms may not significantly differ from adolescents with less severe presentations in achieving end-of-treatment remission. We thus encourage clinicians to 'stay the course' with FBT when treating adolescents who present with elevated core AN symptoms.

To further examine the role of parents in the context of FBT, we characterized bridge pathways between adolescent AN and parental self-efficacy and examined the prognostic utility of these interacting patient- and parent-level factors. Parental beliefs about their responsibility to renourish their child from AN, adolescent dietary restraint, and adolescent discomfort eating in front of others were bridge pathways between parental self-efficacy and adolescent AN. These pathways bridging patient- and parent-level factors may provide insight into how these factors maintain one another and offer possible mechanisms of change via FBT. *Increased* parental sense of responsibility over renourishing their child and *elevated* adolescent dietary restraint and discomfort eating in front of others were predictive of weight restoration but were unrelated to early response. Although seemingly paradoxical, heightened parental responsibility in helping one's child to recover from AN may 'buffer' against core AN symptoms and help to promote weight restoration; though we note that weight restoration is a minimally stringent criterion for recovery (see Limitations). Supporting this idea, previous research found that parental ability to assume responsibility over their child's eating was the strongest predictor of remission from AN at the conclusion of FBT, above and beyond other therapeutic elements of FBT, like externalization of the illness (Ellison et al., 2012). Moreover, a thematic analysis revealed that FBT clinicians considered parental assumption of responsibility over preparing their child's food and supervising their child's eating to be the most critical element in FBT outcomes (Dimitropoulos, Freeman, Lock, & Le Grange, 2017). This conclusion should be tempered with the fact that bridge pathways predicted weight restoration and not full cognitive and weight remission. Nevertheless, research suggests that higher end-of-treatment weight, in and of itself, is associated with favorable long-term outcome in AN (Vail & Wade, 2015).

Strengths and limitations

Our study has several limitations. First, like many network analyses, we conducted a secondary data analysis and the original clinical trials were not designed with this study in mind. Second, the links between parental self-efficacy and adolescent AN are preliminary, due to the small sample size for network analysis (n = 184). Third, networks were modeled cross-sectionally and at the group level; central symptoms and their prognostic utility may not generalize to the individual level. Fourth, the generalizability of results is limited due to the sample's sociodemographic homogeneity. For instance, the majority of the sample identified as Caucasian and non-Hispanic, with parents reporting high levels of income. These factors may influence the family and adolescent's experience of AN and its treatment. For instance, past work has shown that affluence influences the perceived need for treatment and treatment-seeking (Forrest, Smith, & Swanson, 2017; Sonneville & Lipson, 2018). Fifth, little is known about factors that may influence parental self-efficacy in treating AN, such as paid family leave, job flexibility, extended family supports, and/or additional caregivers and we were unable to examine those factors here. Sixth, we used narrow remission definitions and though these definitions have empirical support, multidimensional remission definitions, which incorporate additional factors such as impairment and functioning (Tomba, Tecuta, Crocetti, Squarcio, & Tomei, 2019), may represent remission more holistically.

This study has several strengths. Few studies to date have used network analysis to understand the prognostic utility of central AN symptoms in relation to treatment outcomes and no previous studies have examined how central symptoms of adolescent AN relate to FBT outcome. This study is the first to identify bridge pathways between parent- and patient-level factors in adolescent AN, promoting improved understanding of how the interaction of these factors may contribute to FBT outcomes.

Clinical implications and directions for future research

Results have implications for health professionals who treat adolescent AN. Findings suggest that although increased pretreatment severity of central AN symptoms predicts early nonresponse, pretreatment symptom elevation did not relate specifically to remission after a course of standard FBT. As such, we encourage FBT clinicians to 'stay the course' of treatment, as it may be that adolescents with more severe initial presentations of AN improve more slowly or require a greater dose of FBT, rather than a different treatment. This idea is supported by previous research indicating that adolescents presenting with more severe pretreatment AN psychopathology had similar end-of-treatment outcomes as those with less severe pathology when receiving a greater dose of FBT (Lock et al., 2005).

