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ORIGINAL ARTICLE

An exploratory study on the intergenerational transmission of dieting proneness within an eating disorder population

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Abstract

An Exploratory Study on the Intergenerational Transmission of Dieting Proneness within an Eating Disorder Population (IRB Protocol Number: 160928271).

Purpose Parents and families are not the sole factor in eating disorder (ED) development and their involvement in recovery is crucial. However, parents provide a social and environmental context for a child's eating and weight that cannot be completely discounted. The purpose of this study was to explore the intergenerational transmission of dieting behavior within an ED sample.

Methods Participants ($N=65$) were recruited for this cross-sectional study through four distinct ED treatment sites. Participants completed a questionnaire that was developed previously to examine parental feedback as predictor variables, as well as completing the Eating Pathology Severity Index (EPSI) as an outcome variable. A total of 60 completed the questionnaire items of interest to be included in the analyses. SAS JMP® 13.0 was used for descriptive analyses, correlations, and multivariable linear regressions.

Results Results of the multivariable linear regression showed that the amount of variance explained by the final model for eating pathology severity (via the EPSI) doubled when parental feedback was included (Model 1: $R^2=0.09$, Model 2: $R^2=0.20$). Additionally, there was a significant relationship between the "Negative Direct Parental Feedback Subscale" and EPSI total scores ($\beta=14.1$; SD = 7.0; $p=0.05$).

Conclusion These findings of increased eating pathology associated with direct parental feedback in a clinical population of ED participants even when controlling for parental ED history suggests greater attention is needed within the ED literature on social and environmental factors and their potential associations with eating pathology.

Level of evidence Level V, descriptive study.

Keywords Disordered eating · Dieting behaviors · Fat talk · Family fat talk · Eating disorders · Intergenerational transmission

Introduction

Complex diseases, especially mental health conditions, commonly have a genetic underpinning as well as social or environmental factors or triggers; eating disorders (ED)

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are no different. Weight dissatisfaction and dieting are associated with greater disordered eating behaviors (i.e., subclinical symptoms associated with EDs such as fasting to lose weight) and diagnosable eating disorders, especially for those genetically predisposed to an ED [1]. Although EDs are also, brain-based disorders, the socio-environmental context, such as the association between EDs and familial factors, remains a critical area that could be mediated through intervention. For example, a focus on an intergenerational cycle of risk offers promise in understanding both the genetic predisposition that children of parents with EDs will face, as well as some potential triggering factors that could be present in that familial context [2].

The more recent genetic understanding of EDs has counteracted parent-blaming previously inherent in EDs research, especially in the 1980s and 1990s [3]. That blame created a counter-movement led by parent and caregiver groups such as the International Eating Disorder Action (IED Action) and Families Empowered and Supporting Treatment of Eating Disorders (F.E.A.S.T), which have effectively demonstrated how imperative parents and caregivers are in ED recovery [4]. Parents and families are not the sole factor in determining ED development and their involvement is a crucial piece in recovery, especially for a child or adolescent. However, parents simultaneously provide a social and environmental context for a child's eating and weight that cannot be completely discounted [5]. Parents model eating behaviors for a child from their earliest age and provide most meals when children are not in school, so the eating environment that a parent or caregiver provides can shape eating attitudes and behaviors.

This connection can theoretically be understood as social reinforcement, which is part of Social Cognitive Theory [6]. Social reinforcement consists of comments or actions from close others that help reinforce a certain behavior or idea. In this context, parental or family social reinforcement helps to support and perpetuate the thin ideal through weight-related criticism, encouragement to diet, or modeling dieting behaviors [7]. These external reinforcements can then be internalized by individuals, potentially leading to body dissatisfaction and eating pathology. Conversely, social reinforcement can also support positive behaviors, emphasizing the need for positive rather than negative reinforcement [6]. Although genetics may determine who will be likely to internalize triggers within this context and in which individuals eating pathology will occur, without those triggers that gene–environment nexus may not occur [8].

