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RESEARCH ARTICLE

Adding maintaining factors to developmental models of anorexia nervosa: An empirical examination in adolescents

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Abstract

Objective: A biopsychosocial approach has been proposed to explain the pathogenesis of anorexia nervosa (AN), despite only a few of the existing etiological models having received empirical support. The aim of this study was to empirically investigate Herpertz-Dahlmann, Seitz, and Konrad (2011, https://doi.org/10.1007/s00406-011-0246-y)'s developmental model and to consider if interpersonal reactions to the illness might serve as maintaining factors following the model proposed by Treasure and Schmidt (2013, https://doi.org/10.1186/2050-2974-1-13)

Method: One hundred adolescents and their families were participated in the study: 50 diagnosed with AN, paired by age and parents' socio-economic status with 50 adolescents without a pathology. Biological, psychological and familial variables were assessed using ten questionnaires and a blood analysis test. Additionally, structural equation modeling was conducted to assess two hypothetical models.

Results: The fit of both models was good after the addition of two covariate parameters (e.g., Comparative Fit Index > 0.96 and Tucker–Lewis Index > 0.95). Premorbid traits were linked to body dissatisfaction and to the number of stressful life events; this in turn was linked to AN symptoms. Biological and familial consequences reinforced this pathology.

Conclusions: Our findings provide support for both models, suggesting that inter relationships between bio-psycho-familial variables can influence the course of AN during adolescence.

KEYWORDS
adolescence, anorexia nervosa, developmental model, etiology, maintaining factors, structural equation modeling

Abbreviations: AN, Anorexia Nervosa; Anxiety-S, Anxiety State; Anxiety-T, Anxiety Trait; BMI, Body Mass Index; CFI, Comparative Fit Index; LOI-CV, Leyton Obsessional Inventory-Child Version; RMSEA, Root Mean Square Error of Approximation; SD, Standard Deviation; SLE, Stressful life events; SRMR, Standardized Root Mean Square Residual; TLI, Tucker–Lewis Index; z-BMI, BMI standard deviation scores.

1 | INTRODUCTION

Anorexia nervosa (AN) is an eating disorder characterised by high rates of chronicity and morbidity (Zipfel et al., 2015), a poor quality of life (Winkler et al., 2014) and high rates of negative experiences of caregiving among families and close peers (Hudson & Pope, 2018; Sepulveda et al., 2014). A biopsychosocial model for the pathogenesis of AN has been proposed (Stefanini et al., 2019), with a complex etiology characterised by the interaction of multiple genetic, biological, psychological and sociocultural factors (Culbert et al., 2015; Karwautz et al., 2011; Yilmaz et al., 2015). However, most research has traditionally studied risk factors in isolation, rather than examining a multifactorial model (Krug et al., 2015). Although significant effort has been made to attain integrative biopsychosocial approaches in recent years (Fairburn et al., 2003; D. Garner, 1993; Gorwood et al., 2016; Herpertz-Dahlmann et al., 2011; Lyon et al., 1997; Munro et al., 2017; Toro, 2004; Treasure & Schmidt, 2013), few models have considered a complete biopsychosocial approach that integrates a developmental consideration of AN.

Herpertz-Dahlmann et al. (2011), elaborating on research by Kaye et al. (2009), proposed a developmental model that integrates both the time course and complex phenomenology of AN. This model postulates that typical AN temperament and personality traits, such as perfectionism, harm avoidance, anxiety or obsessive traits, which might represent underlying neurobiological factors (Kaye et al., 2013), become intensified during adolescence due to multiple factors, such as changes in hormones and stressful life events including body shape or weight issues. Adolescents with a genetic predisposition to AN may use excessive dieting and/or restless activity to cope with a dysphoric mood or feelings of worthlessness. Extreme calorie restrictions maintained over time in these adolescents leads to secondary starvation effects including pathophysiological adaptations to conserve energy and also exacerbate premorbid traits, such as anxiety, depression and obsessiveness. These traits can increase weight loss behaviours, leading to a vicious cycle that can perpetuate the disorder.

