Locating the mechanisms of therapeutic agency in family-based treatment for adolescent anorexia nervosa: A pilot study of clinician/researcher perspectives

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Locating the mechanisms of therapeutic agency in family-based treatment for adolescent anorexia nervosa: A pilot study of clinician/researcher perspectives

Stuart B. Murray\textsuperscript{a}, Devin Rand-Giovannetti\textsuperscript{b}, Scott Griffiths\textsuperscript{c}, and Jason M. Nagata\textsuperscript{d}

\textsuperscript{a}Department of Psychiatry, University of California, San Francisco, CA, USA; \textsuperscript{b}Department of Psychology, University of Hawai‘i at Mānoa, Honolulu, HI, USA; \textsuperscript{c}Melbourne School of Psychological Sciences, University of Melbourne, Melbourne, VIC, Australia; \textsuperscript{d}Division of Adolescent and Young Adult Medicine, University of California, San Francisco, CA, USA

ABSTRACT

The theoretical agnosticism of family-based treatment (FBT) has precluded a thorough understanding of treatment mechanisms. We examined clinician and researcher perspectives on the mechanisms of FBT. Thirty-eight clinicians/researchers who had recently published in the domain of anorexia nervosa (AN) indicated their opinions as to the mechanisms of FBT, and content analysis was performed to identify relevant themes. Parental input was deemed crucial for weight-based symptom remission, and weight restoration was deemed crucial for cognitive symptom relief. Participants reported that mechanisms of FBT may be nullified by psychiatric comorbidities (50%), inappropriate weight goals (37%), or a long illness duration (24%). Attributions of causality to parents (66%) or family system factors (37%) were noted in instances of nonsuccessful outcomes. These findings offer important insights into clinician/researcher perspectives on the mechanisms of FBT and highlight beliefs among clinicians and researchers that may not reflect emerging evidence, or the theoretical framework of FBT.

Clinical Implications

- Despite it’s position as an evidence-based treatment for adolescent AN, little evidence to date has explicated the therapeutic mechanisms of FBT. An understanding of treatment mechanisms is crucial as efforts to optimize FBT continue.
- We conducted a content analysis of clinician and researcher perspectives as to the primary mechanisms of FBT, in instances of (i) full symptom remission, (ii) partial treatment response, and (iii) no treatment response.
- Results indicate important beliefs among clinicians and researchers as to the mechanisms of FBT, some of which diverge markedly from the empirical evidence base. These findings therefore offer important implications for the dissemination of FBT.
Family-based treatment (FBT) is an empirically supported treatment for medically stable adolescent anorexia nervosa (AN) of short duration. This exclusively behavioral treatment centrally rests on the notion that parents (i) instinctively know how to feed healthy children and (ii) can be empowered to directly intervene in their child’s behavioral symptoms of AN (Lock & Le Grange, 2013). Treatment is therefore characterized by concerted therapeutic attempts to leverage and mobilize parental efforts to adopt a leadership role in addressing their child’s AN-related behavior. To date, FBT has been the focus of several large-scale randomized clinical trials, and typically results in end-of-treatment remission rates of approximately 30% (Lock, 2015). As such, a clear and important need relates to augmentation of treatment outcomes in FBT.

In improving the efficacy of any treatment modality, broader psychotherapy research posits that treatment augmentations are best undertaken in the context of a deep and synthesized understanding of the precise mechanisms of change of a given treatment (Kazdin, 2001). Herein lies a significant challenge in the context of FBT, as the theoretical agnosticism adopted throughout treatment has precluded specific hypothesizing as to the agent of therapeutic action that facilitates symptom remission. Moreover, the exclusive behavioral focus and weight-related goals in FBT has precluded precise theorizing as to the treatment mechanisms leading to reduced cognitive AN psychopathology.

In elucidating treatment mechanisms, clinician and researcher perspectives on treatment mechanisms and barriers have resulted in important research advances in other treatment modalities (Baker-Ericzén, Jenkins & Schlagel, 2013). Further, clinician perspectives have illustrated important barriers to the dissemination of FBT (Couturier et al., 2013). Thus, the aim of the present study was to explore clinician and researcher opinions as to the potential mechanisms of therapeutic agency throughout FBT. Specifically, we aimed to investigate clinician and researcher perspectives as to the mechanism of change in relation to both (i) weight-based and (ii) cognitive symptom remission during FBT, in addition to the factors that impinge upon these proposed mechanisms in instances of (iii) partially successful outcomes, and (iv) unsuccessful outcomes.

