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Changes in Comorbid Obsessive-Compulsive and Restrictive Eating Disorder Symptoms in Eating Disorder Focused Family Therapy

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ABSTRACT

Objective: Restrictive eating disorders (EDs) are highly comorbid with obsessive-compulsive disorder (OCD), and comorbidity is associated with worsened outcomes. However, it is unclear if (1) Maudsley family therapy (FT-AN) for restrictive EDs reduces comorbid OCD symptoms, and (2) if higher baseline OCD symptoms merely reflect higher severity or are associated with inhibited ED treatment.

Method: Participants with restrictive EDs (Anorexia Nervosa, Atypical Anorexia Nervosa, or other specified feeding and eating disorders) received FT-AN as part of an intensive day program or outpatient services in London from mid-2024 through January 2026. Nonparametric Wilcoxon signed-rank tests examined pre/post differences in ED, OCD, and general impairment symptomology. Linear regressions investigated the effect of baseline OCD scores on changes in ED symptoms across treatment.

Results: Across FT-AN, ED pathology and weight improved while OCD pathology and general impairment did not. After excluding likely under-reporters, OCD symptoms and overall functioning did significantly improve. More severe OCD impairment at baseline did not impair, and in fact was related with more ED improvement. However, more severe ED and OCD symptoms at baseline were both associated with less change in weight. Together, these results suggest that worsened outcomes among patients with OCD represent higher initial severity rather than treatment inhibition.

Discussion: FT-AN's effects on ED symptoms are likely not directly inhibited by, but also do not appear to fully target, OCD symptoms. Future research should examine efficacy and optimal timing of incorporating OCD-specific treatments into care.

Eating disorders (EDs) involve severe, persistent disturbances related to food, body shape, and/or weight that cause distress or dysfunction (American Psychiatric Association 2022). Restrictive EDs are characterized by insufficient energy intake relative to physiological needs, which can stem from behaviors such as undereating or overexercising, rather than binging and purging (American Psychiatric Association 2022; Birgegård et al. 2023). Examples include the restricting subtypes of anorexia nervosa (AN) and certain other specified feeding and eating disorders (OSFED) like atypical anorexia nervosa (AAN). AN is one of the deadliest psychiatric diseases with relatively low remission rates (Arcelus et al. 2011; Chesney et al. 2014; Khalsa et al. 2017). While AN is most often studied, other restrictive EDs are also debilitating. In fact, AAN often shows similar or more

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Summary

- Maudsley Family Therapy improved eating disorder symptoms but not symptoms related to obsessive-compulsive disorder (OCD) or general impairment.
- When patients have comorbid OCD, worsened eating disorder outcomes after family therapies likely reflect more severity prior to treatment, rather than less improvement.

severe psychopathology than AN (Fitterman-Harris et al. 2024; Johnson-Munguia et al. 2024; Sandoval-Araujo et al. 2024).

AN is up to 62% comorbid with obsessive-compulsive disorder (OCD) (Lilenfeld et al. 1998; Godart et al. 2002), the disorders are genetically related (Yilmaz et al. 2023, 2020), and AN/OCD comorbidity is associated with worsened outcomes. Even the presence of OCD symptoms has been associated with longer latency to receiving AN treatment and lower weight at treatment discharge (Blachno et al. 2014). More severe OCD symptoms have also been associated with fewer reductions in ED symptoms across time (Lo Sauro et al. 2013). However, it is unclear if OCD symptomology directly impacts AN symptom change or merely reflects higher psychiatric severity. Further, whether these results extend to samples with clinical levels of OCD, and when including disorders across the restrictive ED spectrum, remains to be seen.

In addition to examining the effects of OCD presence on treatment efficacy for EDs, it is also important to know if ED treatments improve comorbid OCD symptoms. AN and OCD share obsessive-compulsive features including obsessions, compulsions, and executive functioning difficulties (Dingemans et al. 2022), although the content of these features may be distinct (Altman and Shankman 2009). Therefore, if ED treatments are targeting the same mechanisms maintaining both ED and OCD compulsions, ED treatments may also offer improvement for OCD symptoms. Additionally, ED treatments may reduce obsessive-compulsive symptoms that can arise directly from starvation (Spettigue et al. 2025) by alleviating malnourishment. The first-line treatment for adolescent AN, and related disorders like AAN, is family therapy (Vasey et al. 2024). A recent meta-review of meta analyses showed that family therapies were the only treatment type to be more effective than other interventions for improvements in both ED symptoms and weight (Monteleone et al. 2022). Family therapy includes various subtypes like Family-Based Treatment (FBT) (Lock and Le Grange 2019, 2015) and Maudsley Family Therapy for AN (FT-AN) (Eisler et al. 2016). Despite robust evidence that family-based therapies improve ED symptoms (Monteleone et al. 2022), FT-AN has not been previously studied in regards to OCD symptom reduction. Meanwhile, FBT, a parallel form of family therapy for restrictive EDs, does not tend to show improvements in OCD symptomology (Agras et al. 2014; Lock et al. 2018; Schmidt et al. 2015). However, previous research has not typically reported OCD diagnostic status nor included individuals with more severe OCD symptoms (Steinhoff et al. 2025). Previous studies involving family therapies have only included outpatients; these samples on average exhibited subclinical OCD symptomology, unless inclusion criteria necessitated clinical levels of OCD (Agras et al. 2014; Lock et al. 2018; Hughes et al. 2017). Therefore, including both intensive treatment and outpatient samples and those with clinical levels of OCD is critical for improving generalizability and ensuring that any lack of improvement in OCD pathology is not simply due to floor effects. Indeed, Exposure and Response Prevention (ERP) is the gold-standard treatment for OCD (The National Institute for Health and Care Excellence (NICE) 2005), but family therapy does not inherently follow ERP principles, suggesting family therapies may not inherently address comorbid OCD pathology.