The Herpertz-Dahlmann et al. (2011) model does not include social or family factors, which may come as a surprise as AN may also generate consequences in social and family life, which in turn can influence the etiology and the maintenance of the disorder at the same time (Medina-Pradas et al., 2011; Rienecke et al., 2017). However, the interpersonal maintenance model of AN (Treasure & Schmidt, 2013) proposes that families who usually share certain traits with their offspring react to AN symptoms with high levels of anxiety and frustration, which can lead to negative behavioural responses such as expressed emotion, which can exacerbate the symptoms and perpetuate the disorder (Goddard et al., 2011; Schmidt & Treasure, 2006; Sepulveda et al., 2008). Incorporating maternal and parental variables in etiological models may be a necessary step for understanding AN (Krug et al., 2015). The model proposed by Herpertz-Dahlmann et al. (2011) has not yet received empirical support, despite a call for research to focus on the empirical validation of existing theories (Pennesi & Wade, 2016).

The present study aimed to empirically test a bio-psycho-familial model for AN based on the developmental model proposed by Herpertz-Dahlmann et al. (2011) and the interpersonal extension of the model based on Treasure and Schmidt (2013). Based on the first model (Herpertz-Dahlmann et al., 2011), we hypothesise that premorbid traits (perfectionism, obsessiveness, anxiety and harm avoidance) would be associated with body dissatisfaction and stressful life events (SLE) during pubertal stage (measured by estradiol hormones) that would be associated with eating psychopathology, and secondary effects on BMI, and biological (leptin levels) and psychological (depression, anxiety and obsessiveness) consequences that might maintain the disorder (Figure 1a). Regarding the model proposed by Treasure and Schmidt (2013), we hypothesise that eating symptomatology also leads to family consequences, as parents' anxiety would increase and be associated with an

Highlights

- These findings in part provide empirical support for the developmental model proposed by Herpertz-Dahlmann et al. (2011) and the interpersonal maintenance model (Treasure & Schmidt, 2013) for AN.
- Premorbid traits are linked to AN symptoms through body dissatisfaction and stressful life events, and the biological, psychological and familial consequences that follow may act as maintaining mechanisms.
- This model suggests a temporal sequence of symptoms that might explain the course of AN and its maintenance. This has implications for early intervention as interventions into maintenance factors in the early stages of the illness can moderate the course of the disorder.

increase in emotional over-involvement, which could affect the maintenance of the disorder (Figure 1a). We have only considered emotional over-involvement in the model, as it is related to the first stage of the illness in adolescents (Blondin et al., 2019; Schwarte et al., 2017) and is associated with accommodating behaviour (an important maintaining factor) whereas criticism and hostility, the other domains of expressed emotion, emerge later in the course of the illness (Duclos et al., 2014; Koutra et al., 2014).

2 | METHODS

2.1 | Participants and procedure

The ANOBAS study (PSI2012-23127) is a cross-sectional case-control study designed to assess early risk factors for eating disorders (ED), in a consecutive recruitment of a patient sample of 12–17 years, at the onset of their illness. Details of the recruitment and sample selection procedure have been previously published (Sepúlveda et al., 2021). The total sample consisted of one hundred Caucasian participants and their families. Half the sample (50) consisted of female adolescents diagnosed with AN and their families who were recruited from the inpatient or outpatient services of an adolescent ED unit at the onset of the illness. These participants presented the following diagnoses: AN restrictive subtype ($n = 35$; 70%), AN purgative subtype ($n = 8$; 16%) and not otherwise specified feeding and eating disorders, restrictive type ($n = 7$; 14%). The mean age of the AN sample was 14.68 years (SD: 1.39), with a mean BMI of 16.06 kg/m^2 (SD = 1.74). The 50 control adolescents without pathology and their families were recruited from public secondary schools in the region. Exclusion criteria for the control group were the presence of metabolic disorders that could affect the weight status, a BMI of less than 17.5 kg/m^2 or over 30 kg/m^2, and no history of a psychiatric disorder. Three participants in the control group were excluded after the assessment because of the presence of an ED. The mean age of the control sample was 14.66 (SD: 1.32), with a mean BMI of 21.18 kg/m^2 (SD: 2.66).

The study received ethical approval by the Hospital Ethics Committee (Ref. Code, R-0009/10) and the corresponding University Research Ethics Committee (UAM, CEI 25-673).