Additionally, Objectification Theory was also used as a theoretical underpinning for this study to understand the lived experiences and mental health risks based on the sexual objectification of primarily women's bodies, but also men's bodies [9]. This theory posits that girls and women particularly are acculturated to internalize another's perspective as the primary view of their physical selves. Therefore, if another person tells them that they need to lose weight or criticizes their shape or weight, they might internalize that perspective and view themselves through that observer's lens rather than how they previously saw themselves.

For example, a recent meta-analysis of peer and family associations in adolescents found moderate associations between family behavior and dieting, body dissatisfaction, and bulimic symptomatology ($r = 0.221$, 0.224, 0.225, respectively) [10]. Only one article considered the effect of parental modeling behavior (i.e., modeling eating or dieting behavior), so this indirect behavior could not be assessed; therefore, these moderate associations are from the direct association with parental encouragement to diet. However, among peers, modeling dieting had a greater association with bulimic symptoms than encouragement to diet or weight-related teasing. Thus, there is empirical support for sociocultural theoretical models of EDs that suggest the family context could be a mediator of weight concerns and disordered eating [10]. There is also, of course, a genetic correlation, as the same genetic risk for an ED could be expressed in both a parent and their child. Additionally, based on previous research of external pressures such as family, peers and media on EDs [11], we hypothesized that participants who recalled greater parental criticism or encouragement to diet would report greater ED symptomatology with higher Eating Pathology Symptom Inventory (EPSI) subscale scores [12]. More research is necessary, however, to understand both direct and indirect associations between family feedback and child's weight and body satisfaction.

Recent qualitative results with women (both with and without children) who had EDs or a history of an ED provided first-person accounts of intergenerational transmission [13]. Thematic analysis suggested women were highly concerned about intergenerational transmission of their EDs and wanted to model good behaviors and practice positive commentary for their children. Both direct and indirect pressures were consistently mentioned, both in terms of the participants' own upbringing (e.g., having a mother who dieted or encouraged them to diet, etc.) and in their fears of parenting their children (e.g., modeling ED behaviors, not knowing how to discuss weight). Therefore, even though parents do not cause EDs, parental modeling helps shape subsequent lifelong behaviors. Furthermore, parents and potential parents have expressed concern that their children could be susceptible to their behaviors or words.

The primary objective in this study was to understand direct and indirect parental feedback on a participant's weight and body image within a clinical cohort of patients with a current or past eating disorder. The purpose of this study was to explore these social and environmental triggers to understand the intergenerational transmission of dieting behavior within an ED sample. This study builds on

information garnered from a previous study in a community sample [5]. Due to the uncertainty of whether direct or indirect associations exert more pressure upon children, both direct and indirect associations were individually explored with their relation to ED symptomatology as an exploratory study aim. Previous research has proposed a revised Obesity and Dieting Proneness Theoretical Model based on Costanzo and Woody's original Obesity Proneness Model [5, 14], which was adapted to include indirect and direct mechanisms for parental feedback and parental concern about their own weight (see Fig. 1). For the purposes of this study, the revised Obesity and Dieting Proneness Theoretical Model was expanded slightly to also consider disordered eating as an outcome (see Fig. 1). This study provides a novel perspective to previously conducted research by considering indirect and direct parental feedback through a unique questionnaire in a clinical population.

Methods

Questionnaire development

A questionnaire used that has been detailed in previous research [5] was revised using feedback from experts in psychometrics, EDs, and social and behavioral research [see Appendix (Supplementary material) for survey]. Questions were reworded to provide Likert scale options for questions as well as two subscales that could provide scores for indirect and direct parental feedback. In addition, an ED-specific measure, the Eating Pathology Symptom Inventory (EPSI) was provided to participants to identify which ED symptoms are associated with direct and indirect feedback [12]. This inventory contains a total of 45 items on a Likert scale (ranging from Never to Very Often) and comprised eight subscales: Body Dissatisfaction, Binge Eating, Cognitive Restraint, Purging, Restricting, Excessive Exercise, Negative Attitudes towards Obesity, and Muscle Building. The EPSI has excellent convergent and discriminate validity, as well as internal consistency (alpha estimate range = 0.84–0.89) and test–retest reliability over a 2- and 4-week period (mean retest Pearson $r = 0.73$) [12]. The EPSI total score was internally reliable for this population with a Cronbach's alpha estimate of 0.93. Participants were also asked whether their mother or father had a history of an eating disorder.

