RESEARCH ARTICLE

'I Know I Can Help You': Parental Self-efficacy Predicts Adolescent Outcomes in Family-based Therapy for Eating Disorders

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Abstract

Family-based therapy is regarded as best practice for the treatment of eating disorders in adolescents. In family-based therapy, parents play a vital role in bringing their child or adolescent to health; however, little is known about the parent-related mechanisms of change throughout treatment. The present study examines parent and adolescent outcomes of family-based therapy as well as the role of parental self-efficacy in relation to adolescent eating disorder, depressed mood and anxiety symptoms. Forty-nine adolescents and their parents completed a series of measures at assessment, at 3-month post-assessment and at 6-month follow-up. Results indicate that, throughout treatment, parents experienced an increase in self-efficacy and adolescents experienced a reduction in symptoms. Maternal and paternal self-efficacy scores also predicted adolescent outcomes throughout treatment. These results are consistent with the philosophy of the family-based therapy model and add to the literature on possible mechanisms of change in the context of family-based therapy. Copyright © 2012 John Wiley & Sons, Ltd and Eating Disorders Association.

Introduction

Eating disorders (EDs) are the third most common chronic disease among adolescent females after asthma and obesity (Golden et al., 2003). Approximately 0.5% of the population in Canada suffers from anorexia nervosa (AN), 1% from bulimia nervosa (BN) and 3%–5% from other disordered eating patterns severe enough to warrant clinical attention (Garfinkel et al., 1995, 1996). The most exhaustive review of the outcomes of lifetime AN in the 20th century revealed a mean crude mortality rate of 5%, and in the surviving patients, full recovery was found in less than a half of the sample (Steinhausen, 2002). In terms of BN, Steinhausen and Weber (2009) also reported on global outcomes, with only 45% achieving full recovery across studies. Given that EDs also pose particular treatment challenges, it is important to intervene early in the progression of the illness to prevent the possibility of more chronic and treatment-resistant forms of AN and BN from developing.

Family-based therapy has been tested in the context of randomized clinical trials with adolescents suffering from AN and, more recently, from BN (Loeb & Le Grange, 2009 for a review). Currently regarded as best practice for the treatment of adolescent EDs, family-based therapy assigns parents a pivotal role in helping their adolescent recover (Le Grange, 1999; Le Grange & Eisler, 2009).

During Phase 1 of the treatment, parents are responsible for refeeding their adolescent, halting unhealthy behaviours such as purging, and re-establishing normal eating patterns. Later in the recovery process (Phase 2), parents navigate the process of slowly returning control of eating to their adolescent and helping him or her return to age-appropriate activities (Le Grange, 1999; Le Grange, Lock, & Dymek, 2003). In Phase 3, the focus is on adolescent and/or family issues that were initially set aside. Although the family-based therapist works to guide parents through this process, the primary responsibility for recovery falls upon parents. Specifically, one important goal of family-based therapy is to help parents use their existing skills and resources to combat the ED (Le Grange & Eisler, 2009). As such, it is extremely important that parents understand and feel capable of fulfilling this role and that they develop a sense of self-efficacy vis-à-vis the tasks ahead.

Recent research with adult ED populations has explored the impact of skill-based family workshops for carers on various carer and patient outcomes (Goddard, MacDonald & Treasure, 2011; Macdonald, Murray, Goddard, & Treasure, 2011; Sepulveda, Lopez, Todd, Whitaker, & Treasure, 2008; Whitney et al., 2011). For example, Macdonald and colleagues (2011) found that skills training in carers was related to an increase in carer self-efficacy as well as to reductions in carer distress, anxiety and burden. Whitney et al. (2011) compared the effectiveness of skill-based family workshops

to individual family work and found that, in both groups, patients' body mass index improved as did carers' distress. As such, there is emerging research across the lifespan that examines the role that parents and carers can play throughout recovery.

Despite the importance of their role, in the treatment process, few studies have examined the impact of parental factors, including parental self-efficacy, on treatment outcomes in family-based therapy. This dearth of research has left researchers and clinicians hypothesizing about the mechanisms of change (Loeb & Le Grange, 2009). In fact, the research that has examined parental factors in child and adolescent populations has yielded only a few pertinent findings. For example, in AN populations, adolescent outcomes are not predicted by family status (intact families or not) (Lock, Couturier, Bryson, & Agras, 2006), and families high in criticism do better in a separated form of family-based therapy, where the adolescent is seen separately from the parents (Eisler et al., 2000; Le Grange, Eisler, Dare, & Hodes, 1992). Within an adult ED population, preliminary research suggests that changes in caregiving self-efficacy, expressed emotion, accommodation and enabling behaviours are related to reductions in carer distress, as well as an increase in the perceived level of functioning of their loved one (Goddard, Macdonald, Sepulveda et al., 2011).