This study aimed to examine the effect of OCD symptoms on ED treatment outcomes. We hypothesized that higher OCD symptoms would be associated with less restrictive ED symptom improvement. We also aimed to examine changes in OCD and restrictive ED symptoms across FT-AN treatment. We hypothesized that restrictive ED symptoms would improve but OCD symptoms would not in FT-AN, consistent with studies of FBT (Agras et al. 2014; Lock et al. 2018; Schmidt et al. 2015). Importantly, our study included patients with more severe OCD symptomology and we reported likely OCD diagnostic status, addressing a critical gap in the literature (Steinhoff et al. 2025).

1 | Method

1.1 | Ethics

This study was approved by the South London and Maudsley (SLaM) Service Evaluation and Audit Committee (ref: 44). SLaM service evaluation and audit approval allows for analysis and publication of anonymized data extracted from case files without written consent from participants or carers. Outcome measures were administered as part of routine clinical care when possible. Analysis involved data collected routinely as part of clinical practice and therefore no formal patient informed consent and Research Ethics Committee (REC) approval were required. The study was also determined to be exempt via the Washington University in St. Louis IRB (#202311097).

1.2 | Dataset and Treatment Programs

A de-identified clinical dataset was used from the specialist Maudsley Centre for Child and Adolescent Eating Disorders (MCCAED) in London, United Kingdom. MCCAED sees youth up to age 18 and has a catchment area of more than two million people. Data were collected from mid-2024 through January 2026 from both the Intensive day Treatment Programme (ITP) and outpatient services, which operate Monday through Friday at the Maudsley Hospital. For ITP specifically, admissions are rolling and interventions include individual, family, and group therapies, meal support, and psychoeducation, of which 6.25 h per week are group programming. In this study, treatment length was 7.8 (SD = 4.5; range = 0–22.3) months on average, including an average 3.1 (range 0–9.0) months for ITP and an average 8.8 (range 0–22.3) months for outpatient. Of the 150 participants, 27 (18%) were from the ITP clinic.

All participants in this study received FT-AN. FT-AN is a manualized, evidence-based treatment that includes four phases

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over 12 months (Eisler et al. 2016). Meetings with patients and their families are initially weekly and become less frequent as progress is made. Beginning stages of FT-AN focus on practical support for weight gain, symptom improvement, and distress tolerance. Treatment in Phase 1 focuses on engagement in therapy, including developing the therapeutic alliance. Phase 2 focuses on supporting the family to manage the ED symptoms until the patient can have more independence. Phase 3 focuses on developmental and family needs and is individually tailored (e.g., school-related issues, peer relationships, and eating independently). Phase 4 focuses on treatment discharge.

At MCCAED, adolescents completed a diagnostic interview at admission, and assessments at admission and discharge as part of routine clinical practice. Adolescents attended an initial clinical assessment to establish diagnosis. Treatment-related characteristics such as length of treatment and treatment type were retrieved from the electronic Patient Journey System.

For this study, participants were selected based on (a) having a restrictive ED, such as AN, "atypical" AN or OSFED, and (b) completing at least one session of FT-AN at MCCAED, either in the outpatient or ITP clinic. No participants had received any exposure and response prevention (ERP) therapy for OCD or any other disorder.

1.3 | Measures

1.3.1 | ChOCI-R-S

The Child Obsessional Compulsive Inventory—Self Report is a 38-item measure of obsessive-compulsive thoughts and behaviors in children (Shafran et al. 2003; Uher et al. 2008). Symptoms are rated on a 3-point scale and summed to create the Total Symptom Score (TSS; range 0–40). Impairment is rated on a 5-point scale and summed to create the Total Impairment Score (TIS; ranges 0–48). TIS scores >17 are clinically significant. There is no specific reporting period for symptoms; instead, answers are "how much you agree with the statement" or "how much you think this is true of you."

1.3.2 | EDEQ-A

The Eating Disorder Examination Questionnaire (EDE-Q) was adapted for use with adolescents, with reporting periods shortened from the previous 28 days to the previous 14 days and some language simplified (Carter et al. 2001). The scale still contains 36 items and yields the same global score and four subscales (i.e., weight, shape, eating concern, and restraint). The clinical cut-off has been proposed to be 2.8 (Mond et al. 2008; Velkoff et al. 2023).

1.3.3 | %mBMI

Percentage median body mass index (BMI) is calculated by dividing an individual's BMI by the 50th percentile BMI for their age, sex, and height. %mBMI is often used with adolescents, rather than weight, due to normative growth during the adolescent period. %mBMI is used routinely at MCCAED to inform diagnosis, treatment aspects such as meal planning, and level of risk severity, including risk for hospital admission.

1.3.4 | HONOSCA

The Health of the Nation Outcome Scales for Children and Adolescents is a 13-item questionnaire focusing on general health and social functioning (Gowers et al. 1999). Youth self report over the past 2 weeks from 0 (Not at all) to 4 (Severely), which are summed to create a total score from 0 to 52. Higher scores indicate more general impairment.