Due to the nature of this revised questionnaire, we were better able to test the relationship between direct and indirect parental feedback on a child's concern about their weight and disordered eating. In order to determine whether direct or indirect feedback exert more pressure, the questionnaire included both types of parental feedback in two separate subscales with summary measures for each. Scores were averaged from individual items for each subscale, with higher scores in each subscale indicating greater negative indirect or direct parental feedback influence scores.

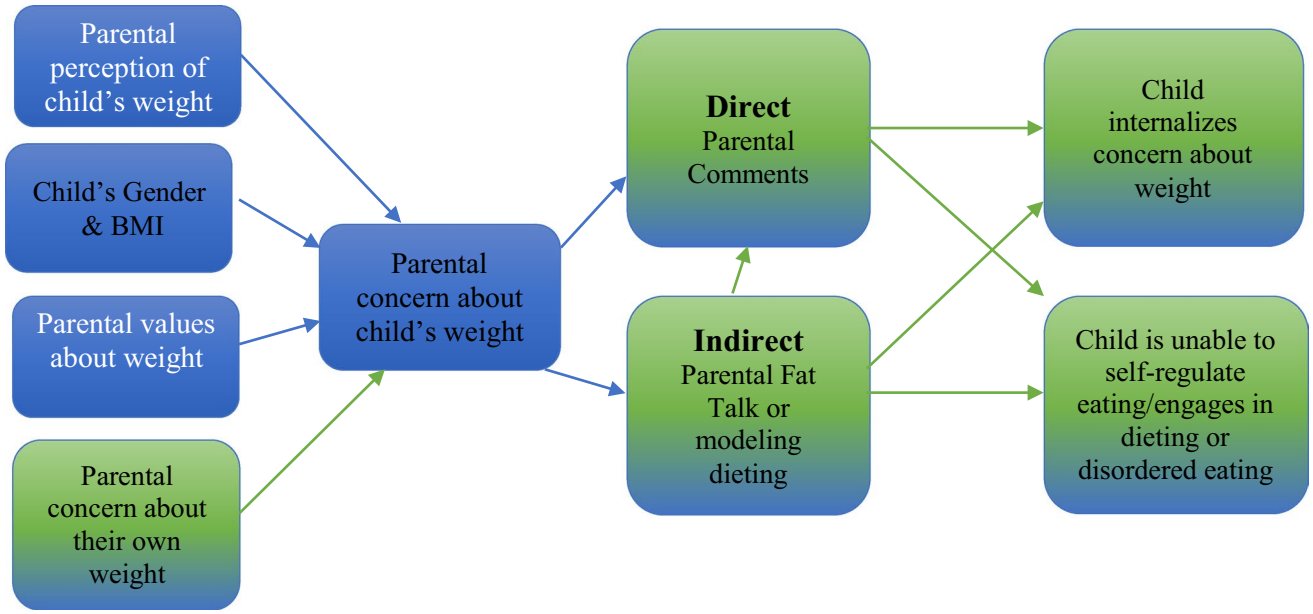

Fig. 1 Revised Obesity and Dieting Proneness Theoretical Model. *Credit to P.R. Costanzo and E.Z. Woody; permission to use this image received from P.R. Costanzo: Costanzo and Woody [14]

Negative Indirect Parental Feedback Subscale

These types of feedback centered on parental behavior directed towards the participant, a participant's sibling, or another close relative. Questions were answered for both parents on a 1 (Never) to 5 (Almost Always) Likert-type response scale. They included the questions about parental weight loss for appearance-related concerns, engagement in "fat talk", encouraging close family members to diet, and weight-related criticism of close family members.

Negative Direct Parental Feedback Subscale

These types of feedback involved direct parental behavior towards the participant in question. Questions were answered for both parents on a 1 (Never) to 5 (Almost Always) Likert-type response scale. They included questions about parental encouragement to lose weight, engagement to diet, encouraging close family members to diet, and weight-related criticism both as a youth and as an adult.

Questionnaire psychometrics

Internal consistency estimates were calculated for the "Negative Direct" and "Negative Indirect Parental Feedback Subscales" combined with a resulting value of 0.90; acceptable values range from 0.70 to 0.95 [15].