2.2 | Measures

Current and lifetime psychiatric diagnoses were evaluated with the *Schedule for Affective Disorders and Schizophrenia for School-Age Children* (K-SADS-PL; Kaufman et al., 1997). This is a semi-structured diagnostic interview designed to assess current and lifetime psychopathology in all of the adolescents.

Participants completed a battery with eight self-report questionnaires, and their fathers and mothers completed two questionnaires. All of the questionnaires have shown adequate psychometric validity with Spanish samples (in the present sample, internal consistency using Cronbach α values ranged between 0.75 and 0.98). Biological variables were also assessed through a physical examination and laboratory analyses of blood markers.

Regarding *Premorbid traits*, perfectionism was assessed with the self-oriented perfectionism subscale of the Child Adolescent Perfectionism Scale (CAPS; Flett et al., 2000). Obsessiveness was evaluated with the Leyton Obsessional Inventory-Child Version (LOI-CV; Berg et al., 1986) and anxiety with the anxiety-trait subscale of the State-Trait Anxiety Inventory for Children (STAIC; Spielberger et al., 1973). Harm avoidance was evaluated using a subscale of the Junior Temperament and Character Inventory (JTCI; Luby et al., 1999).

To evaluate the changes occurring during puberty, we used the Body Shape Questionnaire (BSQ; Cooper et al., 1987) to assess body dissatisfaction as a result of social pressure (Thompson et al., 1999). We also considered the number of severe SLE that had occurred during the year prior to onset, evaluated with the Children's Life Events Inventory (Mardomingo & Gonzalez, 1998). The year prior to the onset was calculated considering the moment when they were diagnosed. The rationale for considering this period was based on the fact that previous studies have reported an increase in the number of SLEs in the year prior to the onset of eating behaviours compared with other control groups (Hay & Williams, 2013; Pike et al., 2008). In addition, we also evaluated the levels of estradiol, a major hormone puberty indicator in females (Baker et al., 2012), with a blood analysis.

The model differentiates between the AN symptomatology in a first stage of *eating psychopathology* and a second stage of weight loss and starvation. To assess the first stage, we used the Eating Attitudes Test (EAT-26; Garner et al., 1982), which provides a suitable screening of ED. For the second stage, we used BMI standard deviation scores (BMI *z*-scores). These were calculated by comparing the adolescent's BMI with the ideal BMI of the Spanish population of the same age and sex (Sobradillo et al., 2004).

Eating symptomatology has consequences on three levels. For the biological consequences, we assessed the base-10 logarithm of leptin concentration as a change induced by malnutrition (Monteleone & Maj, 2013). For

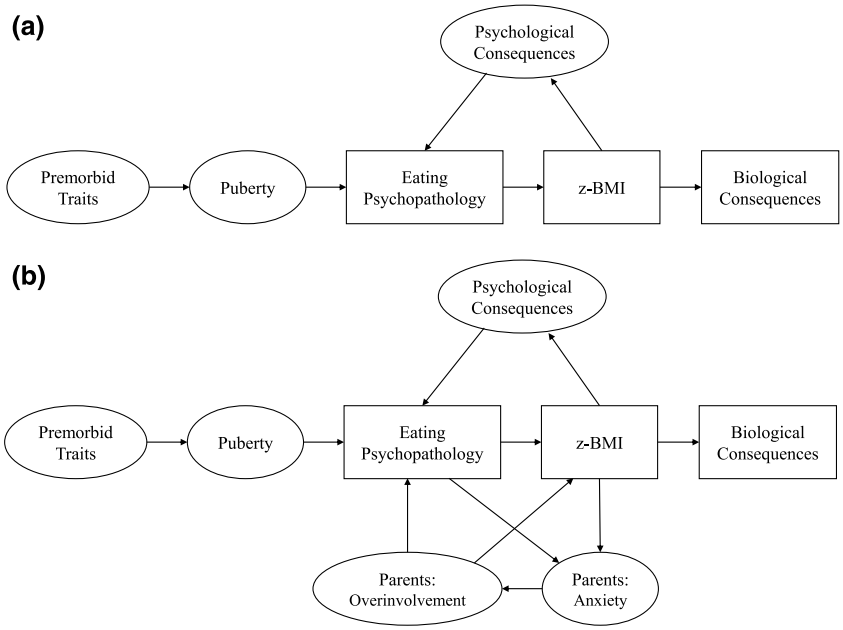

FIGURE 1 Hypothetical models. Note. The biopsychofamilial model proposed in the present study complements the developmental model (Herpertz-Dahlmann et al., 2011) adding familial variables in order to explain the familial contribution to the disorder (Treasure & Schmidt, 2013)