To add to the literature on parent-related mechanisms of change throughout family-based treatment for adolescent ED and in light of the fact that the primary responsibility for recovery is assigned to the parents, the purpose of the present study is to examine both parent and adolescent outcomes in response to family-based treatment and to determine whether increases in parental self-efficacy predict a reduction in adolescent ED, depressed mood and anxiety symptoms.

Specifically, adolescent ED behaviours and psychological symptoms, as well as parental self-efficacy were assessed prior to beginning family-based therapy, after 3 months in a family-based ED programme and again after 6 months in the programme. In addition, change in parental self-efficacy over time was used to predict adolescent ED, depressed mood and anxiety symptoms over the course of treatment.

Hypotheses

Family-based treatment is delivered in such a way as to motivate parents and increase their self-efficacy in supporting their adolescent throughout the recovery process. As such, we hypothesized that

1. parental self-efficacy would increase over the course of therapy;
2. adolescent ED, depressed mood and anxiety symptoms would decrease over the course of therapy;

and

3. increases in self-efficacy would be predictive of positive outcomes on adolescent ED, depressed mood and anxiety symptoms.

Method

Participants

Forty-nine adolescents and their parents participated in the study. Thirty adolescents and their families were treated as outpatients,

and 19 received day treatment. Among the adolescents in the sample, the mean age at assessment was 15.55 ($SD = 1.46$). ED diagnoses included AN ($n = 12$), BN ($n = 13$), eating disorder not otherwise specified (EDNOS) – restricting subtype ($n = 15$) and EDNOS – purging subtype ($n = 9$). Comorbid psychiatric diagnoses included anxiety disorders ($n = 16$) and mood disorders ($n = 3$). Of these, three adolescents were diagnosed with more than one comorbidity. Thirty-three adolescents had no comorbid diagnoses. At assessment, 14 adolescents were on small doses of a variety of SSRIs initiated by the referral sources, mainly family physicians. No additional information was available regarding the use of medication at the time of the study.

At assessment, 11 of the 49 adolescents were at ideal body weight, as determined by information obtained through clinical history, growth curves and menstrual threshold, whereas the remaining 38 adolescents averaged 87.95% of ideal body weight. At the 3-month follow-up, 21 of the adolescents were at or above ideal body weight, with the remaining 27 adolescents averaging 93.67% of ideal body weight. At the 6-month follow-up, 28 of the adolescents were at or above ideal body weight, with the remaining 18 adolescents averaging 96.5% ideal body weight. Weight data was not available for one adolescent at 3 months and for three adolescents at 6 months. There was no observed correlation between the pattern of weight gain, clinical diagnosis and the use of medication.

Among the 49 sets of parents in the sample, 40 couples were married, two were separated and seven were divorced. In each case, separated and divorced parents worked together as a unit with respect to their daughter's recovery.

Setting

The Eating Disorder Program where the study took place is located within a regional hospital in Ontario, Canada. The programme uses a multidisciplinary approach with family-based therapy and principles playing a central role in day treatment and outpatient services. Additional treatments are adjunctive (e.g. dietetic counselling, individual therapy) with the exception of medical monitoring, which is a required component of the programme for all clients.

A number of staff where the study took place received training in the model by Daniel Le Grange (for single family outpatient treatment), Ivan Eisler and Pennie Fairbairn (for multifamily group therapy), and James Lock (in a day hospital context). These staffs also provided in-house trainings to staff on a regular basis.

As part of both the day treatment and outpatient treatment programmes, the family met on a regular basis with the family-based therapist. Early in treatment, the therapist emphasized the seriousness of the disorder and delineated the health consequences of disordered eating behaviours. Parents were then encouraged to refeed their adolescent by providing regular meals and snacks and to halt behaviours such as purging as soon as possible so that the health effects could be reversed. It was left to parents to decide how to best refeed their adolescent and prevent ED behaviours. Therapists provided encouragement and support to the family and conveyed their confidence in the parents in taking charge of their adolescent's recovery. From week to week, therapists celebrated successes and supported parents when faced with challenges in an effort to support parents' development of

self-efficacy vis-à-vis recovery. Consistent with the family-based therapy model, after this first phase of treatment, the therapist then worked with parents to slowly allow their adolescent to have more control over her eating and, ultimately, to restore normal family functioning and age-appropriate activities.