To check the quality of the subscales, principal component analyses were run for both questionnaire subscales separately: the "Negative Indirect Parental Feedback Subscale" and the "Negative Direct Parental Feedback Subscale". This was performed to determine if the subscales grouped together shared variance as hypothesized. For the "Negative Indirect Parental Feedback Subscale", one component accounted for 41.5% of the total variance, with all items loading above 0.55. Similarly, the "Negative Direct Parental Feedback Subscale" had a one-component solution which accounted for 42.3% of the total variance, with all items loading above 0.49, suggesting that the items were moderately to highly correlated with that component.

Data collection

Participants were recruited through four distinct ED treatment sites due to existing connections and collaborations with the PI: The Eating Recovery Center in Cincinnati, Ohio; The University of North Carolina Center of Excellence for Eating Disorders; The Chestnut Ridge Center in Morgantown, West Virginia; and the eating disorders partial hospitalization program (PHP) at New York-Presbyterian Hospital. We also obtained permission to advertise this survey on a provider listserv, the West Virginia Eating Disorder Network (personal communication from Dr. Jessica Luzier).

Participants

A total of 65 participants were recruited between January and March of 2017; participants were included in the analysis if they provided demographic information, completed the EPSI, and had completed at least 25% of the questionnaire (if a participant had only lived with their mother, they would have been able to complete 50% of the questionnaire due to the built-in skip pattern), reducing the total sample to 60. A power analysis conducted based on findings from previous research in a community sample required a sample of 58 participants, suggesting our sample size was adequate [5]. The majority of respondents were female ($n=56$, 93.3%), White ($n=59$; 98.4%), had at least a college degree ($n=47$, 78.3%), and were privately insured ($n=47$, 78.3%). Most were young and in the normal weight range, although there was high variance on both age (mean age 31.8 years $\pm$ 9.9) and BMI (22.0 ± 6.8). Annual family income was normally distributed, but most reported incomes were between $46,000 and $100,000 per year. Twenty-one percent ($n=13$) of the respondents were parents themselves (see Table 1).

Human subjects

This study was filed with West Virginia University's Institutional Review Board and exempt status has been acknowledged (IRB#: 1609282716). A copy of the complete survey can be found in the Appendix (Supplementary material). Signed informed consent was not required due to the anonymous nature of the project, but a cover letter explaining the study was presented prior to the survey and all participants had to select that they agreed to participate in order to continue to the survey. Qualtrics software was used to host and distribute the survey online. A list of resources and referrals for ED treatment and support groups was provided at the end of the survey for all individuals. No protected health information (PHI) was obtained. Participants were able to provide their email address for entry into drawing for a $50 Amazon gift card, but they were not required to do so; all email addresses were kept confidential and were separated from the rest of the data.

Data analysis

To answer the primary research question, bivariate analyses included a correlation matrix to describe the relationship between recall of parental feedback in this ED-specific population and ANOVAs were run to assess the subscales by different current diagnosed EDs. Multivariable linear regressions were run using stepwise regression with EPSI total scores to determine whether direct (e.g., encouragement to diet, criticism of weight) or indirect (e.g., parental dieting or fat talk) feedback corresponded with higher scores. We

