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ORIGINAL ARTICLE

Anorexia nervosa versus bulimia nervosa: differences based on retrospective correlates in a case–control study

Bárbara C. Machado[1,2] · Sónia F. Gonçalves[2] · Carla Martins[2] · Isabel Brandão[3] · António Roma-Torres[3] · Hans W. Hoek[4,5,6] · Paulo P. Machado[2]

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Abstract
Objective This study is the result of two Portuguese case–control studies that examined the replication of retrospective correlates and preceding life events in anorexia nervosa (AN) and bulimia nervosa (BN) development. This study aims to identify retrospective correlates that distinguish AN and BN
Method A case–control design was used to compare a group of women who met Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria for AN ($N = 98$) and BN ($N = 79$) with healthy controls ($N = 86$) and with other psychiatric disorders ($N = 68$). Each control group was matched with AN patients regarding age and parental social categories. Risk factors were assessed by interviewing each person with the Oxford Risk Factor Interview.
Results Compared to AN, women with BN reported significantly higher rates of paternal high expectations, excessive family importance placed on fitness/keeping in shape, and negative consequences due to adolescent overweight and adolescent objective overweight.
Discussion Overweight during adolescence emerged as the most relevant retrospective correlate in the distinction between BN and AN participants. Family expectations and the importance placed on keeping in shape were also significant retrospective correlates in the BN group.

Keywords Anorexia nervosa · Bulimia nervosa · Risk factors · Adolescent overweight

✉ Bárbara C. Machado
bcmachado@porto.ucp.pt

✉ Paulo P. Machado
pmachado@psi.uminho.pt

1 Faculty of Education and Psychology, Catholic University of Portugal, Rua Diogo Botelho, 1327, 4169-005 Porto, Portugal

2 Psychotherapy and Psychopathology Research Unit–CIPsi, School of Psychology, University of Minho, Campus de Gualtar, P-4710 Braga, Portugal

3 Psychiatry Department, Faculty of Medicine, Hospital of S. João, Braga, Portugal

4 Parnassia Bavo Academy, Parnassia Psychiatric Institute, The Hague, The Netherlands

5 Department of Epidemiology, Columbia University, New York, NY, USA

6 Department of Psychiatry, University of Groningen, Groningen, The Netherlands

Introduction

Eating disorders (ED) are among the 10 leading causes of disability in young women [1, 2]. Anorexia nervosa (AN) and bulimia nervosa (BN) have been consensually described as severe psychiatric disorders that primarily affect adolescents or young women [3]. Both typically start in the middle of adolescence because of food restriction onset [4, 5]. AN and BN are characterized by an over-evaluation of weight and body shape and a conviction about the power in their control [6–8]. In approximately 10–20 %, AN is intractable and continuous [8–13]; mortality rate has been estimated to be between 5 and 5.6 % per decade with AN associated with a 50 times higher suicide risk [4, 14]. BN is usually described as having a chronic course with remission over time ranging from 31 to 74 % [5].

ED have been conceptualized as heterogeneous disorders with a multifactorial etiology involving a complex interaction between genes and environment [2, 15, 16].

According to Schmidt [17], the main types of studies used in the investigation of risk factors are cross-sectional studies with case–control designs and prospective longitudinal studies with cohorts of subjects. Numerous factors have been identified in the development of ED. However, the results obtained from several studies are difficult to understand. Several of the primary critics advocate that (a) most of the researches have investigated a limited number of potential risk factors; (b) the methods used in these studies did not allow for the prediction of ED onset, and did not control initial symptoms or risk factors precedence; and (c) there are few studies that address control groups with other psychiatric disorders and control groups with other ED [18–20].