the psychological consequences, we evaluated depression with the Child Depression Inventory (CDI; Kovacs, 1992), anxiety with the anxiety-state subscale of the STAIC (Spielberger et al., 1973) and obsessiveness with the LOI-CV (Berg et al., 1986). For the familial consequences, we assessed the anxiety of the parents with the State-Trait Anxiety Inventory (STAI, Spielberger et al., 1970) and emotional over-involvement with the Family Questionnaire (FQ) subscale (Wiedemann et al., 2002).

2.3 | Data analysis

Variables were explored using descriptive analysis (mean and standard deviation) and Pearson correlation coefficients. The two hypothetical models proposed in the present study were tested using structural equation modeling with lavaan package (Rosseel, 2012) in R software. Specifically, robust maximum-likelihood (MLR) was used as the estimator to examine the models as several indicators did not follow a normal distribution (Tabachnick & Fidell, 2007). Moreover, full information maximum likelihood (FIML) was used as the missing imputation procedure (Enders & Bandalos, 2001) to avoid participant loss. All variables were standardised before testing the models. Model fit was evaluated as positive using the following criteria: Comparative Fit Index (CFI) and Tucker–Lewis Index (TLI) greater than 0.95, Root Mean Square Error of Approximation (RMSEA) less than or equal to 0.06, and Standardised Root Mean Square Residual (SRMR) less than or equal to 0.08 (Byrne, 2011). Modification indices were examined in order to improve the model fit of the hypothetical models.

3 | RESULTS

Table 1 presents the descriptive analysis (mean and standard deviation) for all study variables as well as Pearson correlation coefficients between variables. In general, relationship between the variables were as expected.

The developmental model (Herpertz-Dahlmann et al., 2011) proposed in Figure 1a did not present a good fit to the data: $\chi^2(50) = 148.14$, $p < 0.001$, CFI = 0.84, TLI = 0.79, RMSEA = 0.144 [0.118–0.172], SRMR = 0.105. Thus, modification indices were examined, and two free parameters of covariation were progressively included in the model based on χ^2 differences. First, a free parameter of covariation between Premorbid Traits and Psychological Consequences was included as the Obsessiveness (LOI-CV) is an indicator of both factors. Second, a free parameter of covariation between leptin and estradiol was included as they were the only biological variables included in the model. This model obtained a good fit to the data: $\chi^2(48) = 69.87$, $p = 0.02$, CFI = 0.97, TLI = 0.95, RMSEA = 0.067 [0.026–0.101], SRMR = 0.060. Similarly, the biopsychofamilial model proposed in Figure 1b did not present a good fit to the data: $\chi^2(95) = 207.08$, $p < 0.001$, CFI = 0.86, TLI = 0.83, RMSEA = 0.112 [0.091–0.133], SRMR = 0.098. Thus, the same two free parameters of covariation were progressively included in the model, following the modification indices. This model obtained a good fit to the data: $\chi^2(93) = 127.27$, $p < 0.001$, CFI = 0.96, TLI = 0.95, RMSEA = 0.061 [0.030–0.086], SRMR = 0.063. The estimated parameters of both models were subsequently explored. Table 2 presents the factor loadings of the measurement models.