1.3.5 | DAWBA

The Development and Well-Being Assessment (DAWBA) (Goodman et al. 2011) is a structured assessment that provides predicted psychiatric diagnoses based on DSM-5 and ICD-10 criteria (American Psychiatric Association 2013; World Health Organization 2004). Therefore, rating periods depend on the relevant diagnosis. Results are given in bands from 0 to 5 indicating the probability of meeting diagnostic criteria for the disorder: 0 ($<0.1\%$), 1 (~0.5%), 2 (~3%), 3 (~15%), 4 (~50%), and 5 ($\geq 70\%$). Consistent with previous research, computer-predicted diagnosis was considered present for those in the top two bands of disorder probability (50% and $\geq 70\%$ likelihood) (Goodman et al. 2011). These bands have shown to be decent estimates, especially in exchange for decreased economic costs relative to clinician-administered diagnosis, although they may underestimate prevalences by 20%–30% (Goodman et al. 2011). Eating disorder diagnosis was confirmed at the intake clinical assessment.

1.4 | Demographics

We examined demographic information (age, biological sex, gender, and race/ethnicity) with outcome measures. Ethnicity was coded as White versus non-White to examine the effects of systemic racism while maximizing power, especially given small cell sizes for each non-White subgroup (see Table 1). Biological sex and gender were almost perfectly multicollinear, but more participants had a response for biological sex, so only biological sex was used. Age was significantly associated with discharge scores for the EDEQ-A Global ($r=0.47$, $p<0.001$), ChOCI TIS ($r=0.40$, $p=0.002$), %mBMI ($r=-0.29$, $p=0.001$), and HONOSCA ($r=0.37$, $p=0.004$). Sex was significantly associated with discharge scores for the EDEQ-A Global ($r=0.41$, $p=0.002$), ChOCI TIS ($r=0.39$, $p=0.002$), and HONOSCA ($r=0.45$, $p<0.001$). Ethnicity was not significantly associated with any discharge scores. Demographics that demonstrated significant associations with at least one outcome were added step-wise to regression analyses; therefore, age and biological sex were included as model covariates.

1.5 | Analyses

Analyses were intent-to-treat such that participants could have completed any amount of FT-AN. Missing data were excluded pairwise across analyses; for instance, for a pre/post analyses, only individuals with the relevant measure at both admission and discharge were included, but could be included in other

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TABLE 1 | Baseline demographics.

	Full sample	ChOCI TIS response			ChOCI TSS response		
Characteristic	***N*=150**	**NR *n*=25**	**R *n*=27**	**Unknown *n*=98**	**NR *n*=25**	**R *n*=29**	**Unknown *n*=96**
Diagnosis							
AN	104 (69%)	20 (80%)	20 (74%)	64 (65%)	19 (76%)	22 (76%)	63 (66%)
Atypical AN	29 (19%)	2 (8%)	4 (15%)	23 (23%)	2 (8%)	5 (17%)	22 (23%)
EDNOS/OSFED	17 (11%)	3 (12%)	3 (11%)	11 (11%)	4 (16%)	2 (6.9%)	11 (11%)
Ethnicity							
Asian or Asian British (including Chinese)	8 (5%)	1 (4%)	0 (0%)	7 (7%)	1 (4%)	0 (0%)	7 (7%)
Black or Black British	6 (4%)	1 (4%)	1 (4%)	4 (4%)	1 (4%)	1 (3%)	4 (4%)
Mixed or multiple ethnic origins	21 (14%)	1 (4%)	4 (15%)	16 (16%)	3 (12%)	3 (10%)	15 (16%)
Other ethnic group (please specify)	6 (4%)	0 (0%)	1 (4%)	5 (5%)	0 (0%)	1 (3%)	5 (5%)
Prefer not to say	2 (1%)	0 (0%)	0 (0%)	2 (2%)	0 (0%)	0 (0%)	2 (2%)
White, White European or Caucasian	107 (71%)	22 (88%)	21 (78%)	64 (65%)	20 (80%)	24 (83%)	63 (66%)
Sex							
Female	132 (88%)	24 (96%)	20 (74%)	88 (90%)	23 (92%)	22 (79%)	86 (90%)
Male	18 (12%)	1 (4.0%)	7 (26%)	10 (10%)	2 (8%)	6 (21%)	10 (10%)
Gender							
Female	124 (82%)	24 (96%)	19 (73%)	81 (81%)	23 (92%)	22 (76%)	78 (81%)
Male	15 (10%)	1 (4%)	7 (27%)	7 (8%)	2 (8%)	6 (21%)	7 (8%)
Nonbinary/other	1 (1%)	0 (0%)	0 (0%)	1 (1%)	0 (0%)	0 (0%)	1 (1%)
Not reported	11 (7%)	0 (0%)	0 (0%)	10 (10%)	0 (0%)	1 (3%)	10 (10%)

Note: $N=150$. Unknown ChoCI response means participants did not report either a baseline or end-of-treatment ChOCI score, so a change score could not be calculated.
Abbreviations: AN, anorexia nervosa; EDNOS, eating disorder not otherwise specified; NR, nonresponse, or a symptom change of greater than or equal to zero; OSFED, other specified feeding and eating disorder; R, Response, or a symptom change of less than zero.

analyses if those measures were completed. Further, some individuals had an EDEQ-A Global score of zero ($n=22$), despite meeting criteria for an ED diagnosis based on clinical assessment and presentation, suggesting significant under-reporting. Therefore, analyses were re-run only including individuals with (1) EDEQ-A Global scores >0 ($n=128$) and (2) EDEQ-A Global scores ≥ 2.8 ($n=95$) to examine possible effects of under-reporting (see Supplement).