Bolstering parental empowerment (self-efficacy) is considered the most salient principle of FBT and is deemed by clinicians to be critical in FBT outcome (Dimitropoulos et al., 2017). Our results lend further support to the idea that parental self-efficacy, reflected in the degree to which a parent believes it is their responsibility to renourish their child from AN, relates to FBT outcome. Thus, it is imperative that FBT providers identify parent-, patient-, and provider-level factors that affect parental self-efficacy and determine ways in which they can therapeutically enhance parental self-efficacy. Ongoing supervision and training to support the FBT clinician's efforts to increase parental sense of responsibility over re-nourishing

their child, without inducing blame and guilt, are critical. Additionally, helping clinicians to implement *in-vivo* strategies to enhance parental self-efficacy is crucial. For example, the family meal session provides opportunities for the clinician to directly bolster parental self-efficacy by supporting the parents in their goal of increasing their child's food consumption and disrupting AN behaviors. Indeed, therapeutic coaching of parents during the family meal session may enhance parental self-efficacy (Darcy et al., 2013; White et al., 2015).

Our results point to several future clinical-research directions. Results support investigations on incorporating intensive parental coaching in FBT phase one to enhance parental self-efficacy and promote favorable outcomes in adolescents who may not have been expected to remit from AN (i.e. early nonresponders) (Lock et al., 2015). Further research on parental support groups that aim to bolster self-efficacy as adjunctive interventions to FBT is warranted, as preliminary evidence supports the utility of such groups in enhancing parental self-efficacy (Spettigue et al., 2015) and promoting adolescent weight restoration (Rhodes, Baillee, Brown, & Madden, 2008). Additionally, future treatment research may use personalized, intraindividual networks prior to FBT to provide the clinician with information about specific elements of idiographic patient- and parent-level factors to target early in FBT (Levinson, Vanzhula, & Brosof, 2018b). Future research may also benefit from examining the prognostic utility of central and bridge symptoms on other end-of-treatment outcomes, like functional impairment, and using multidimensional remission definitions. Finally, assuring sociodemographic diversity within future research is critical to the further understanding of associations among sociodemographic characteristics and FBT outcome.

Results also have implications for treatment research extending beyond AN and FBT to other forms of psychopathology and their treatment Our results provide support for network theory's hypothesis that central symptoms maintain psychopathology and have prognostic value concerning treatment outcomes. Thus, future treatment research in eating disorders and other psychiatric disorders could use network analysis to identify central psychopathological symptoms prior to treatment, design interventions that target central symptoms, and test whether targeting central symptoms decreases the severity of other symptoms and promotes remission. Finally, the aforementioned personalized networks may also be extended to the treatment of other forms of psychopathology.

Conclusions

Results demonstrate the utility of using network analysis to understand the prognostic value of patient- and parent-level factors that may influence FBT outcomes. Although increased severity of core AN symptoms (feeling fat, desiring weight loss, dietary restraint) predicted early nonresponse, these pretreatment symptoms were unrelated to end-of-FBT remission. Moreover, the extent to which parents believed that it was their responsibility (versus their child's) to renourish their child from AN buffered against adolescent AN symptoms (including elevated restraint and fear of eating in front of others) and predicted end-of-FBT weight restoration, but not full remission. Results highlight the importance of clinical efforts to bolster parental self-efficacy and underscore the need to empirically test the efficacy of adaptations to FBT, including adjunctive treatments designed to enhance parental self-efficacy. Future research investigating the impact of evidence-based interventions