**Method**

**Participants**

Participants were researchers and clinicians who had recently published scientific articles relating to AN. We identified authors via a keyword search of “adolescent anorexia nervosa treatment” on PubMed, and an exhaustive list of corresponding authors who had published articles in this domain in
From this list, we attempted to contact 150 randomly selected corresponding authors who had provided contact information for an article published in English. Of the authors contacted, 61 initiated the survey, two were deemed ineligible because they indicated they were “not at all” knowledgeable about FBT, and 38 completed the survey. An additional two responders declined to participate on the grounds that they felt unable to answer the questions. Upon agreeing to participate, each participant was asked to provide their thoughts relating to four key questions: (i) what are the primary mechanisms by which FBT exerts a therapeutic effect on weight-based AN symptoms throughout treatment? (ii) what are the primary mechanisms by which FBT exerts a therapeutic effect on cognitive AN symptoms throughout treatment? (iii) why might cognitive symptoms persist in instances of a partial remission (defined as full weight restoration, but ongoing cognitive symptoms)? and (iv) when FBT does not bring about a successful outcome (defined as nonrestored weight and no remission of cognitive symptoms), why might the mechanisms you outlined not lead to symptom change? In all instances, participants were asked to assume a suitable and motivated family when responding. This research was considered exempt from the Institutional Review Board at the University of California, San Francisco Committee on Human Research.

**Analyses**

To analyze these data, participants’ written responses for each of the four key research questions were de-identified and collated, and independent content analyses were performed by two members of the team (SBM, DRG). Given the dearth of literature relating to the proposed mechanisms of change throughout FBT, no *a priori* variables or categories were developed before coding. As such, a general inductive approach was adopted for raw data analysis (Thomas, 2006), which entailed preliminary in-depth analyses of the raw content, such that frequently occurring themes and categories could be identified.

Following these preliminary analyses, emergent coding methods (Harshbarger, Ahlers-Schmidt, Mayans, Mayans, & Hawkins, 2009) were utilized and the research team created a codebook to log frequently occurring thematic categories, together with category exemplars. The internal consistency of the coding was ensured by performing all coding separately, with consensus reached through discussion when needed. Inter-rater reliability between the two data coders, as determined by Cohen’s kappa, was .87. Further, an additional member of the research team not involved in the coding process (SG) reviewed each thematic category across the answers provided by participants for each key research question, in order to confirm that each theme was evident.
Results

Participants (N = 38) were 78.2% female with a mean age of 46.2 years (SD = 11.4), ranging from 29 to 70 years. Participants were psychologists (n = 16), psychiatrists (n = 9), physicians (n = 6), and other health professionals (n = 7) with a mean length of practice of 18.9 years (SD = 10.3), ranging from 2 to 38 years. Respondents reported specializing in children and/or adolescents (n = 22), adults (n = 5), and both adults and children/adolescents (n = 11). On average, participants reported spending 40.6% of their time on clinical work and 56.8% of their time on research work, with estimates ranging from 0 to 95% clinical work and 5 to 100% research work. Across participants, 84.4% reported that eating disorders were their main area of clinical/research focus. Of those who specialized in eating disorders, the average length of their specialization was 15.2 years (SD = 10.4), ranging from 2 to 40 years. Most participants considered themselves to be very knowledgeable or expert in the area of adolescent AN (65.3%), while the remainder identified themselves as being either knowledgeable (28.9%) or somewhat knowledgeable (5.8%). Most participants also indicated that they were very knowledgeable or expert in their understanding of FBT (68.2%), with the rest reporting being knowledgeable (28.6%) or somewhat knowledgeable (3.2%). Of those participants who reported treating adolescent AN (n = 33), 87.8% stated FBT would be their first-line of treatment. Other first-line treatments included CBT (6.1%) and behavior therapy (6.1%). The remaining participants (n = 5) stated that they do not directly provide treatment for adolescent AN.

Themes from the reported mechanisms for weight and cognitive symptom remission in FBT (Table 1) and partial or nonresponses in FBT (Table 2) are summarized.