1.5.1 | Pre/Post Changes

EDEQ-A Global, ChOCI total impairment, ChOCI total symptom, %mBMI, and HONOSCA were all significantly nonnormally distributed based on Kolmogorov–Smirnov tests. Therefore, nonparametric Wilcoxon signed-rank tests were used to examine pre/post differences across treatment.

1.5.2 | Linear Regressions

Linear regression models were tested for assumptions, including heteroscedasticity via Breusch-Pagan tests, autocorrelation through Durbin Watson tests, and normality with Shapiro–Wilk tests. No models were significantly heteroskedastic, autocorrelated, or nonnormal. Interactions investigated the effect of baseline ChOCI scores on changes in (1) EDEQ-A Global scores and (2) %mBMI across treatment.

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1.5.3 | Correlations

Correlations were used to examine baseline variables in relation to change across treatment in other variables (calculated as Discharge—Admission) using Kendall's rank correlation tau due to nonnormality of pre/post data.

Fifty-two individuals had ChOCI impairment scores at both admission and discharge. Of these, 27 individuals (52%) saw improvements in ChOCI total impairment (calculated as a change score greater than zero; "responders") whereas 25 (48%) stayed the same or worsened ("nonresponders"). Similarly, 54 individuals had ChOCI symptom scores at both admission and discharge. Among these, 29 (54%) individuals saw improved ChOCI total symptom scores and 25 (46%) stayed the same or worsened. Given the large number of individuals who saw no reduction or worsening of their OCD symptoms over treatment, we ran analyses examining effects comparing responders to nonresponders.

1.6 | Lived Experience Involvement Statement

No specific efforts were undertaken to involve persons with lived experience in the study design or execution, or in the preparation of this manuscript.

2 | Results

Participant demographics are described in Table 1 ($N=150$). Baseline values for each measure are included in Table 2, and comorbid diagnoses based on the DAWBA are in Table 3. The number of individuals with discharge measures varied (ChOCI TSS = 61, ChOCI TIS = 60, EDEQ-A = 53, %mBMI = 136, HONOSCA = 60) and was lower than the number with intake measures (ChOCI TSS = 143, ChOCI TIS = 142, EDEQ-A = 128, %mBMI = 149, HONOSCA = 146). Participants with and without self-reported measures at discharge did not differ significantly based on any demographic or clinical admission scores. Among participants who completed the DAWBA, 10 (8.8%) met for a likely OCD diagnosis. The average ChOCI TIS score was at the clinical cutoff of 17 ($M=17.0$, SD = 9.3), indicating clinically significant OCD-related impairment on average (Shafran et al. 2003).

2.1 | Pre/Post Improvements

Across treatment, there were significant improvements in EDEQ-A Global scores ($V=1078$, $p<0.001$) and %mBMI ($V=429.5$, $p<0.001$). However, neither ChOCI total impairment ($V=730$, $p=0.080$) nor ChOCI total symptom scores ($V=804$, $p=0.108$) significantly improved. HONOSCA also did not significantly improve across treatment ($V=1050$, $p=0.076$).

2.2 | Linear Regressions

Interactions examined the effect of baseline ChOCI scores on changes in eating pathology and weight across treatment. Neither

TABLE 2 | Baseline clinical values.

Baseline variable	Mean	SD	Min	Max
Age (years)	14.40	1.62	10.00	17.00
Treatment length (months)	7.73	4.52	0.00	22.33
%mBMI	84.59	10.74	63.50	132.42
HONOSCA total	14.76	8.56	0.00	50.00
WSAS total	16.20	8.74	0.00	40.00
RCADS total internalizing	63.44	25.57	9.00	124.00
RCADS total anxiety	47.89	20.25	6.00	98.00
RCADS social phobia	17.30	5.66	2.00	27.00
RCADS panic disorder	9.17	6.51	0.00	25.00
RCADS major depression	15.55	6.85	0.00	30.00
RCADS separation anxiety	6.09	4.24	0.00	19.00
RCADS GAD	8.87	4.13	1.00	18.00
RCADS OCD	6.35	4.21	0.00	18.00
ChOCI compulsion symptom	5.38	3.91	0.00	18.00
ChOCI compulsion impairment	9.10	5.56	0.00	20.00
ChOCI obsession symptom	6.20	4.79	0.00	19.00
ChOCI obsession impairment	8.60	5.49	0.00	20.00
ChOCI Total Symptom Score	11.58	7.92	0.00	33.00
ChOCI Total Impairment Score	16.97	9.29	0.00	39.00
FFOCI inflexibility	11.46	3.24	4.00	20.00
YOU-C undercontrol	30.29	11.29	12.00	62.00
YOU-C overcontrol	55.53	14.34	23.00	92.00
EDEQ-A restraint	3.58	1.91	0.00	6.00
EDEQ-A shape	4.21	1.71	0.00	6.00
EDEQ-A weight	3.91	1.63	0.00	6.00
EDEQ-A eating	3.24	1.49	0.00	5.60
EDEQ-A global	3.73	1.54	0.10	5.87

Note: $N=150$. For baseline values by ChOCI responder status, see Supplemental Table S1.

Abbreviations: %mBMI, percent median body mass index; ChOCI, child obsessional compulsive inventory; EDEQ-A, Eating Disorder Examination Questionnaire—adolescent version; FFOCI inflexibility, five factor obsessive compulsive inventory—inflexibility subscale (Samuel et al. 2012); HONOSCA, health of the nation outcome scales for children and adolescents; RCADS, Revised Child Anxiety and Depression Scale (Chorpita et al. 2000, 2005); WSAS, Work and Social Adjustment Scale (Mundt et al. 2002); YOU-C, youth over- and under-control (Lenz et al. 2021).