Studies that assessed risk factors for AN and BN using the Oxford Risk Factors Interview (RFI) addressed several of the limitations cited in the previous research using an interview to establish a diagnosis and the precedence of the risk factor evaluated and by considering a wide array of potential risk factors. Regarding AN risk factors, Fairburn's [18] study found perfectionism and negative self-evaluation were specific retrospective correlates for AN development. Temperamental traits, sexual abuse and parental pressure increased the risk for developing AN in Karwautz's [21] study. Pike's [22] study showed that women with AN had significantly higher rates of negative affectivity, perfectionism, family discord and higher parental demands. Karwautz et al. [23] found that disruptive events, interpersonal problems and dieting environment increased the risk for AN independent of genotype. Finally, Machado et al. [24] showed that women with AN reported significantly higher rates of perfectionism, negative attitudes toward parents' shape and weight, significant concern regarding feeling fat and family history of AN or BN. In assessing BN risk factors with the Oxford RFI, Fairburn et al. [19] found that exposure to factors that were likely to increase the risk for dieting and negative self-evaluation and certain parental problems (such as alcoholism and obesity) were substantially more common among those with BN. Day et al. [25] investigated risk factors, correlations and markers associated with early-onset BN and showed that adolescents with early-onset BN were more likely to report an earlier age of menarche. Recently, Gonçalves et al. [26] found that childhood overweight was the most significant BN retrospective correlates.

To our knowledge, only two studies used RFI and compared AN and BN risk factors. Fairburn et al. [18] using psychiatric and non-psychiatric control groups, found that parental obesity was the only retrospective correlate that distinguished both ED, with BN participants having higher rates of exposure; childhood obesity, which was also higher in BN participants, had marginally significant results. Hilbert et al. [27] studied the risk factors across ED comparing with non-psychiatric controls and concluded that all retrospective correlates for BN were shared by individuals with either AN or Binge Eating Disorder (BED). Considering that only two studies explored retrospective correlates that differentiate AN and BN, replication with different samples will shed light on the stability and power of such correlates. Therefore, the present study aims to contribute to the literature by expanding our knowledge about the differences between AN and BN etiology targeting to transfer to practice the input of scientific knowledge.

In previous studies we used RFI and compared AN and two control groups [24] and BN and two control groups [26]. In the present study, we aim at contributing to the understanding of the differences between AN and BN etiology. We used a case–control design with two control groups (healthy controls and controls with other psychiatric disorders) and, we compared AN participants with BN participants around a primary objective: identify retrospective correlates that distinguish AN and BN. We included participants of the previous studies and added new ones.

Method

Recruitment procedure

Participants who met Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV [28]) for AN or BN were recruited from specialized ED treatment settings. Psychiatric control (PC) group participants were also recruited from treatment settings. Potential non-psychiatric control (normal control; NC) group participants were recruited from schools and a university campus (healthy control group).

Exclusion criteria for all four groups were physical disorders likely to influence eating habits or weight, psychosis or current pregnancy. Inclusion criteria for the NC group were absence of past or current clinically significant ED or other psychiatric disorder. Inclusion criteria for the PC group included a current DSM-IV Axis I diagnosis and no previous or present history of ED symptoms.

Participants

Participants in this study were 98 women with a DSM-IV [28] diagnosis of AN ($n = 63$ restricting type and $n = 35$ binge eating/purging type); 79 women with diagnosis of BN ($n = 72$ binge eating/purging type and $n = 7$ non purging type); 68 women with current Axis I DSM-IV psychiatric diagnoses other than ED (PC group); and 86 women with no psychiatric disorder diagnosis (NC group). Of these, 86 women with AN, 60 with BN and 154

participants from both control groups were already evaluated in two studies described elsewhere [24, 26] using a conditional logistic regression analysis appropriate for a case–control design with individual matching.

PC group participants had the following primary DSM-IV diagnosis: anxiety disorder ($n = 35$; 51.4 %) and depressive disorder ($n = 32$; 47.1 %); one PC group member had a current diagnosis of somatoform disorder.

The NC and PC participants were individually matched to the participants with AN based on current age (±1 year) and parental socioeconomic status (within two parental socioeconomic status categories) and were assigned an index age corresponding to the index age of AN to which they were matched. Both control groups were questioned about their life until the age of onset of disturbed eating (index age) of their particular matched subject with AN. Index age was conservatively defined as the age at onset of at least one of the following symptomatic behaviors [18, 19, 22]: sustained dieting, sustained overeating, sustained purging (as determined by the Oxford RFI), rather than the age at which the participants first met all the criteria for an ED diagnosis. The assessment of risk factors focused on the period prior to the index age, thereby ensuring that the risk factor preceded the onset of clinically significant eating pathology [22]. Adjusting case–control comparisons for age at onset (i.e., index age) minimized differences in the time the participants were exposed to the risk factor [18].