Discussion

Despite the widespread uptake of FBT, and its position as a leading treatment option for adolescent AN, little evidence has explicated the mechanisms of therapeutic agency. This study illustrates clinician and researcher perspectives on potential mechanisms of change throughout FBT, in relation to both weight and cognitive symptoms of AN throughout treatment, in addition to illustrating factors implicated in nonsuccessful outcomes.

In terms of weight-based symptom remission during FBT, the vast majority of clinicians and researchers centrally implicated parental input as the crucial mechanism. More than three quarters of respondents report parental efficacy and empowerment to be the primary driver of weight restoration, and approximately 55% noted the centrality of meal support to this end. This assertion is empirically well supported, as effective meal support strategies
are known to improve outcome in FBT (White, Haycraft, & Madden et al., 2017), and parental efforts are typically correlated with a favorable outcome in FBT (Ellison et al., 2012).

In terms of cognitive symptom relief, most clinicians and researchers deemed weight restoration to be the primary agent of cognitive symptom relief, via its impact on alleviating state-related cognitive rigidity. Certainly, long-standing lines of evidence have suggested elevated state-related cognitive rigidity in those with AN during starvation (Keys, Brožek, Henschel, Mickelsen, & Taylor, 1950; Szmukler et al., 1992), although the degree to which this dissipates upon weight restoration remains unclear (Danner et al., 2012; Roberts, Tchanturia, & Treasure, 2010). Moreover, little evidence to date suggests that enhanced cognitive flexibility, in and of itself, is sufficient to ameliorate the complex cognitive symptoms of AN. In fact, robust evidence suggests that many symptoms persist after weight restoration,
including body dissatisfaction (Keel, Dorer, Franko, Jackson, & Herzog, 2005), a fear of weight gain (Murray et al., 2016b), and an avoidance of calorie-dense foods (Schebendach et al., 2008). Further, inpatient refeeding protocols are well known to effectively restore patient weight, but typically do not alter cognitive symptoms (Fennig, Klomek, Shahar, Sarel-Michnik, & Hadas, 2015). As such, while weight restoration may temporally precede cognitive symptom change in FBT (Accurso, Ciao, Fitzsimmons-Craft, Lock, & Le Grange, 2014), the notion that weight gain mechanistically causes cognitive symptom remission remains underdeveloped. Thus, while weight restoration is the first goal of treatment, and by definition comes before cognitive symptom recovery, this in and of itself does not cause cognitive symptom recovery.

**Table 2.** Summary of reported factors implicated in partial or nonresponses in FBT.

<table>
<thead>
<tr>
<th>Description</th>
<th>Example</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Comorbidity</strong></td>
<td>“Additional psychopathology (i.e., depression, anxiety, obsessionality) contribute to the stickiness of cognitive symptoms”</td>
<td>19 (50%)</td>
</tr>
<tr>
<td></td>
<td>“Ongoing self-esteem issues lead to ongoing AN cognitions”</td>
<td></td>
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<tr>
<td></td>
<td>“Continued, active symptoms can be associated with various comorbidities including perfectionism, depression, neuroticism and anxiety”</td>
<td></td>
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<tr>
<td><strong>Weight status</strong></td>
<td>“Most partial remission cases could be fixed by setting a higher goal weight”</td>
<td>14 (36.8%)</td>
</tr>
<tr>
<td></td>
<td>“Insufficient weigh restoration can lead to ongoing cognitive symptoms”</td>
<td></td>
</tr>
<tr>
<td><strong>Illness characteristics</strong></td>
<td>“Cognitive symptoms may persist because the eating disorder is of unusually long duration”</td>
<td>9 (23.7%)</td>
</tr>
<tr>
<td></td>
<td>“Often in these instances, cognitions have been longstanding, entrenched core beliefs, that unless addressed in individual therapy, remain stuck”</td>
<td></td>
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<tr>
<td><strong>Parental factors</strong></td>
<td>“Parents are often very tired and willing to cave more to the idea that their kid is ‘less sick’, even if not fully well”</td>
<td>7 (18.4%)</td>
</tr>
<tr>
<td><strong>Treatment factors</strong></td>
<td>“The therapist needs to be made of steel for this treatment to work”</td>
<td>3 (7.9%)</td>
</tr>
<tr>
<td></td>
<td>“Incomplete exposure to feared foods promote ongoing fears”</td>
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Interestingly, approximately 30% of participants noted the role of exposure and response prevention processes in promoting cognitive symptom remission in FBT. Despite FBT making no reference to exposure-related processes, an elegant model put forth by Hildebrandt and colleagues (2012) outlines how FBT may be conceptualized as a parent-driven and ecologically valid (i.e., in the home context) form of exposure and response prevention. However, exposure-based approaches to the treatment of AN may be hindered by the imprecise delineation of feared cues versus feared outcomes in AN, which nullifies attempts to precisely violate threat expectancies throughout exposures (Murray, Loeb, & Le Grange, 2016b; Murray et al., 2016a). For instance, it is unclear as to whether the feared outcomes in AN relate to food consumption, weight normalization, exponential weight gain, interoceptive physiological states, or a deviation from one’s self-concept as adherent to the thin ideal (2016b; Murray et al., 2016a). This variability in potential feared outcomes in AN is precipitated by a differing array of feared cues, and this largely unknown nature of the core fear associations in AN renders exposure treatments diffuse and nonprecise in their targets. As such, systematic attempts to delineate the precise nature of fear and anxiety pathways in AN may allow for targeted attempts to optimize exposure processes throughout FBT.