TABLE 1 Means, standard deviations and Pearson correlation coefficients of observed variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Anxiety-T	-	0.53**	0.29**	0.76**	0.73**	−0.21*	0.27**	0.69**	−0.25*	−0.24*	0.83**	0.77**	0.32**	0.25*	0.33**	0.17
2. Perfectionism		-	0.20[a]	0.54**	0.42**	−0.07	0.13	0.38**	−0.22*	−0.24*	0.38**	0.39**	0.36**	0.23*	0.38**	0.14
3. Harm avoidance			-	0.37**	0.10	0.06	0.08	0.09	0.07	0.12	0.22*	0.17[a]	0.02	0.16	0.10	0.22[a]
4. Obsessiveness				-	0.53**	−0.13	0.29**	0.44**	−0.13	−0.11	0.60**	0.50**	0.35**	0.23*	0.25*	0.00
5. Body dissatisfaction					-	−0.10	0.20[a]	0.82**	−0.18[a]	−0.13	0.70**	0.63**	0.31**	0.26**	0.34**	0.11
6. Estradiol						-	−0.12	−0.27**	0.46**	0.45**	−0.23*	−0.16	−0.31**	−0.30**	−0.36**	−0.12
7. SLE							-	0.24*	−0.13	−0.14	0.27**	0.16	0.13	0.19[a]	0.09	0.13
8. Eating psychopathology								-	−0.42**	−0.40**	0.74**	0.65**	0.33**	0.38**	0.44**	0.13
9. z-BMI									-	0.78**	−0.27**	−0.31**	−0.39**	−0.38**	−0.60**	−0.56**
10. Leptin										-	−0.25*	−0.25*	−0.44**	−0.43**	−0.53**	−0.60**
11. Depression											-	0.75**	0.35**	0.32**	0.37**	0.24*
12. Anxiety-S												-	0.21[a]	0.25*	0.37**	0.16
13. Father: Anxiety-S													-	0.60**	0.56**	0.32**
14. Mother: Anxiety-S														-	0.46**	0.19[a]
15. Father: Overinvolvement															-	0.46**
16. Mother: Overinvolvement																-
Mean	35.36	36.00	6.93	8.24	78.22	51.46	1.90	15.38	−0.76	7.89	11.37	30.84	17.50	21.47	22.94	19.01
Standard deviation	9.56	9.34	4.30	4.32	40.33	60.27	1.85	17.04	1.30	1.18	7.91	9.78	8.91	9.92	5.56	4.22

Note: $N = 100$. Variables from shaded columns (#13–16) were only included in the biopsychofamilial model. Variables were standardised to test the models, but this table presents means and standard deviations of non-standardised variables.

[a]$p < 0.10$, *$p < 0.05$, **$p < 0.01$.

Figure 2 presents the estimated parameters of the structural model, that is, the relations between the latent and observed variables included in the model. Estimations were congruent with the predictions of the present study, with the exception of Eating Psychopathology–Parents: Anxiety and z-BMI–Psychological Consequences relations that were non-statistically significant. Statistically significant correlations between these variables (z-BMI with depression, anxiety and obsessiveness as well as Eating Psychopathology and Parents anxiety) were found, as can be seen in Table 1, but their relation was less relevant in the estimated model.

4 | DISCUSSION

The etiological model proposed by Herpertz-Dahlmann et al. (2011) describes the development of AN between the prenatal period and adolescence. Our results provide some support for the model although there are aspects that were not empirically confirmed, such as the role of psychological and biological consequences associated with starvation and their interaction. The findings also support the inclusion of familial variables in a bio-psycho-familial model of AN, as proposed by Treasure and Schmidt (2013). To the best of our knowledge, this is the first study that has empirically examined a model that integrates biological, psychological and familial variables to explore the etiology of AN amongst adolescent.

Although the majority of our original hypotheses were supported, there was one exception, which was that there was no significant link between z-BMI and psychological consequences. Thus, in our model psychological symptoms can influence eating psychopathology independently of z-BMI status. This is consistent with previous studies suggesting that depression (Ivanova et al., 2015), obsessionality and anxiety (Treasure et al., 2015) may maintain dieting. Similarly, Herpertz-Dahlmann et al. (2015) found that they could also be key factors in predicting the persistence of ED. However, these results contrast with previous experimental studies that have found psychological consequences associated with starvation (Eckert et al., 2018; Keys et al., 1950; Watkins & Serpell, 2016). Even with extreme BMIs, a direct relationship was found between higher BMI and psychological consequences (Sagar & Gupta, 2018), with rates of depression and anxiety (Puder & Munsch, 2010) higher in obesity samples than in non-obesity samples. In this line, it is worth to mention that we found statistically significant correlations between those variables, but they were not related in the estimated structural equation model. Therefore, it is unclear whether other factors, such as biological consequences, may be mediating the relationship between BMI and psychological consequences or if psychological symptoms are actually premorbid traits that predict the eating psychopathology. In this sense, there is preliminary evidence of the possible implication of lower levels of gonadal hormones, oxytocin and leptin, and higher cortisol and PYY in the anxiety and depression symptoms of AN (Miller, 2011; Misra & Klibanski, 2014).