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TABLE 3 | DAWBA diagnoses.

Diagnosis	Likely yes	Percent	Likely not	Unknown
Depression	47	40.5	69	34
GAD	34	29.3	82	34
Social phobia	28	23.9	89	33
BDD	28	23.9	89	33
ODD	15	15.3	83	52
OCD	10	8.8	104	36
Specific phobia	8	7	107	35
Separation anxiety—DSM	8	7.1	105	37
Panic	6	5.3	108	36
Conduct	6	5.4	106	38
Separation anxiety—ICD	6	5.3	107	37
Agoraphobia	5	4.4	109	36
ADHD—DSM	3	3.1	95	52
ADHD—ICD	2	2	96	52
Tic—ICD	2	2	96	52
ASD/PDD—DSM	2	2.1	95	53
Tic—DSM	1	1	97	52
DMDD	0	0	12	138
ASD/PDD—ICD	0	0	94	56
PTSD	0	0	113	37

Note: $N = 150$. Likely yes represented the number of individuals with the predicted psychiatric diagnosis in the top two bands, 4 (~50%), and 5 (≥70%). Percent included the percentage of participants with a likely diagnosis, out of the number of participants who complete the module. Likely not represented the other bands, 0–3 (<50%). Unknown represented participants who had not completed that module. Depression = major depressive disorder.
Abbreviations: ADHD, attention-deficit/hyperactivity disorder; ASD/PDD, autism spectrum disorder/pervasive developmental disorder; BDD, body dysmorphic disorder; DAWBA, the Development and Well-Being Assessment; DMDD, disruptive mood dysregulation disorder; DSM, diagnostic and statistical manual of mental disorders; GAD, generalized anxiety disorder; ICD, international classification of diseases; OCD, obsessive compulsive disorder; ODD, oppositional defiant disorder; PTSD, post-traumatic stress disorder; Tic, tic disorder.

admission ChOCI TIS nor TSS had a significant interaction with admission EDEQ-A Global scores when predicting discharge EDEQ-A Global scores. This remained when age and sex were added. Similarly, admission ChOCI TIS did not interact with admission %mBMI in predicting discharge %mBMI. However, admission ChOCI TSS significantly interacted with admission %mBMI predicting discharge %mBMI, even after adding age and sex to the model. In other words, lower ChOCI TSS, compared to higher ChOCI TSS, was associated with more weight gain in %mBMI across treatment, but the effect size was small (Table 4).

2.3 | Correlations

2.3.1 | Baseline Functioning Correlated With Change Across Treatment

Next, the relationship between baseline functioning and change in clinical outcomes was examined (Table 5). First, baseline ChOCI scores were examined with changes in clinical measures. Higher baseline ChOCI TIS was associated with more improvements in EDEQ-A Global scores, although baseline ChOCI TSS was not associated with change in EDEQ-A Global. Conversely, lower baseline ChOCI TSS was associated with more weight gain in %mBMI. However, baseline ChOCI TIS was not associated with change in %mBMI. Neither baseline ChOCI score was associated with change in HONOSCA. Baseline EDEQ-A Global scores were not associated with change in either ChOCI score. Lower baseline EDEQ-A Global was correlated with less improvement in HONOSCA and more improvement in %mBMI. Next, baseline %mBMI was not associated with change in any outcome, including changes in EDEQ-A Global, either ChOCI score, or HONOSCA. Lastly, baseline HONOSCA was not significantly correlated with change in either ChOCI score; however, lower HONOSCA was associated with more improvements in %mBMI. Moreover, higher baseline HONOSCA was associated with more improvement in EDEQ-A Global scores.

Individuals with more baseline OCD-related impairment tended to see more improvements in ED symptoms, with a medium effect size, whereas those with fewer baseline OCD symptoms tended to see more improvements in weight, with a small-to-medium effect size. Therefore, we ran post hoc tests of correlations between these variables to better understand these findings. Baseline OCD-related impairment and OCD symptoms were moderately-to-strongly correlated ($r_\tau = 0.38$, $p < 0.001$), and change in ED symptoms was not correlated with change in weight ($r = -0.02$, $p = 0.86$). The correlation between OCD-related impairment and ED symptoms was stronger, with a larger effect size, than the correlation between OCD symptoms and weight.

2.4 | OCD Responders Versus NonResponders

In analyses comparing OCD responders to OCD nonresponders at baseline, there were no significant differences in: age, sex, EDEQ-A, CHOCI TSS, %mBMI, or diagnoses. The only difference was on ChOCI TIS baseline scores, with higher baseline TIS scores demonstrating higher reductions in TSS and TIS across treatment.

The significant correlations between baseline functioning and change in clinical outcomes when including the entire sample (above) were then examined with just OCD responders (Table 5).

3 | Discussion

This study examined changes in ED and OCD symptoms across Maudsley family treatment for adolescent restrictive EDs, which

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TABLE 4 | Interactions.