Procedure

During a multidisciplinary assessment in the Adolescent Eating Disorder Program, patients and their parents completed measures and were offered treatment in a day treatment or outpatient setting, depending on the severity of the presentation. Patients and their parents were then reassessed at 3-month post-assessment, and at 6-month post-assessment.

Adolescent measures

Eating-disordered symptomatology

Eating disorder symptomology was assessed using the Eating Disorder Inventory, 3rd Edition (EDI-3; Garner, 2004), which is a 91-item measure of psychological traits and ED symptoms relevant to the development and maintenance of AN, BN and EDNOS. The EDI-3 was designed for use with females aged 13–53 years. A zero-to-four-point scoring system is used, and responses are categorized into four subscales as well as six composite scores. For the purposes of this study, we examined two key subscales of the EDI: Drive for Thinness and Body Dissatisfaction.

Anxiety

Anxiety was measured with the Multidimensional Anxiety Scale for Children (MASC), a 39-item, four-point Likert self-report scale that robustly represents the factor structure of anxiety in children and adolescents aged 8–18 years (March, 1997). It is a well-validated and reliable instrument that assesses anxiety symptoms across clinically significant symptom domains, distinguishes between important anxiety symptoms and demonstrates changes in symptom type and level for treatment monitoring and outcome evaluations. Main factors include (i) physical symptoms (tense/restless and somatic/autonomic), (ii) social anxiety (humiliation/rejection and public performance fears), (iii) harm avoidance (perfectionism and anxious coping) and (iv) separation anxiety. The MASC factor structure shows excellent internal reliability and test–retest reliability (March, Parker, Sullivan, & Stallings, 1997).

Depressed mood

Depressed mood was measured with the Children's Depression Inventory (CDI; Kovacs, 1992). This self-report inventory measures levels of depressive symptomatology in children and adolescents, aged 7–17 years. The measure comprises 27 items, each containing three statements. Respondents are instructed to choose the statement that best describes their feelings within the last two weeks. Levels of internal consistency are generally very good (.71–.87; Kovacs, 1992), good criterion validity has been reported and extensive normative data have been collected (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

Parent measures

Parental self-efficacy

Parental self-efficacy was assessed using a revised version of the Parent Versus Anorexia Scale (PvA; Rhodes, Baillee, Brown, & Madden, 2005).[1] The PvA was designed to study parental self-efficacy, that is, 'the ability of a parent to adopt a primary role in taking charge of the [ED] in the home setting for the purpose of bringing about the recovery of their child' (Rhodes et al., 2005). Seven items are included in the scale and these are rated on a four-point Likert scale (strongly disagree, disagree, agree, strongly agree). Scores can range from 7 to 35. In the current study, the word 'eating disorder' was used instead of 'anorexia' throughout the questionnaire. Sample items include 'I feel equipped with specific strategies for the task of bringing about the complete recovery of my child in the home setting' and 'It is more my responsibility than my child's to bring him/her to a healthy weight'. There is preliminary evidence of adequate psychometric properties for the PvA (Rhodes et al., 2005).

Results

Sphericity assumption

A routine check on the question of whether the data met all the assumptions for within-subjects analysis of variance (ANOVA) revealed that the sphericity assumption had been violated in some of the repeated measures analyses. This is a common occurrence in within-subjects designs (Howell, 2010). As a result, the degrees of freedom for within-subjects effects in all of the analyses where sphericity was violated were handled using the Greenhouse–Geisser correction.[2]

Adolescents' eating disorder symptomatology

We predicted that adolescents' ED symptomatology would decrease over time. To assess this, we examined two key subscales of the EDI: Drive for Thinness and Body Dissatisfaction. To test our hypothesis, we conducted two repeated measures ANOVAs with time (Time 0, Time 1, Time 2) as the within-subjects factor and adolescents' score on each of the two subscales of the EDI as the dependent variable. Both analyses revealed significant time effects, $F > 9.5$, $p < .001$. Although EDI scores did not reach the low clinical range during treatment, adolescents' ED symptoms did decrease significantly over time. We conducted a series of post hoc analyses to further break down the main effect of time. These analyses revealed that adolescents significantly decreased in ED symptomatology from T0 to T1, and again from T1 to T2 (see Table 1 for means, standard errors and a summary of post hoc significant differences).

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[1]The revision of the scale was minor. Statements referring to anorexia were revised to reflect the different eating disorder presentations. For example, the item 'It is not always advisable to get tough with a child with anorexia because he/she will experience too much trauma and distress' was revised to 'It is not always advisable to get tough with a child with an eating disorder because he/she will experience too much trauma and distress'.