Table 1 Demographic characteristics

Characteristic	Total $N=60$ N (%)	Mean ± SD $N=60$
Age		31.8 ± 9.9
Gender		
Male	4 (6.7)	
Female	56 (93.3)	
Hispanic or Latino/a		
Yes	1 (1.7)	
No	59 (98.4)	
Race/ethnicity		
White/Caucasian	59 (98.4)	
Asian	1 (1.7)	
BMI		22.0 ± 6.8
Education		
College or higher	47 (78.3)	
High school/GED	12 (20.0)	
Less than high school	1 (1.7)	
Annual family income		
< $46,000	14 (23.3)	
$46,000–$100,000	21 (35.0)	
> $100,000	18 (30.0)	
Prefer not to answer	7 (11.1)	
Parent		
Yes	13 (21.3)	
No	47 (78.7)	
Insurance type		
Privately insured	45 (75.0)	
Medicare	8 (13.3)	
Uninsured	4 (6.7)	
Medicare	3 (5.0)	
Maternal eating disorder history[a]		
Yes, professionally diagnosed	2 (3.4)	
Yes, undiagnosed	15 (25.9)	
No	32 (55.2)	
Unsure	9 (15.5)	
Paternal eating disorder history[b]		
Yes, professionally diagnosed	0 (0)	
Yes, undiagnosed	2 (3.9)	
No	46 (88.5)	
Unsure	4 (7.7)	
Past eating disorder diagnoses[c]		
Anorexia	30	
Bulimia	14	
Binge eating disorder	7	
Eating disorder not otherwise specified[d]	12	
Current eating disorder diagnoses[c]		
Anorexia	14	
Bulimia	7	
Binge eating disorder	3	
Eating disorder not otherwise specified[d]	2	

Table 1 (continued)

[a]Missing information for two participants

[b]Missing information for nine participants

[c]Since one individual can have multiple eating disorders and both present and past eating disorders, these columns will not necessarily add up to the total N and therefore, percentages are not appropriate

[d]Eating disorder not otherwise specified (EDNOS), now labeled 'Other Specified Feeding or Eating Disorder' in DSM-5; EDNOS was used in the questionnaire because more patients are still familiar with that terminology

used the total score since we were interested in total eating pathology, rather than particular subscales. We controlled for potential confounders such as parental eating disorder history to account for some genetic influences and we also controlled for body mass index (BMI), which could confound many of the subscales on the EPSI. There were not enough male patients to conduct subgroup analyses. Data analysis was conducted using SAS JMP® 13.0 and α was set equal to 0.05 for all analyses.

Results

Item responses

Twenty-seven participants had complete data for the questionnaire and 12 participants had data missing for only one item. Twenty percent ($n=12$) had 2–3 items missing while 15% ($n=9$) had four or more items missing. Single imputation was conducted for missing items when averaging questionnaire subscales. This allowed for 60 participants to be included in the analyses.

Item-by-item responses for both subscales are reported in Table 2, including number of participants endorsing the category with the valid percentage reported. Notably, stronger positive patterns of endorsement were seen for maternal than paternal items. There were especially low levels of endorsement for current items about maternal and paternal feedback.

None of the ANOVAS for the "Negative Indirect Parental Feedback Subscale" or "Negative Direct Parental Feedback Subscale" were significant when compared to current ED diagnoses. Since there were no significant associations, there was not a need to conduct a Bonferroni correction to account for a potential family-wise type I error rate.

Correlation matrix

The Pearson correlation matrix (see Table 3) showed that the "Negative Indirect Parental Feedback Subscale" was significantly positively correlated with the "Negative Direct Parental Feedback Subscale" ($r=0.58$; $p<0.0001$). The EPSI total score was significantly positively correlated with the "Negative Direct Parental Feedback Subscale" ($r=0.28$; $p=0.04$), but not with the "Negative Indirect Parental Feedback Subscale" ($r=0.06$; $p=0.68$). BMI was significantly positively correlated with both questionnaire subscales ($r=0.26$, $p=0.05$) for the "Negative Indirect Parental Feedback Subscale"; $r=0.33$, $p=0.05$ for the "Negative Direct Parental Feedback Subscale"), but not the total EPSI score.

Multivariable linear regression findings

Results of the multivariable linear regression with stepwise regression are displayed in Table 4. There was no evidence of multicollinearity and model assumptions were met. The regression was conducted in two steps, first including only demographic variables or covariates and second by including the variables of interest, the "Negative Indirect Parental Feedback Subscale" and the "Negative Direct Parental Feedback Subscale". The amount of variance explained by the final model increased 11% in comparison to the initial model (Model 1: $R^2=0.09$, Model 2: $R^2=0.20$). Findings suggest a significant relationship between the "Negative Direct Parental Feedback Subscale" scores and total EPSI scores. For every additional one point on the "Negative Direct Parental Feedback Subscale", EPSI total scores increased by an average of 14.1 points (SD = 7.0; $p=0.05$). There was no significant relationship between the "Negative Indirect Parental Feedback Subscale" scores and total EPSI scores ($\beta=0.9\pm8.6$; $p=0.92$). None of the demographic covariates, including maternal or paternal EDs, past ED diagnoses (for participants), or participant BMI, were significant predictors of total EPSI scores (p values ranging from 0.39 to 0.99).