Predicting EDEQ global at discharge ($n = 46$)						
Parameter	**Coefficient**	**SE**	**95% CI**	***t*(40)**	***p***	**Sign.**
Intercept	−6.60	1.83	(−10.19, −3.01)	−3.60	< 0.001	**
ChOCI TIS Admission	0.02	0.07	(−0.11, 0.15)	0.34	0.736	
EDEQ GS admission	0.55	0.25	(0.05, 1.05)	2.17	0.03	*
Age	0.32	0.14	(0.05, 0.59)	2.35	0.019	*
Sex	1.22	0.6	(0.04, 2.40)	2.03	0.042	*
ChOCI TIS × EDEQ global admission	−0.01	0.02	(−0.04, 0.02)	−0.78	0.437	
Predicting EDEQ global at discharge ($n = 47$)						
Parameter	**Coefficient**	**SE**	**95% CI**	***t*(41)**	***p***	**Sign.**
Intercept	−6.37	1.79	(−9.89, −2.85)	−3.55	< 0.001	**
ChOCI TSS admission	0.04	0.08	(−0.12, 0.21)	0.51	0.613	
EDEQ global admission	0.42	0.21	(0.01, 0.83)	2.02	0.043	*
Age	0.31	0.14	(0.04, 0.58)	2.27	0.023	*
Sex	1.23	0.59	(0.07, 2.38)	2.08	0.037	*
ChOCI TSS × EDEQ global admission	−0.01	0.02	(−0.05, 0.03)	−0.62	0.534	
Predicting %mBMI at discharge ($n = 122$)						
Parameter	**Coefficient**	**SE**	**95% CI**	***t*(116)**	***p***	**Sign.**
Intercept	92.62	12.72	(67.69, 117.56)	7.28	< 0.001	**
ChOCI TSS admission	−1.85	0.75	(−3.33, −0.37)	−2.46	0.014	*
%mBMI admission	0.25	0.13	(0.00, 0.50)	2.01	0.044	*
Age	−1.34	0.41	(−2.15, −0.53)	−3.26	0.001	**
Sex	0.92	2.01	(−3.03, 4.87)	0.46	0.648	
ChOCI TSS × %mBMI admission	0.02	0	(0.00, 0.04)	2.15	0.031	*
Predicting %mBMI at discharge ($n = 122$)						
Parameter	**Coefficient**	**SE**	**95% CI**	**t(116)**	***p***	**Sign.**
Intercept	93.72	15.03	(64.27, 123.18)	6.24	< 0.001	**
ChOCI TIS admission	−1.08	0.64	(−2.33, 0.17)	−1.69	0.092	
%mBMI admission	0.25	0.15	(−0.05, 0.55)	1.63	0.102	
Age	−1.44	0.43	(−2.28, −0.59)	−3.35	< 0.001	**
Sex	0.4	2.09	(−3.69, 4.48)	0.19	0.849	
ChOCI TIS × %mBMI admission	0.01	0	(−0.00, 0.03)	1.62	0.106	

Note: Sign. = significance where ** = $p < 0.01$ and * = $p < 0.05$, Admission = score at treatment admission, Discharge = score at treatment discharge.
Abbreviations: ChOCI, child obsessional compulsive inventory; EDEQ Global, Eating Disorder Examination Questionnaire—Adolescent version Global Scale; TIS, Total Impairment Score; TSS, Total Symptom Score.

included patients with AN, AAN, and OSFED with clinically significant OCD on average. ED pathology and weight improved following FT-AN, but OCD and overall impairment did not. However, supplemental analyses excluding possible under-reporters of eating pathology suggested significant improvement in all symptoms.

Higher baseline OCD *impairment* did not impair ED pathology change and, in fact, was correlated with more improvement, both overall and in OCD responder subgroups. In terms of weight, the opposite relationship was initially seen with higher baseline OCD *symptoms* associated with less change in %mBMI, but this relationship did not hold when examining OCD responders, or after removing under-reporters. Further, lower baseline ED pathology was also associated with less improvement in weight, suggesting that less change in weight across treatment is likely a function of more severe psychopathology overall rather than

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TABLE 5 | Correlations between baseline symptomology and treatment-related changes.

		All			TSS responders		TIS responders	
Baseline	**Change**	**Higher baseline, change improvement**	r_τ	***p***	r_τ	***p***	r_τ	***p***
ChOCI TSS	EDEQ-A	—	−0.115	0.258	—	—	—	
	%mBMI	Less	−0.198	<0.001**	−0.222	0.104	−0.186	0.181
	HONOSCA	—	−0.074	0.440	—	—	—	
ChOCI TIS	EDEQ-A	More	−0.298	0.004**	−0.294	0.033*	−0.294	0.042*
	%mBMI	—	−0.050	0.400	—	—	—	—
	HONOSCA	—	−0.178	0.064	—	—	—	—
EDEQ-A Global	ChOCI TSS	—	−0.066	0.525	—	—	—	—
	ChOCI TIS	—	−0.122	0.244	—	—	—	—
	%mBMI	Less	−0.186	0.003**	−0.160	0.243	−0.010	0.944
	HONOSCA	More	−0.258	0.01*	−0.412	0.003**	−0.357	0.014*
%mBMI	ChOCI TSS	—	0.063	0.514	—	—	—	—
	ChOCI TIS	—	0.031	0.752	—	—	—	—
	EDEQ-A	—	−0.059	0.557	—	—	—	—
	HONOSCA	—	0.044	0.629	—	—	—	—
HONOSCA	ChOCI TSS	—	−0.181	0.064	—	—	—	—
	ChOCI TIS	—	−0.081	0.410	—	—	—	—
	EDEQ-A	More	−0.227	0.03*	−0.142	0.294	−0.135	0.349
	%mBMI	Less	−0.151	0.011*	−0.229	0.092	−0.127	0.358