[2]Note that, in some case, different degrees of freedom are reported and reflect the fact that not all participants filled out each item on all of the measures.

Table 1 Means and standard errors for adolescent dependent variables

	Time 0		Time 1		Time 2	
	Mean	SE	Mean	SE	Mean	SE
EDI – Drive for Thinness	44.75$_a$	2.57	37.00$_b$	1.93	31.39$_c$	2.47
EDI – Body Dissatisfaction	44.41$_a$	2.17	39.76$_b$	2.06	34.83$_c$	2.18
MASC	59.63$_a$	1.66	54.11$_b$	2.39	49.30$_c$	2.41
CDI	64.89$_a$	3.39	57.50$_b$	3.22	48.39$_c$	2.72

Note: Means with different subscripts are significantly different from one another.

Adolescents' anxiety

We expected that adolescents' anxiety (assessed by the MASC) would decrease over time. To test this prediction, we conducted a repeated measures ANOVA with time (Time 0, Time 1, Time 2) as the within-subjects factor and adolescents' score on the MASC as the dependent variable. A significant time effect, $F_{(2, 52)} = 12.29$, $p = .0001$, revealed that adolescents decreased in anxiety over time. Post hoc analyses further breaking down the main effect of time revealed that adolescents significantly decreased in anxiety from T0 to T1, and again from T1 to T2 (Table 1). Anxiety scores were in the clinical range at T0 and in the normal range at T1 and T2.

Adolescents' depressed mood

We predicted that adolescents' depressed mood symptoms (measured with the CDI) would decrease over time. We conducted a repeated measures ANOVA with time (Time 0, Time 1, Time 2) as the within-subjects factor and adolescent's score on the CDI as the dependent variable to test this prediction. A significant time effect, $F_{(2, 54)} = 12.00$, $p = .0001$ revealed that adolescents reported fewer symptoms of depressed mood over time. Post hoc analyses further breaking down the main effect of time revealed that adolescents significantly decreased in depressed mood symptoms from T0 to T1, and again from T1 to T2 (Table 1). Depressed mood scores were in the clinical range at T0 and T1 but had moved into the normal range by T2.[3]

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[3]For each of the adolescent outcome measures, we tested whether day treatment patients differed from outpatients. To test this, we conducted a series of mixed ANOVAs with time (Time 0, Time 1, Time 2) as the within-subjects factor, treatment type (day treatment vs. outpatient treatment) as the between-subjects factor and adolescent's score on the outcome measures as the dependent variable. We ran separate analyses for each of the dependent measures (EDI – Drive for Thinness, EDI – Body Dissatisfaction, MASC and CDI). Three out of the four ANOVAs (for EDI – Drive for Thinness, EDI – Body Dissatisfaction and CDI) revealed no significant treatment effects or time × treatment interactions ($F < 2.27$, $p > .10$). However, the mixed ANOVA with adolescent's score on the MASC as the dependent variable did reveal a significant time × treatment interaction, $F_{(2, 50)} = 3.93$, $p = .03$. Post hoc analyses to break down this interaction revealed that outpatients' anxiety improved significantly from T0 to T1, then levelled off at T2. Day treatment patients' anxiety, on the other hand, continued to improve throughout the entire course of treatment (i.e. there were significant improvements in anxiety at each period). This time × treatment interaction for adolescent's anxiety does not alter the main findings from these analyses. It simply reveals a steeper initial trajectory of improvement for outpatients than day treatment patients.

Parental self-efficacy

We predicted that parents would improve in self-efficacy over time. To test this, we conducted a repeated measures ANOVA with parent (mother vs. father) and time (Time 0, Time 1, Time 2) both as within-subjects factors and score on the PvA scale as the dependent variable. A significant time effect, $F_{(2, 38)} = 48.06$, $p = .0001$, revealed that both mothers and fathers improved in self-efficacy over time (refer to Table S1 for means and standard errors). No other effects were significant ($F < 1.4$, $p > .24$). We conducted a series of post hoc analyses to further break down the main effect of time. These analyses revealed that, overall, parents significantly increased on self-efficacy from T0 to T1, and again from T1 to T2 (means and standard errors for T0, T1 and T2 are 17.43 (.58), 21.17 (.69) and 22.61 (.67), respectively).