Discussion

This study aimed to understand both direct and indirect parental feedback within a clinical sample of ED patients based upon previous research from a community sample [5]. The results of this study showed several important findings. The correlation matrix indicated that many maternal and paternal feedback items were significantly positively correlated and that many indirect and direct items were also significantly positively correlated. These results are similar to the contingency analysis findings in previous research that

Table 2 Frequency and percentage endorsing of all subscale items

Subscale	Item					
Negative Indirect Parental Feedback Subscale		Never	Almost never	Some of the time	Usually	Almost always
	Q16. Did your MOTHER engage in diets specifically to lose weight (primarily for appearance-related concerns)?	16 (28.1%)	8 (14.0%)	13 (22.8%)	12 (21.1%)	8 (14.0%)
	Q17. Did your MOTHER engage in 'fat talk,' that is degrading self-talk specifically about her weight, shape, or size?	13 (22.4%)	11 (19.0%)	17 (29.3%)	7 (12.1%)	10 (17.2%)
	Q20. Did your MOTHER ever encourage a sibling or close family member to diet to lose weight?	30 (51.7%)	11 (19.0%)	13 (22.4%)	3 (5.2%)	1 (1.7%)
	Q22. Did your MOTHER ever criticize a sibling or close family member's weight, shape, or eating behaviors?	19 (32.8%)	17 (29.3%)	13 (22.4%)	6 (10.3%)	3 (5.2%)
	Q25. Did your FATHER engage in diets specifically to lose weight (primarily for appearance-related concerns)?	26 (50.0%)	14 (26.9%)	5 (9.6%)	3 (5.8%)	4 (7.7%)
	Q26. Did your FATHER engage in 'fat talk,' that is degrading self-talk specifically about his weight, shape, or size?	28 (53.8%)	14 (26.9%)	6 (11.5%)	3 (5.8%)	1 (1.9%)
	Q29. Did your FATHER ever encourage a sibling or close family member to diet to lose weight?	30 (57.7%)	12 (23.1%)	8 (15.4%)	0 (0.0%)	2 (3.8%)
	Q31. Did your FATHER ever criticize a sibling or close family member's weight, shape, or eating behaviors?	26 (50.0%)	13 (25.0%)	9 (17.3%)	4 (7.7%)	0 (0.0%)
Negative Direct Parental Feedback Subscale		Strongly disagree	Disagree	Neither agree nor disagree	Agree	Strongly agree
	Q19. Did your MOTHER encourage you to diet to lose weight?	29 (50.0%)	13 (22.4%)	6 (10.3%)	2 (3.5%)	8 (13.8%)
	Q21. Did your MOTHER ever criticize your weight, shape, or eating behaviors?	18 (31.0%)	9 (15.5%)	22 (37.9%)	4 (2.9%)	5 (8.6%)
	Q28. Did your FATHER encourage you to diet to lose weight?	32 (61.5%)	12 (23.1%)	3 (5.8%)	2 (3.8%)	3 (5.8%)
	Q30. Did your FATHER ever criticize your weight, shape, or eating behaviors?	21 (40.4%)	15 (28.9%)	11 (21.2%)	3 (5.8%)	2 (3.8%)
	Q33. Now that you are an adult (over 18), does your MOTHER encourage you to diet?	37 (75.5%)	11 (22.4%)	0 (0.0%)	1 (2.0%)	0 (0.0%)

Table 2 (continued)

Q34. Now that you are an adult (over 18), does your MOTHER criticize your weight, shape, or eating behaviors?	16 (41.0%)	16 (41.0%)	1 (2.6%)	5 (12.8%)	1 (2.6%)
Q35. Now that you are an adult (over 18), does your FATHER encourage you to diet?	45 (85.0%)	7 (13.2%)	0 (0.0%)	1 (1.9%)	0 (0.0%)
Q36. Now that you are an adult (over 18), does your FATHER criticize your weight, shape, or eating behaviors?	30 (62.5%)	16 (33.3%)	0 (0.0%)	2 (4.2%)	0 (0.0%)