and adaptations on clinical improvement and remission is vitally important to enhance therapeutic outcomes and improve the lives of patients and families affected by AN.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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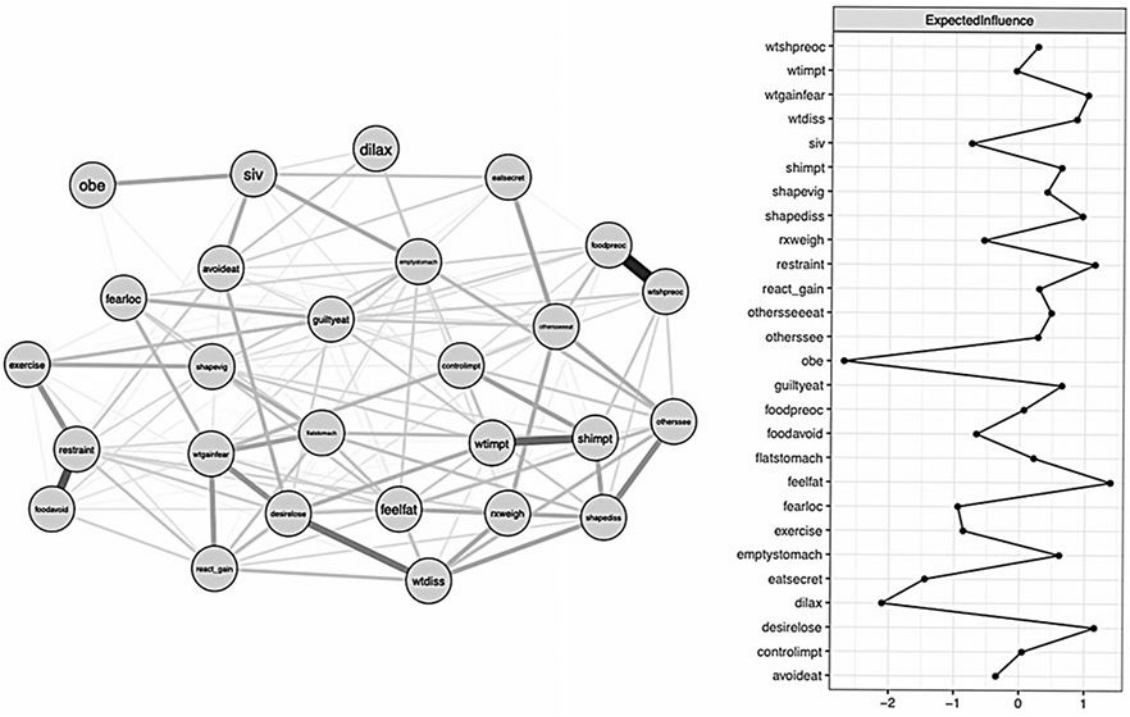

Fig. 1.

Adolescent AN network visualization and expected influence plot. *Note.* Node names are presented in Table 2. Expected influence plots the centrality of nodes via *z*-scores along the *x*-axis.