The link between parental self-efficacy and adolescents' eating symptomatology

We predicted that improvements in parental self-efficacy would predict reductions in adolescents ED symptomatology. Adolescent ED symptomatology was assessed using the EDI. Parental self-efficacy was measured using the PvA scale. To test our predictions, we conducted a series of multiple regression analyses. Our analytic approach was to examine the change from baseline in parent self-efficacy (by controlling for baseline scores to assess variance in self-efficacy at later time points over and above baseline levels) as a predictor of the change in adolescent outcome (by entering adolescents baseline scores in Step 1 and their scores at the target time in Step 2). This allowed us to examine how the change and progress made by parents over a 3-month period was associated with the change and progress made by their adolescents over the same period. We assessed (i) whether mothers' self-efficacy at T1 (controlling for their baseline self-efficacy) predicted adolescents' ED symptoms at T1 (controlling for their baseline ED symptoms), (ii) whether mothers' self-efficacy at T2 (controlling for their baseline self-efficacy) predicted adolescents' ED symptoms at T2 (controlling for their baseline ED symptoms), (iii) whether fathers' self-efficacy at T1 (controlling for their baseline self-efficacy) predicted adolescents' ED symptoms at T1 (controlling for their baseline ED symptoms) and finally (iv) whether fathers' self-efficacy at T2 (controlling for their baseline self-efficacy) predicted adolescents' ED symptoms level at T2 (controlling for their baseline ED symptoms). We examined two key subscales of the EDI: Drive for Thinness and Body Dissatisfaction separately, for each parent and each period, resulting in a total of eight regression analyses.

In each of the analyses, adolescents' baseline EDI score was entered in Step 1. Adolescents' baseline EDI score positively predicted T1 EDI levels for two of the analyses (T1 EDI – Body Dissatisfaction for both parents (standardized $bs > .40$, $ps < .03$) but did not predict T1 EDI levels for the remaining analyses and never significantly predicted T2 EDI scores (standardized $bs < |.37|$, $ps > .06$). Parents' baseline PvA scores (self-efficacy) were always entered at Step 2. Fathers' baseline PvA score significantly predicted adolescent's T1 EDI – Body Dissatisfaction (standardized $bs > -.39$, $ps < .04$), such that the higher fathers scored on self-efficacy at baseline, the fewer ED symptoms their adolescent reported. Parents' baseline PvA score did not predict adolescents' later EDI levels for the remaining analyses

(standardized $bs < |.27|$, $ps > .23$). Results for the key analyses for parents T1 and T2 levels of self-efficacy predicting adolescents' T1 and T2 EDI levels are reported in Table 2. Analyses revealed that fathers' increase in self-efficacy from T0 to T1 predicted a decrease in adolescents' ED symptoms at T1, whereas mothers' increase in self-efficacy from T0 to T2 predicted a decrease in adolescents' ED symptoms at T2.

The link between parental self-efficacy and adolescents' level of anxiety

We predicted that parental improvements in self-efficacy would predict reductions in their adolescents' anxiety levels over time (as assessed by the MASC). To test our predictions, we conducted a series of multiple regression analyses. In four separate regression analyses, we assessed (i) whether mothers' self-efficacy at T1 (controlling for their baseline self-efficacy) predicted adolescents' anxiety level at T1 (controlling for their baseline anxiety), (ii) whether mothers' self-efficacy at T2 (controlling for their baseline self-efficacy) predicted adolescents' anxiety level at T2 (controlling for their baseline anxiety), (iii) whether fathers' self-efficacy at T1 (controlling for their baseline self-efficacy) predicted adolescents' anxiety level at T1 (controlling for their baseline anxiety) and finally (iv) whether fathers' self-efficacy at T2 (controlling for their baseline self-efficacy) predicted adolescents anxiety level at T2 (controlling for their baseline anxiety). In each of the analyses, adolescent baseline MASC score was entered in Step 1 and positively predicted T1 MASC levels (standardized $bs > .60$, $ps < .001$) and T2 MASC levels when mother's self-efficacy was included in the analysis (standardized $b = .50$, $p = .01$) but did not predict T2 MASC levels when father's self-efficacy was included in the analysis (standardized $b = .23$, $p = .33$). Parents' baseline PvA scores (self-efficacy) were always entered at Step 2. Parents' baseline self-efficacy did not predict adolescent's later MASC level (standardized $bs < .23$, $ps > .13$), with one exception: fathers' baseline PvA score predicted adolescents MASC level at

T1 (standardized $b = -.33$, $p = .03$). The higher fathers scored on self-efficacy at T0, the less anxiety their adolescent experienced at T1. Results for the key analyses for parents T1 and T2 levels of self-efficacy predicting adolescent T1 and T2 MASC levels are reported in Table 2. Analyses revealed that fathers' increase in self-efficacy from T0 to T1 predicted a decrease in adolescent anxiety at T1. Mothers' increase in self-efficacy was not related to adolescent anxiety at either period.