Table 3 Bivariate correlation matrix

Measure	1	2	3	4	5	6	7	8	9
1. Negative Indirect Parental Feedback Subscale	–								
2. Negative Direct Parental Feedback Subscale	0.58***	–							
3. Eating pathology symptoms inventory (EPSI) total score	0.06	0.28*	–						
4. Maternal eating disorder, diagnosed	−0.07	−0.13	−0.15	–					
5. Maternal eating disorder, undiagnosed	0.58***	0.29*	0.16	−0.12	–				
6. Maternal eating disorder, none	−0.58***	−0.42**	−0.11	−0.21	−0.66***	–			
7. Paternal eating disorder, undiagnosed	0.16	−0.03	−0.17	0.46**	0.12	−0.22	–		
8. Paternal eating disorder, none	−0.30*	−0.24	0.14	−0.23	−0.06	0.25	−0.55***	–	
9. Body mass index (BMI)	0.26*	0.33*	−0.12	−0.11	0.17	−0.21	−0.05	−0.10	–
Descriptive statistics (N=)	58	60	56	2	15	32	2	46	52
Mean	2.1	1.8	112						22.0
STD or %	0.7	0.8	30.3	3.4%	25.9%	55%	3.8%	88.5%	6.8

***$p < 0.0001$, **$p < 0.001$, *$p < 0.05$

found increased odds of participants recalling direct feedback if they recalled indirect feedback [5]. It lends additional credence to our reasoning for including indirect and direct feedback separately in our proposed Obesity and Dieting Proneness Theoretical Model, as they appear to influence each other (see Fig. 1).

Additionally, the multivariable linear regression showed that there was a significant association between "Negative Direct Parental Feedback" and the EPSI total score. This relationship was significant even after controlling for maternal and paternal eating disorder history in the model, suggesting that these measured social and environmental factors were possibly associated with eating pathology independent of genetic contributions in this clinical sample. Conversely, "Negative Indirect Parental Feedback" was not found to be significantly associated with EPSI scores in this sample. This reinforces results from a community sample where a direct example of parental feedback (recall of maternal encouragement to diet), but not indirect feedback, was associated with a higher BMI for participants as adults [5]. This may suggest that direct parental feedback is more salient for the child, and therefore more likely to have an association with eating behavior or weight. However, just because the other factors in the model were not statistically significant, it does not mean that they were not clinically significant or that they may not be significant for another population. For example, indirect parental feedback might be less memorable as an adult looking back, but may have shaped earlier eating behaviors. Additionally, indirect parental feedback might be a significant predictor for BMI among males or among minority groups, neither of which could be explored in our sample. Future research will also be needed to tease out potential differences between maternal and paternal feedback in this population.

Results from this study suggest the need for carefully planned and executed parental interventions about how direct feedback (e.g., parental encouragement to diet, parental criticism of weight or shape, etc.) regarding eating,

Table 4 Multivariable linear regression using stepwise regression with indirect and direct feedback subscale predictors of EPSI total score ($N = 49$)

Overall model	Model 1				Model 2			
	F	*df*	*p*	R^2	*F*	*df*	*p*	R^2
	0.6	7	0.77	0.09	1.1	9	0.41	0.20
Predictors of EPSI	ß ± SE		*p*			ß ± SE		*p*
Intercept	131.5 ± 21.1		<0.0001*			98.3 ± 28.2		0.0012*
Indirect feedback						0.9 ± 8.6		0.92
Direct feedback						14.1 ± 7.0		0.05*
Maternal eating disorder								
Yes, diagnosed	− 19.4 ± 24.5		0.43			− 7.4 ± 24.3		0.76
Yes, undiagnosed	− 0.7 ± 12.6		0.96			− 0.55 ± 13.4		0.97
No	− 10.5 ± 10.9		0.34			− 0.13 ± 11.6		0.99
Paternal eating disorder[a]								
Yes, undiagnosed	− 13.1 ± 26.9		0.63			− 5.1 ± 26.1		0.85
No	1.7 ± 14.8		0.91			9.4 ± 14.8		0.53
Past ED diagnosis	− 2.6 ± 4.5		0.57			− 4.1 ± 4.7		0.39
BMI	− 0.7 ± 0.6		0.26			− 1.0 ± 0.6		0.92