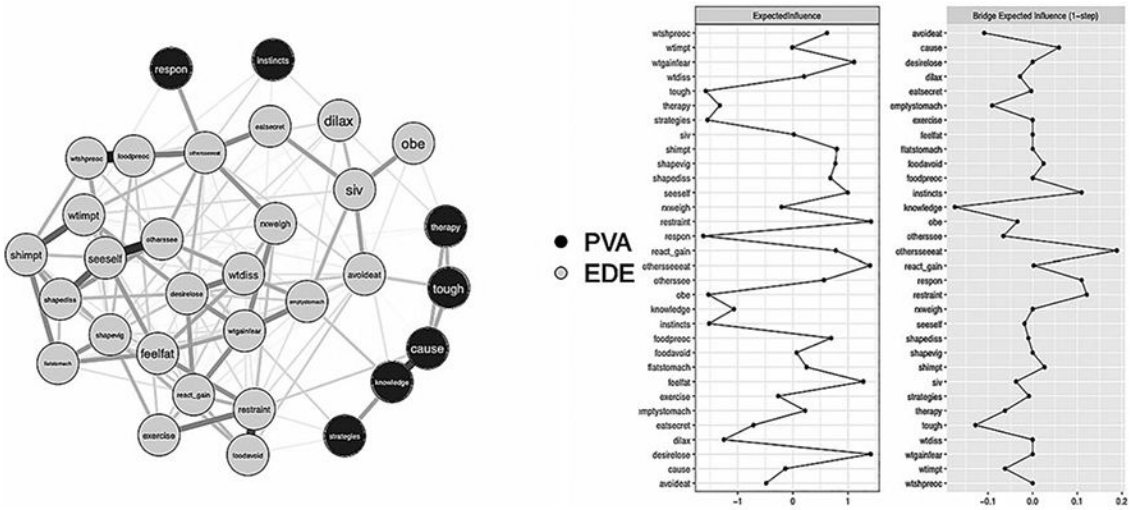

Fig. 2.

Parental self-efficacy/AN bridge network visualization and expected influence plots for central and bridge symptoms. *Note.* Expected influence plots the centrality of nodes via *z*-scores along the *x*-axis. Bridge expected influence (1-step) plots the influence of bridge pathways via *z*-scores on the *x*-axis.

Table 1.

Sample demographic and clinical characteristics

Characteristic	Mean (S.D.) or *n* (%)
Child age (in years)	14.68 (1.63)
Child sex	
Female	274 (67%)
Male	135 (33%)
Other	–
Child race[a]	
Caucasian	284 (69.4%)
African American/Black	3 (0.7%)
Asian	51 (12.5%)
American Indian/Alaska Native	1 (0.2%)
Native Hawaiian/Pacific Islander	19 (4.6%)
Multi-racial	31 (7.6%)
Child ethnicity[a]	
Hispanic/Latinx	49 (12.0%)
Non-Hispanic/non-Latinx	284 (69.4%)
Intact family[b]	
Yes	248 (60.6%)
No	74 (18.1%)
Family income	
<US$50 000	49 (12.0%)
US$50 000–80 000	46 (11.2%)
US$81 000–100 000	57 (13.9%)
US$101 000–150 000	80 (19.6%)
>US$150 000	160 (41.1%)
AN duration (in months)	10.67 (9.61)
Pretreatment EDE global score	2.02 (1.47)
Pretreatment %EBW	82.89 (4.27)
Psychiatric comorbidities[c,d]	
Major depressive disorder	71 (17.4%)
Dysthymia	4 (0.97%)
Depression NOS	31 (7.6%)
Generalized anxiety disorder	53 (13%)
Social phobia	5 (1.22%)
Separation anxiety	3 (0.73%)
Specific phobia	2 (0.49%)

Characteristic	Mean (S.D.) or *n* (%)
Panic disorder	8 (1.96%)
Agoraphobia	2 (0.49%)
Anxiety disorder NOS	8 (1.96%)
Post-traumatic stress disorder	3 (0.73%)
Obsessive-compulsive disorder	23 (5.62%)
Attention-deficit/hyperactivity disorder	5 (1.2%)
Oppositional defiant disorder	3 (0.73%)
Adjustment disorder	3 (0.73%)
Any psychiatric comorbidity[d]	
Yes	158 (38.6%)
No	171 (41.8%)
Prior treatment (including inpatient medical stabilization for AN)[e]	
Yes	303 (74.1%)
No	82 (20.0%)

%EBW is percent expected body weight based on normative data for age, sex, height, and current weight; EDE, Eating Disorder Examination; NOS, not otherwise specified; US$, United States dollars.

[a]Certain participant demographics are missing due to changes in National Institute of Health race and ethnicity reporting standards over time.

[b]Intact family data missing for 87 participants.

[c]Total percentage of psychiatric comorbidities exceeds 100% because some participants reported two or more psychiatric comorbidities.