Note: Higher baseline, change improvement: Higher baseline scores on the baseline variable were associated with significantly what kind of improvement (more or less) in the change variable, where improvement represents less psychopathology or increased weight. For example, more means higher baseline scores were associated with more improvement, or less pathology, in the change variable. TSS responders = those with a change score greater than zero on the Child Obsessional Compulsive Inventory Total Symptom Score, $n = 54$. TIS responders = those with a change score greater than zero on the Child Obsessional Compulsive Inventory Total Impairment Score, $n = 52$. EDEQ-A Global = Eating Disorder Examination Questionnaire – Adolescent version Global Scale, Admission = score at treatment admission, Discharge = score at treatment discharge, HONOSCA = The Health of the Nation Outcome Scales for Children and Adolescents, %mBMI = percentage median body mass index. Change scores were calculated as Discharge—Admission. Correlations for OCD responders were only calculated for correlations that were significant when including all participants.

specific impairment due to OCD presence. Therefore, FT-AN as a treatment for restrictive EDs does not seem to be directly inhibited by higher OCD severity. Further, FT-AN did not seem to target OCD symptoms until under-reporting of symptoms at baseline was accounted for.

In the main analyses, ED symptomology and weight improved across FT-AN treatment while OCD and general impairment scores did not. This mirrors the broader literature, including in other family therapies like FBT, wherein ED symptoms and weight tend to improve across ED treatment but comorbid OCD symptoms do not (Agras et al. 2014; Lock et al. 2018; Schmidt et al. 2015). This finding highlights the behavioral focus of FT-AN, targeting almost exclusively eating-related behaviors such that FT-AN may not be sufficient to address comorbid OCD symptoms or noneating-related cognitive rigidity, leading to minimal change in OCD symptoms and general impairment. However, previous research has typically examined samples where OCD symptoms are minimal, and exclusively in outpatient settings (Blachno et al. 2014; Steinhoff et al. 2025). Therefore, this investigation uniquely extended these findings to a sample with clinical levels of OCD on average, across both outpatient and intensive outpatient settings, providing critical evidence that previous nonimprovement of OCD symptoms in family treatment was likely not just attributable to floor effects. We also extended previous results to examine general impairment across treatment with HONOSCA, showing that nonspecific impairment also does not significantly improve across FT-AN, despite overall ED improvements. This suggests that addressing OCD symptomology is also needed to improve patients' overall quality of life and general functioning. However, previous studies have not excluded individuals with significant symptom under-reporting. After excluding individuals who reported low EDEQ-A scores despite meeting criteria for an ED, significant improvements *were* seen across all symptoms, including OCD symptoms, OCD impairment, and HONOSCA. Therefore, the nonimprovement in OCD symptomology in this study and in prior literature may be a result of smaller improvements in OCD symptoms versus ED symptoms, compounded by patients under-reporting symptoms. More research is needed to disentangle the effects of weight restoration versus ED symptom reduction on OCD symptom improvement.

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Higher OCD symptoms have been theorized to impair ED treatment due to their associations with worsened outcomes, but these studies have often not accounted for baseline severity or included participants with clinically significant OCD pathology (Blachno et al. 2014). In our study, baseline OCD did not interact with changes in ED symptoms across treatment. While caution is needed when interpreting the lack of significant findings, we also saw that higher baseline OCD impairment was associated with more ED pathology improvement. Therefore, higher OCD impairment was, surprisingly, associated with *more* reductions in ED pathology across FT-AN. On the other hand, higher baseline OCD *symptoms* were associated with less change in weight, although this relationship did not hold when examining OCD responders, or when excluding for possible under-reporting, suggesting it is less robust. Further, lower baseline ED pathology was also associated with less weight gain, suggesting that changes in weight across treatment are likely a function of overall psychopathology severity rather than specific impairment due to OCD presence. Findings provide preliminary evidence that worsened ED outcomes observed when OCD is comorbid may represent higher initial severity, rather than impaired treatment effects. Therefore, obsessions, compulsions, and the resulting impairment may not prevent FT-AN from working. This may partially be explained by the role of caregivers, rather than patients, driving refeeding and recovery. Whether or not patients experience some improvements in OCD, those with OCD symptoms at baseline likely will need OCD treatments integrated into care or provided afterwards. ERP is likely to be effective in this realm. Not only is ERP a first-line treatment for OCD (The National Institute for Health and Care Excellence (NICE) 2005), it is also the only treatment currently known to consistently show reductions in both OCD and AN symptoms (Steinhoff et al. 2025; Simpson et al. 2013). Specifically, ERP may be especially beneficial for those with higher baseline ED symptomology since these patients saw less improvements in weight across FT-AN. In this study, changes in OCD were not associated with any baseline variable, and no associations between variables seemed to specifically characterize OCD "responders." However, future research should investigate factors that may distinguish OCD "responders" from "nonresponders" to better allocate OCD-specific treatments for improved outcomes. Moreover, family therapy remains the first-line treatment for adolescent AN. Therefore, it is unknown whether OCD-specific care should be integrated as soon as possible, or should wait until weight restoration occurs, especially since ERP has also been shown to reduce AN symptoms (Steinhoff et al. 2025; Simpson et al. 2013). While ED treatment is often prioritized due to medical sequelae and risks to patient safety, and there have been theories that low weight could impair ERP treatment (Steinglass 2007), there is no known empirical evidence in this realm, necessitating further research.