*$p \leq 0.05$

[a]No reported cases of diagnosed paternal eating disorders

weight, and shape is associated with potentially negative outcomes for children, including eating disorders. In support of this rationale, the American Academy of Pediatrics recently released recommendations to prevent both eating disorders and obesity among children and adolescents [16]. These recommendations included having pediatricians encourage more frequent family meals and emphasizing families not talk about weight, but rather frame discussions around health and healthy behaviors. The AAP suggested that families try to facilitate healthy eating without a focus on dieting or eating to lose weight while encouraging physical activity for both the child and larger family unit. Even if parents employ these strategies, children still might struggle with EDs; regardless, it is crucial for parents to provide positive environments in which children can foster a constructive relationship and conversation about food and body diversity.

There is also a need for this positive environment to continue so that families can be effective allies to help their loved ones in ED recovery. Often, parents and carers are unsure of how best to help and the burden of caregiving can become emotionally and physically overwhelming. Having families integrated into treatment so that they can learn about the illnesses and gain skills to aid with recovery will be a critical to helping carers and their loved ones alike [17, 18].

Limitations

This study has several limitations. First, although the questionnaire used in this study had been further refined from previous research, it still has not been validated. However, it demonstrated acceptable estimates of internal consistency reliability. Second, there is the possibility of recall bias due to the nature of the questions asking about childhood. Additionally, because this was a clinical cohort of ED patients, there could be additional recall bias due to their heightened shape and weight concerns making them more likely to recall childhood issues. The association could also be mediated by genetic vulnerabilities rather than environmental factors. Third, relying on second-hand diagnostic information about maternal and paternal ED history, rather than having direct diagnostic confirmation limits the linear regression. Fourth, the sample only had four male participants, preventing subgroup analyses between male and female participants on recall. Therefore, future research with a more equal proportion of male and female ED participants is warranted. Fifth, findings cannot be considered causal owing to the cross-sectional study design. It could be that ED participants with more weight and body image concerns now are more likely to recall parental feedback. Longitudinal studies are needed to clarify this association. Sixth, we did not gather data from the participants' parents, which could have proven useful to understand the social and environmental context better. We also did not collect diagnostic data about the parents' ED if they had a diagnosis, which might have been helpful to cross-reference with their child's diagnosis. Seventh, although this study looked at clinical samples broadly, these associations are not examined across ED status, onset, recovery status, or course. It could be that these associations are more abundant in samples of people in treatment for a specific ED or in individuals who are struggling with an active ED in comparison to an individual in recovery. Finally, some of these results could be due to a cohort effect

and more research will need to be done with participants who are children today in order to understand the type and extent of current parental feedback. Parental behavior may have changed either positively or negatively in the time since these participants were children. However, especially with the continued and increased use of social media which often includes encouraging dieting and weight loss, it is important for parents to provide positive feedback to counter negative messages that their children might be receiving from other sources. Finally, results are not considered generalizable because of the convenience sampling methodology.

Conclusion

Parents are not to blame for child's eating disorder and can be their best allies for treatment [19]. However, developing research, including the findings reported here, suggest social and environmental factors are associated with EDs and there is evidence of increased body dissatisfaction, weight problems, and eating pathology among teens and adults who recalled direct parental feedback to engage in dieting [20–25]. The finding in the current study of increased eating pathology associated with direct parental feedback in a clinical population of ED participants even when controlling for parental ED history, complements research conducted with a non-clinical sample [1] and suggests greater attention is needed within the ED literature on social and environmental factors and their potential associations with eating pathology. Nonetheless, additional research is necessary to further replicate our findings as well as to study this topic from different methodological standpoints such as an intervention or case–control.

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Compliance with ethical standards

Conflict of interest S.C. Zerwas has consulted with Coleman Research. No other authors have a conflict of interest to declare.

Statement of human and animal rights All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed consent Informed consent was obtained from all individual participants included in the study.

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