[d]Baseline psychiatric comorbidity data missing for 80 participants.

[e]Treatment history data missing for 24 participants.

Table 2.

Node abbreviations and descriptions

Node abbreviation	Node description
EDE	
avoideat	Going eight or more waking hours without eating
controlimpt	Importance of strict control over eating in self-evaluation
desirelose	Strong desire to lose weight
dietrules	Rules around food and dieting
dilax	Diuretic, laxative, and/or diet pill misuse episodes
eatsecret	Eating in secret
emptystomach	Desire to have an empty stomach
exercise	Excessive exercise episodes
fearloc	Fear of losing control over eating
feelfat	Feeling fat
flatstomach	Desire to have a flat stomach
foodavoid	Avoidance of certain foods
foodpreoc	Thinking about food, eating, calories
guiltyeat	Felt guilty after eating
obe	Objectively large binge eating episodes
otherssee	Discomfort with others seeing body
othersseeeat	Discomfort eating in front of others
react_gain	Reaction to weight gain
restraint	Dietary restraint
rxweigh	Reaction to prescribed weighing
shapediss	Dissatisfaction with shape
seeself	Discomfort seeing oneself
shapevig	Vigilance about shape
shimpt	Importance of shape in self-evaluation
siv	Self-induced vomiting episodes
wtdiss	Dissatisfaction with weight
wtgainfear	Magnitude of fear of weight gain
wtimpt	Importance of weight in self-evaluation
wtshpreoc	Thinking about shape or weight interferes with daily activities
PVA	
cause	Parents should explore the cause of AN before being involved in its treatment
instincts	Parental instincts are a better guide for helping the child to recover from AN than the expertise of professionals
knowledge	Parents have knowledge to achieve victory over AN
respon	Parents have more responsibility than the child to restore child’s weight
strategies	Parent feels equipped with strategies to treat AN at home

Node abbreviation	Node description
therapy	Child with AN needs individual therapy to get better
tough	Being tough on the child with AN will cause too much trauma and distress

EDE, Eating Disorder Examination; PVA, Parent Versus Anorexia scale. EDE symptoms were assessed over the past 28 days. PVA and EDE items (not subscales) were used in network analysis.

Table 3.

Central and bridge symptoms predicting FBT outcomes

Outcome	β (S.E.)	Wald's *z*-statistic	OR (95% CI)
Adolescent AN network			
Early response (0 = yes, 1 = no)			
Intercept	0.065 (0.046)	1.993 *	1.907 (1.021–3.660)
Pretreatment central symptoms	**0.047 (0.029)**	**2.182 ***	**1.048 (1.006–1.094)**
Prior treatment			
No (reference group)			
Yes	−0.169 (0.595)	−0.531	0.845 (0.444–1.552)
Duration of illness	0.016 (0.259)	1.129	1.016 (0.990–1.048)
EOT remission (0 = yes, 1 = no)			
Intercept	−0.664 (0.349)	−1.904	0.515 (0.255–1.007)
Pretreatment central symptoms	0.022 (0.023)	0.867	1.022 (0.974–1.074)
Prior treatment			
No (reference group)			
Yes	0.980 (0.323)	3.036 **	2.663 (1.419–5.047)
Duration of illness	0.043 (0.019)	2.256 *	1.044 (1.008–1.087)
EOT weight restoration (0 = yes, 1 = no)			
Intercept	−0.497 (0.336)	−1.486	0.608 (0.311–1.162)
Pretreatment central symptoms	−0.030 (0.023)	−1.291	0.971 (0.928–1.015)
Prior treatment			
No (reference group)			
Yes	0.562 (0.320)	1.760	1.754 (0.941–3.303)
Duration of illness	0.048 (0.018)	2.755 **	1.050 (1.016–1.089)
Parental self-efficacy/AN bridge network			
Early response (0 = yes, 1 = no)			
Intercept	0.655 (0.465)	1.410	1.926 (0.786–4.962)
Pretreatment *central* symptoms	0.010 (0.012)	0.537	1.010 (0.973–1.051)