Both restrictive EDs and OCD share obsessive-compulsive features (Dingemans et al. 2022) and have been theorized as distinct because they manifest with different content (Altman and Shankman 2009). For instance, in both disorders negative affect from feared outcomes, such as weight gain or intrusive thoughts, is decreased by compulsive behaviors, including restriction (Altman and Shankman 2009; Swinbourne and Touyz 2007). This conceptualization aligns with the targeted mechanisms in ERP, which may help explain why ERP improves both sets of symptoms. Specifically, ERP focuses on reducing compulsions, offering benefits for both disorders regardless of the obsessional content. Even the validity of labeling AN and OCD as distinct disorders has been called into question given substantial overlaps, although ultimately differences have often been identified including in neural processes, genetic risk, and metabolic associations (Yilmaz et al. 2023, 2020; Serpell et al. 2002; Thomas et al. 2022). Further, AN specifically is highly associated with medical complications and morbidity (Trapani and Rubino 2025). In this way, the improvements in ED symptoms, but not OCD symptoms after FT-AN in the main analyses suggests FT-AN is targeting mechanisms unique to restrictive EDs, reinforcing the notion that these are distinct phenomena. OCD symptomology improving when under-reporters were excluded may point to FT-AN reducing obsessive-compulsive symptoms by reducing starvation syndrome (Spettigue et al. 2025). However, more work is needed to better understand the overlap and divergence between the disorders, for more accurate prevention efforts and improved treatment outcomes.

3.1 | Limitations

DAWBA OCD diagnoses were computer-based and not checked or administered by clinicians. Based on this, participants are labeled as having a high likelihood of having OCD. However, the DAWBA decreases financial and clinical burden, allowing for routine collection of comorbid diagnoses that are not always reported in ED research. Since data were collected secondary to care previsioning, participants received different lengths of FT-AN. This represents the reality of ED treatment, and FT-AN is designed to vary based on patient and family needs, but introduces variability that could explain differences in outcomes. However, treatment length was not accounted for in analyses given insufficient power for additional covariates. In the same vein, many participants did not complete discharge measures. While it is less likely that our results were systematically impacted since baseline measures were the same across discharge completers and noncompleters, we cannot rule this out. We also saw a decreased sample size, and therefore diminished power, due to participants not filling out measures at discharge. Lastly, the EDEQ-A rates symptoms across the past 14 days, whereas the ChOCI does not include a specified time period. Although the ChOCI has been consistently used in similar research studies, this could partially account for less improvement in OCD symptoms.

This study did not have a comparator condition as patients at MCCAED start with FT-AN and are allocated to additional treatments after, if needed. Therefore, caution is needed when interpreting the causes of change. Phenomena like regression-to-the-mean could be at play in addition to improvements from treatment. However, this investigation built off prior research that did include comparator treatments and randomized controlled trials when examining the effects of family therapies on AN and OCD symptom reductions (Steinhoff et al. 2025), lending more credibility to our conclusions despite the lack of a comparator condition. Lastly, while this sample was largely racially representative of the United Kingdom (Office for National Statistics (ONS) 2022), and included more men than typical clinical samples (Sweeting et al. 2015), future work is needed

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specifically to examine these effects in historically marginalized populations.

3.2 | Strengths

Our study included patients with more severe OCD symptomology than previous studies, in addition to reporting on likely OCD diagnostic status, addressing critical gaps in the literature (Steinhoff et al. 2025). Moreover, we included supplemental robustness analyses to examine the effects after accounting for possible under-reporting, which has not been done previously and is suggested for future research. In this vein, there is still limited research into the efficacy of treatments for comorbid OCD and restrictive EDs, despite high comorbidity. Previous reviews have focused on AN (Steinhoff et al. 2025), with a need for future efforts to include all restrictive EDs like we did in this study. In fact, our inclusion of disorders across the restrictive spectrum is a strength, particularly given growing evidence of similar symptomology and severity across these disorders (Fitterman-Harris et al. 2024; Johnson-Munguia et al. 2024; Sandoval-Araujo et al. 2024). Similarly, the proportion of men in this study (18%) is consistent with estimates that men comprise 10%–25% of those with EDs, and was higher than typical clinical samples which have to have 10% or fewer men (Sweeting et al. 2015).

We demonstrated unique associations with %mBMI compared to EDEQ-A pathology, highlighting the benefits of our including both constructs in analyses. We also included a measure of nonspecific impairment to assess functioning not tied to OCD or ED symptoms, representing a more transdiagnostic measure of outcomes. Future studies should continue to examine eating pathology separate from weight outcomes, and investigate transdiagnostic markers of treatment success.

4 | Conclusions

Across FT-AN, ED symptomology improved while OCD pathology and general impairment did not. However, when excluding likely under-reporters, OCD symptoms and overall functioning did significantly improve, suggesting FT-AN may offer improvements in these areas, albeit with smaller effects. More severe baseline OCD impairment did not seem to directly hinder, and in fact was related to more ED improvement. More severe baseline ED and OCD symptoms were associated with less improvement in weight. These results likely suggest that worsened outcomes among patients with OCD represent higher initial severity rather than treatment inhibition due to OCD presence. Future research should examine efficacy and optimal timing of incorporating OCD-specific treatments into care, particularly with treatments that have also shown improvements in ED symptoms such as ERP.

Author Contributions

Molly Fennig Steinhoff: conceptualization, methodology, formal analysis, visualization, project administration, writing – original draft. **Julian Baudinet:** conceptualization, methodology, investigation, writing – review and editing. **Anna Konstantellou:** data curation, writing – review and editing. **Lucinda Gledhill:** data curation, writing – review and editing. **Serena J. Gu:** writing – review and editing, visualization. **Kirsten E. Gilbert:** conceptualization, methodology, writing – review and editing, supervision.

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AI was only used for code debugging, which was validated by checking code outputs. AI was not used for any other purposes.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Supplemental Table S1:** Baseline clinical values by ChOCI responder status.

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