In the context of partially successful outcomes, respondents reported that the mechanisms of FBT may be nullified by psychiatric comorbidities, an insufficient weight goal, or specific illness characteristics such as a long duration of illness. Indeed, while comorbidities are associated with more complex eating disorder presentations (Hughes et al., 2013), moderator studies consistently show that this does not nullify treatment outcome in FBT (Le Grange, Lock, & Agras et al., 2012; 2016). This may be an important misconception among clinicians and researchers. In contrast, the assertion that illness duration impacts treatment response is supported in moderator analyses (Le Grange et al., 2012; 2016). The notion that insufficient weight gain precludes cognitive symptom recovery reflects the belief that weight gain causes cognitive symptom relief. Clearly, one cannot recover from AN in an underweight state. However, the notion that weight gain in itself causes cognitive symptom recovery remains both counterintuitive and under-supported by evidence. If one accepts the notion that cognitive symptoms (e.g., drive for thinness, fear of weight gain) drive the behavioral symptoms of AN (e.g., dietary restriction), which in turn drives weight loss, the notion that greater weight status causes improved cognitive symptoms remains an oversimplification of a complex array of cognitive symptoms, which is, in many instances, originally developed in non-underweight settings.

Last, in instances of nonsuccessful outcomes, an attribution of causality to parent or family systems factors was apparent. This notion of holding family or parental pathology culpable for treatment nonsuccess is antithetical to the
theoretical frame of FBT and is of concern when considering that approximately 70% of those engaging in FBT may not experience full remission (Lock, 2015). The propensity among parents to blame themselves for their child’s illness is well documented (Whitney, Murray, Gavan, & Todd, 2005), and may be powerfully underscored by implicit assumptions from clinicians that parents have been unable to execute the treatment. Indeed, while FBT aims to theoretically conceptualize parents as the experts in the treatment of their child, an important area to target in the dissemination of FBT may relate to the avoidance of parent blaming in instances of treatment nonsuccess.

Important limitations should be noted. The low response rate and concomitant small sample size necessitates appropriate caution in interpreting these findings; however, several of the themes reached saturation. Similarly, response bias ought to be considered, and the extent to which these findings are representative of all clinicians/researchers practicing FBT is unknown. While all respondents reported being knowledgeable in the areas of adolescent AN and FBT, not all reported having practiced as clinicians. Similarly, the extent to which these findings extend to clinicians not practicing in academic settings is unknown. Notwithstanding, the perspectives from clinicians and researchers in this study identify potential mechanisms of FBT that are deserving of empirical pursuit. For example, exposure principles may be an important mechanistic pathway for the treatment of cognitive AN symptoms. Further, these findings highlight particular beliefs among clinicians/researchers that may not be reflected in emerging evidence and/or the theoretical underpinnings of FBT, such as implicating parental factors if FBT is not successful. This research provides clear targets to address in the ongoing dissemination of FBT, and offers potential targets for ongoing attempts to optimize outcomes in FBT.

References