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Outcome	*β* (S.E.)	Wald's *z*-statistic	OR (95% CI)
Prior treatment			
No (reference group)			
Yes	−0.013 (0.464)	−0.028	0.987 (0.383–2.404)
Duration of illness	0.008 (0.022)	0.354	1.008 (0.967–1.058)
Early response (0 = yes, 1 = no)			
Intercept	0.607 (0.541)	1.121	1.835 (0.643–5.477)
Pretreatment *bridge* symptoms	0.020 (0.046)	0.420	1.020 (0.931–1.118)
Prior treatment			
No (reference group)			
Yes	−0.016 (0.470)	−0.035	0.984 (0.377–2.425)
Duration of illness	0.009 (0.022)	0.385	1.009 (0.968–1.059)
EOT remission (0 = yes, 1 = no)			
Intercept	−0.601 (0.506)	−1.187	0.548 (0.196–1.456)
Pretreatment *central* symptoms	−0.009 (0.022)	−0.409	0.991 (0.950–1.035)
Prior treatment			
No (reference group)			
Yes	0.773 (0.465)	1.662	2.166 (0.875–5.504)
Duration of illness	0.041 (0.026)	1.555	1.042 (0.992–1.102)
EOT remission (0 = yes, 1 = no)			
Intercept	−0.502 (0.589)	−0.851	0.605 (0.185–1.898)
Pretreatment *bridge* symptoms	−0.023 (0.049)	−0.466	0.977 (0.887–1.076)
Prior treatment			
No (reference group)			
Yes	0.772 (0.463)	1.666	2.165 (0.878–5.483)
Duration of illness	0.040 (0.026)	1.509	1.041 (0.991–1.101)
EOT weight restoration (0 = yes, 1 = no)			
Intercept	−0.641 (0.512)	−1.252	0.527 (0.186–1.415)
Pretreatment *central* symptoms	**−0.046 (0.022)**	**−2.053***	**0.955 (0.913–0.997)**
Prior treatment			

Outcome	β (S.E.)	Wald's z-statistic	OR (95% CI)
No (reference group)			
Yes	0.404 (0.472)	0.854	1.498 (0.597–3.872)
Duration of illness	0.068 (0.028)	2.481 *	1.071 (1.018–1.134)
EOT weight restoration (0 = yes, 1 = no)			
Intercept	−0.255 (0.587)	−0.435	0.775 (0.239–2.431)
Pretreatment *bridge* symptoms	**−0.010 (0.049)**	**−2.012 ***	**0.905 (0.820–0.996)**
Prior treatment			
No (reference group)			
Yes	0.380 (0.472)	0.805	1.462 (0.584–3.773)
Duration of illness	0.063 (0.027)	2.317 *	1.065 (1.012–1.127)

CI, confidence interval; EOT, end-of-treatment; OR, odds ratio; S.E., standard error.

Adolescent AN network central symptoms were feeling fat, dietary restraint, and desiring weight loss. Parental self-efficacy/AN network central symptoms were dietary restraint, feeling fat, desiring weight loss, discomfort eating in front of others, and fear of weight gain. Parental self-efficacy/AN network bridge symptoms were adolescent discomfort eating in front of others, adolescent dietary restraint, and parental sense of responsibility in renourishing their child with AN.

Early response is defined as an adolescent gaining at least 2.4 kg by the fourth session of FBT. End-of-treatment remission is defined as an adolescent being at least 95% of expected body weight for height, age, and sex and having an EDE global score within 1 standard deviation of norms at end-of-treatment. End of treatment weight restoration is defined as an adolescent being at least 95% of expected body weight for height, age, and sex at end-of-treatment.

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$.

Bolding indicates that the composite of central or bridge symptoms significantly predicted outcome.