The link between parental self-efficacy and adolescents' depressed mood

We predicted that improvements in parental self-efficacy would predict reductions in adolescent mood symptoms (measured using the CDI) over time. In four separate regression analyses, we assessed (i) whether mothers' self-efficacy at T1 (controlling for their baseline self-efficacy) predicted adolescents' depressed mood symptoms level at T1 (controlling for their baseline CDI score), (ii) whether mothers' self-efficacy at T2 (controlling for their baseline self-efficacy) predicted adolescents' mood symptoms level at T2 (controlling for their baseline CDI score), (iii) whether fathers' self-efficacy at T1 (controlling for their baseline self-efficacy) predicted adolescents' mood symptoms level at T1 (controlling for their baseline CDI score) and finally (iv) whether fathers' self-efficacy at T2 (controlling for their baseline self-efficacy) predicted adolescents' mood symptoms level at T2 (controlling for their baseline CDI score). In each of the analyses, adolescents' baseline CDI score was entered in Step 1 and positively predicted T1 CDI levels (standardized $bs > .46$, $ps < .01$) but did not predict T2 CDI levels (standardized $bs < .24$, $ps > .27$). Parents' baseline PvA scores (self-efficacy) were always entered at Step 2 and never significantly predicted adolescents' later CDI levels (standardized $bs < |.23|$, $ps > .19$). Results for the key analyses for parents' T1 and T2 levels of self-efficacy predicting adolescents' T1 and T2 CDI levels are reported in Table 2. Analyses revealed that fathers' increase in self-efficacy from T0 to T1 predicted a decrease in adolescents' depressed mood symptoms at T1, whereas mothers' increase in self-efficacy from T0 to T2 predicted a decrease in adolescents' depressed mood symptoms at T2.[4]

Table 2 Multiple regression results for change in parent self-efficacy over time on adolescent outcomes

	Mothers				Fathers			
	b	t	p	R^2	b	t	p	R^2
EDI – Drive for Thinness								
T1	−.09	−0.42	.68	.01	−.72	−4.08	<.0001	.38
T2	−.67	−3.66	<.001	.33	−.35	−1.59	.13	.11
EDI – Body Dissatisfaction								
T1	.10	0.54	.59	.01	−.35	−2.39	.03	.09
T2	−.41	−1.86	.08	.11	.07	0.34	.74	.01
CDI								
T1	−.30	−1.71	.10	.08	−.42	−2.29	.03	.13
T2	−.66	−3.29	<.01	.32	−.21	−0.90	.38	.04
MASC								
T1	−.16	−1.08	.29	.02	−.52	−3.91	<.001	.21
T2	−.25	−1.19	.25	.05	−.28	−1.22	.24	.07

Note. Predictor 1 in all cases is PvA Time 1 and Predictor 2 in all cases is PvA Time 2.

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[4]We also ran each of the regression analyses with type of treatment (day treatment vs. outpatient treatment) as a factor (i.e. we entered treatment type as a dummy code in Step 1 and entered the treatment × parental self-efficacy interaction in Step 3). For 14 of the 16 regression analyses, there were no treatment effects and no treatment × parental self-efficacy effects (standardized bs 3.16, $ps > .10$). However, we did find a significant interaction between fathers' self-efficacy and treatment at Time 1 for the EDI: Drive for Thinness and for the MASC (standardized $bs > 1.43$, $ps < .05$). Post hoc analyses revealed that fathers' parental self-efficacy significantly predicted adolescent's improvements in both anxiety and ED symptomatology for outpatients. Father' self-efficacy only marginally predicted improvement in anxiety for day treatment patients and did not predict improvements in adolescent's ED symptomatology for day treatment patients. These findings suggest that, among adolescents in day treatment programmes, fathers' improvements in self-efficacy play less of a role for adolescent's Time 1 improvements on the MASC and EDI: Drive for thinness, possibly reflecting that fathers are somewhat less involved (by T1) in day treatment than outpatient adolescent care. It is important to note though that for the vast majority of the regression analyses, there were no differences between the treatment groups at all.