Our results partially support the direct pathway between AN symptoms and emotional over-involvement through parental anxiety. Eating psychopathology does not appear to influence parents' anxiety, although z-BMI does elicit anxiety in the parents, which leads to an increase in emotional over-involvement, which in turn increases the exacerbation of the symptoms resulting in an increase of eating psychopathology and a decrease in z-BMI status. The short duration of the illness of our sample (less than one year) may explain the parents' reaction to z-BMI alone. A longer duration of AN is usually associated with higher levels of parents' expressed emotions (Rienecke et al., 2017; Treasure et al., 2015). Future research with adults should strive to clarify whether parents' emotional experiences are related to the stage of the illness (Anastasiadou et al., 2014) and if a longer duration explains the relationship between eating psychopathology and parents' emotional reactions.

This study presents several limitations. First, the cross-sectional design of the study does not permit causal assumptions and further research with a longitudinal design would be needed to study the temporal relation between the factors and the symptoms. Second, our sample was composed of adolescents, limiting the generalisability of these results. Future research should test this model in adults to analyze its potential structural and measurement differences and to empirically support the AN developmental transition to adulthood proposed by Herpertz-Dahlmann et al. (2011). Third, we did not consider genetic variables in the model, although adolescence is the period when genetic predisposition to AN is activated (Kaye et al., 2009). Fourth, the majority of the variables were assessed via self-report questionnaires. Lastly, the study was underpowered considering the number of variables examined. Thus, future research requires the use of larger sample sizes to achieve a complete definition of etiological variables avoiding computational burden limitations with appropriate statistical power.

5 | CONCLUSIONS

In conclusion, we attained evidence in favor of the developmental model proposed by Herpertz-Dahlmann

TABLE 2 Standardised factor loadings for the measurement models of latent variables

Latent variables	Indicator variables	Factor loadings	
		Developmental model	Biopsychofamilial model
Premorbid traits	Anxiety-T	0.99	0.98
	Perfectionism	0.54	0.54
	Harm avoidance	0.31	0.31
	Obsessiveness	1.29[a]	1.29[a]
Puberty	SLE	0.26	0.26
	Body dissatisfaction	0.87	0.87
	Estradiol	–0.10[b]	–0.10[b]
Psychological consequences	Anxiety-S	0.84	0.84
	Depression	0.91	0.91
	Obsessiveness	–0.57	–0.57
Parents: Anxiety	Father: Anxiety-S	-	0.77
	Mother: Anxiety-S	-	0.78
Parents: Emotional over-involvement	Father: E. Over-involvement	-	0.77
	Mother: E. Over-involvement	-	0.86

Note: Factor loadings of the measurement model were estimated conjointly with the structural model of Figure 2a for the developmental model (Herpertz-Dahlmann et al., 2011) and the structural model of Figure 2b for the bio-psychofamilial model, respectively. All factor loadings were statistically significant at $p < 0.05$, except estradiol.

[a]The magnitude of factor loadings can legitimately be larger than 1 in the standardised solution (Jöreskog, 1999; see also; Deegan, 1978).

[b]Non-statistically significant factor loading.