Discussion

The results of this study indicate that, in the context of family-based treatment, adolescents experience reductions in ED symptomatology, as well as in ratings of depressed mood and anxiety symptoms. Parents also experienced significant benefit from their involvement in treatment. Specifically, both mothers and fathers showed increases in self-efficacy throughout the course of treatment. The results of this study also support the hypothesis that parental self-efficacy is an important predictor of adolescent ED, depressed mood and anxiety symptoms. Specifically, both parents' self-efficacy scores were predictive of positive adolescent outcomes with respect to ED and depressed mood symptoms, whereas fathers' increase in self-efficacy was also predictive of improvements of anxiety symptoms. These findings are particularly important given the tenets of the model. Therapists work extensively with parents to increase their hope and their self-confidence vis-à-vis their ability to bring about recovery in their adolescent. The results suggest that the therapy is having the desired effect over time—an effect so specific that it is not likely to be random.

In terms of parent-specific effects, it was notable that fathers' increases in self-efficacy earlier in treatment (T0–T1) predicted improvements for adolescents, whereas mothers' increase from T0 to T2 was predictive of their adolescents' improvement. This result may reflect what is commonly seen in therapy and reported in the literature, where family climate is related to the impact of the illness and improves following changes in ED symptoms (Nilsson, Engström, & Hägglöf, 2012; Whitney & Eisler, 2005). For example, according to Whitney and Eisler (2005), when families initially present for service, the ED has already begun to reorganize family patterns; therefore, an initially less close relationship with a father can be amplified to the point where they are on the periphery. As a result, it could be that the adolescent responded more quickly to changes in their father, who was less involved at the outset. As fathers increased in self-efficacy and felt they could handle the ED symptoms, their adolescent improved in response. For mothers, the increase in self-efficacy was similar, but their adolescent responded more

gradually to their mother's new confidence. Additional research will be required to clarify this process; however, these results certainly highlight the importance of involving both parents in therapy. These findings also underline the positive contributions made by fathers specifically, as they have been reported to be less involved in mental health interventions in general (Phares, Lopez, Fields, Kamboukos, & Duhig, 2005).

This study is not without limitations. There is no control group, as the programme's structure was not conducive to such a design. In addition, the sample size is fairly low. The sample's heterogeneity also makes it difficult to ascertain whether other individual and/or treatment factors are related to outcomes. Specifically, future studies would include variables such as treatment intensity, impact of specific ED presentation and comorbid diagnoses and allow for other parental and family characteristics to be explored, including the reported experience of invalidating environments in childhood and its relationship with negative core beliefs (Ford, Waller, & Mountford, 2011). An increase in sample size would also allow for comparisons to be made between intact, separated and divorced parents, as well as the impact of psychopharmacological interventions. Despite these limitations and in light of the dearth of literature relating to process and outcomes of family-based therapy, the clear and consistent pattern of results reported here is significant and makes an important contribution to the understanding of parental factors associated with adolescent outcomes in the area of EDs.

Overall, the results of the present study suggest that family-based treatment leads to an increase in feelings of empowerment in both mothers and fathers that translate into higher self-efficacy. This increase not only leads to positive outcomes for parents in terms of feeling ready and capable of taking control of their adolescent's illness but it is also directly related to their adolescent moving forward on the road to recovery. This result is consistent with the philosophy of the family-based therapy model, which postulates that both parents are key in the recovery process, and that unless there is a clear rationale to proceed otherwise, the involvement of both mothers and fathers should be the rule, not the exception.

REFERENCES

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561–571.

Eisler, I., Dare, C., Hodes, M., Russell, G., Dodge, E., & Le Grange, D. (2000). Family therapy for adolescent anorexia nervosa: The results of a controlled comparison of two family interventions. *Journal of Child Psychology and Psychiatry*, 41, 727–736.

Ford, G., Waller, G., & Mountford, V. (2011). Invalidating childhood environments and core beliefs in women with eating disorders. *European Eating Disorders Review*, 19, 316–321.

Garfinkel, P. E., Lin, E., Goering P., Spegg, C., Goldbloom, D. S., Kennedy, S., et al. (1995). Bulimia nervosa in a Canadian community sample: Prevalence and comparison of subgroups. *The American Journal of Psychiatry*, 152, 1052–1058.

Garfinkel, P. E., Lin, E., Goering, P., Spegg, C., Goldbloom, D. S., Kennedy, S., et al. (1996). Should amenorrhea be necessary for a diagnosis of anorexia nervosa: Evidence from a Canadian community sample? *The British Journal of Psychiatry*, 168, 500–506.

Garner, D. M. (2004). *Eating Disorder Inventory-Third Edition (EDI-3)*. Luts, FL: Psychological Assessment Resources.

Goddard, E., Macdonald, P., Sepulveda, A. R., Naumann, U., Landau, S., Schmidt, U., et al. (2011). Cognitive interpersonal maintenance model of eating disorders: Intervention for carers. *The British Journal of Psychiatry*, 199, 225–231.