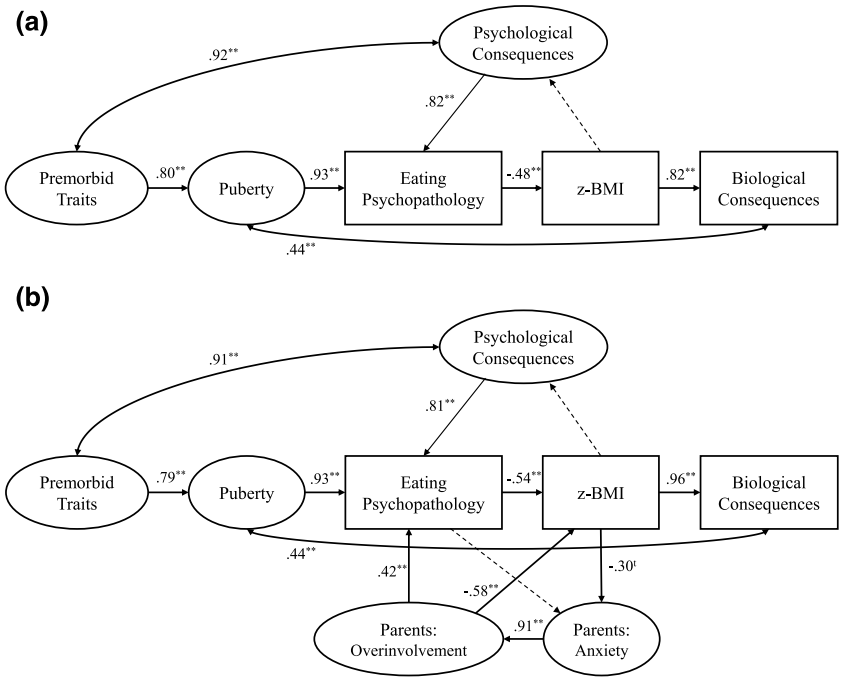

FIGURE 2 Standardised regression weights for the structural models. $t = p < 0.10$. ** = $p < 0.01$. Discontinuous lines mean non-statistically significant regression weight. Puberty-Biological Consequences free correlation coefficient only relate the estradiol indicator variable with Biological Consequences (leptin)

et al. (2011), alongside the contribution of the interpersonal component of the maintenance model of AN proposed by Treasure and Schmidt (2013). Research into maintenance processes (Lampard et al., 2013) is of interest as it may be possible to target these mechanisms (Dakanalis et al., 2017). This has implications for early intervention, as recent evidence suggests that interventions into maintenance factors in the early stages of

the illness can moderate the course of the disorder (Anastasiadou et al., 2016; Pennesi & Wade, 2016).

In addition, this model suggests that a temporal sequence of symptoms might explain the chronic course of AN and its maintenance (Gicquel, 2013; Stice et al., 2012). The trajectory of traits and symptoms identified in the model that precede the onset of AN could lead to improved intervention strategies (McClelland et al., 2020). Furthermore, the high personal standards that characterise adolescents with AN, in addition to the autonomy or independence required to manage the transition into adulthood (Kaye et al., 2009), might explain the difficulties found in AN to cope with the environmental triggers of adolescence, as they usually require a considerable degree of flexibility. In this sense, AN premorbid traits, such as perfectionism, harm avoidance or obsessive traits have been associated with cognitive rigidity (Búhren et al., 2012). As adolescent brains tend to be more flexible, addressing cognitive intervention in this period could be useful to improve cognitive functioning and to prevent relapses and comorbidity (Lock, 2010). Different interventions have been proven to improve these cognitive difficulties. Remediation cognitive therapy (Leppanen et al., 2018; Tchanturia et al., 2017) demonstrated to be an effective intervention targeting executive functioning and central coherence. Also radically open dialectical behaviour Therapy targets these temperament traits and the cognitive style (Hempel et al., 2018). On the other hand, the Neurobiologically Informed Treatment (Knatz et al., 2015) aims to reduce the symptoms focusing on the specific AN neurobiological mechanism through the development of constructive coping strategies in AN to manage their own temperament traits (Wierenga et al., 2018). At the same time, the negative consequences of the disorder can be reduced by skills training programs for family members (Treasure et al., 2020). Increasing carers coping skills can reduce their burden and distress and their response to the illness (Hibbs et al., 2015). The New Maudsley Model of collaborative care reduces family' expressed emotion and accommodation as well as improves patient outcomes (Treasure & Nazar, 2016). Future research should investigate if these interventions could indeed deter the progression of AN symptoms and their maintenance proposed in this model.

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CONFLICT OF INTEREST

The authors do not have any conflict of interest to declare.

DATA AVAILABILITY STATEMENT

There are no data available.

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