Goddard, E., Macdonald, P., & Treasure, J. (2011). An examination of the impact of "the Maudsley collaborative care skills training workshops" on patients with anorexia nervosa: A qualitative study. *European Eating Disorders Review*, 19, 150–161.

Golden, N. H., Katzman, D. K., Kreipe, R. E., Stevens, S. L., Sawyer, S. M., Rees, J., et al. (2003) Eating disorders in adolescents:

Position paper of the Society of Adolescent Medicine. *Journal of Adolescent Health*, 33, 496–503.

Howell, D. C. (2010). *Fundamental Statistics for the Behavioral Sciences (7th ed)*. Wadsworth Publishing.

Kovacs, M. (1992). *Manual of the Children's Depression Inventory*. Toronto, ON: Multi-Health Systems.

Le Grange, D. (1999). Family therapy for adolescent anorexia nervosa. *Journal of Clinical Psychology*, 55, 727–739.

Le Grange, D., & Eisler, I. (2009). Family interventions in anorexia nervosa. *Child and Adolescent Psychiatric Clinics of North America*, 18, 159–173.

Le Grange, D., Eisler, I., Dare, C., & Hodes, M. (1992). Family criticism and self starvation: A study of expressed emotion. *Journal of Family Therapy*, 14, 177–192.

Le Grange, D., Lock, J., & Dymek, M. (2003). Family-based therapy for adolescents with bulimia nervosa. *American Journal of Psychotherapy*, 57, 237–251.

Lock, J., Couturier, J., Bryson, S., & Agras, S. (2006). Predictors of dropout and remission in family therapy for adolescent anorexia nervosa in a randomized clinical trial. *International Journal of Eating Disorders*, *39*, 639–647.

Loeb, K. L., & Le Grange, D. (2009). Family-based treatment for adolescent eating disorders: Current status, new applications and future directions. *International Journal of Child and Adolescent Health*, *1*, 243–254.

Macdonald, P., Murray, J., Goddard, E., & Treasure, J. (2011). Carer's experience and perceived effects of a skills based training programme for families of people with eating disorders: A qualitative study. *European Eating Disorders Review*, *19*, 475–486.

March, J. S. (1997). *Manual for the Multidimensional Anxiety Scale for Children (MASC)*. Toronto, ON: Multi-Health Systems.

March, J. S., Parker, J. D. A., Sullivan, K., & Stallings, P. (1997). The multidimensional anxiety scale for children (MASC): Factor structure, reliability, and validity. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 554–565.

Nilsson, K., Engström, I., & Hägglöf, B. (2012). Family climate and recovery in adolescent onset eating disorders: A prospective study. *European Eating Disorders Review*, *20*, e96–e102. DOI: 10.1002/erv.1127

Phares, V., Lopez, E., Fields, S., Kamboukos, D., & Duhig, A. M. (2005). Are fathers involved in pediatric psychology research and treatment? *Journal of Pediatric Psychology*, *30*, 631–643.

Rhodes, P., Baillee, A., Brown, J., & Madden, S. (2005). Parental efficacy in the family based treatment of anorexia: Preliminary development of the Parents Versus Anorexia Scale (PVA). *European Eating Disorders Review*, *13*, 399–405.

Sepulveda, A. R., Lopez, C., Todd, G., Whitaker, W., Treasure, J. (2008). An examination of the impact of 'the maudsley eating disorder collaborative care skills workshops' on the well being of carers: A pilot study. *Social Psychiatry and Psychiatric Epidemiology*, *43*, 584–591.

Steinhausen, H.-C. (2002). The outcome of anorexia nervosa in the 20th century. *The American Journal of Psychiatry*, *159*, 1284–1293.

Steinhausen, H.-C., & Weber, S.. (2009). The outcome of bulimia nervosa: Findings from the one-quarter century research. *The American Journal of Psychiatry*, *166*, 1331–1341.

Whitney, J., & Eisler, I. (2005). Theoretical and empirical models around caring for someone with an eating disorder: The reorganization of family life and inter-personal maintenance factors. *Journal of Mental Health*, *14*, 575–585.

Whitney, J., Murphy, T., Landau, S., Gavan, K., Todd, G., Whitaker, W., et al. (2011), A practical comparison of two types of family intervention: An exploratory RCT of family day workshops and individual family work as a supplement to inpatient care for adults with anorexia nervosa. *European Eating Disorders Review*. DOI: 10.1002/erv.1076