Assessment

Diagnostic assessment

Current and lifetime psychiatric disorders were assessed with the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-IV [29]). ED diagnosis and psychopathology were assessed with the diagnostic items of the Eating Disorder Examination (EDE [30]). The Eating Disorder Examination Questionnaire (EDE-Q [31]) was used as the primary instrument to screen potentially healthy controls.

Risk factor assessment

Exposure to putative risk factors for ED was assessed with the Oxford Risk Factor Interview for Eating Disorders (RFI [19]). Each woman in the four groups was interviewed (there were no additional informants). The interviews focused on the period before the onset of the ED (retrospective reporting), with age of onset being defined as the age at which the first significant and persistent eating pathology behaviors began [19]. For risk factors believed to have a hereditary component (e.g., family history of psychiatric disorders and parental overweight and obesity) the interview focused on both the pre and post disorder onset period. The RFI was investigator-based and used behavioral definitions of key concepts to minimize problems related to retrospective data [18]. Many putative risk factors were assessed (Tables 2, 3). They were categorized into one of three domains: personal vulnerability domain, environmental domain and dieting vulnerability domain. Within each domain, we organized risk factors into several subdomains to reflect certain types of exposure. Additional risk factors were also evaluated (e.g., menarche age). The degree of exposure to a potential risk factor was rated on a five-point rating scale ranging from 0 = no exposure to 4 = high severity, long duration, or high frequency of exposure. A score of 3 or 4 was considered to indicate significant severity, duration, or frequency of exposure.

Socioeconomic status

An adaptation of the Graffar schedule [32] was used in which scores ranged from 5 to 25, with higher scores indicating lower socioeconomic level. This schedule considers the years of formal education and profession of the parents, sources of income, and type of housing and neighborhood to assign the family to one of 5 socioeconomic status categories.

Procedure

Participants in the AN and BN groups had been previously diagnosed by clinicians and were then interviewed using the EDE diagnostic items [19]. The PC group participants had a previous diagnosis by a clinician; however, true case status was established and confirmed using the Structured Clinical Interview for DSM-IV (SCID-I [29]). Participants in the NC group were screened using the EDE-Q [31]. They were selected using the following criteria: (a) score < 4 on all the 4 EDE-Q subscales and (b) absence of dysfunctional eating behaviors (i.e., binge eating episodes and inappropriate weight control methods). They were also interviewed with the SCID-I [29] to rule out any DSM-IV diagnosis. Participants of both control groups were interviewed with EDE diagnostic items [19] to rule out ED pathology.

All participants of the study were interviewed using the Oxford RFI [19], and all of the interviews were performed face-to-face and were conducted by clinical psychologists trained in the use of the standardized interview procedure of the EDE, SCID-I and RFI. Risk factor interviews were conducted by an assessor who was aware of the case status of the participant. To address this limitation and minimize the risk of biased assessment, interviewer bias was discussed during training and supervision, as suggested by Fairburn et al. [18].

Data analysis

Comparisons between the AN, control (PC and NC) and BN groups were performed using a logistic regression analysis (Statistical Package for the Social Sciences/SPSS version 15.0). First, we analyzed some relevant statistical assumptions previous to the regression analysis: (1) we studied the variability of each risk factor in the three participants groups excluding the risk factors that did not show variability between the groups; (2) we assessed the relative significance of different types of exposure in each subdomain and domain—individual putative risk factors and case status were first assessed by univariate analysis with each risk factor being considered as a single indicator variable and coded 0 for absence and 1 for presence (we only considered risk factors for the regression analysis if they showed statistically significant values between the groups at $p < .05$); (3) then, and despite having $p < .05$ results, we studied the cases in which cells presented a percentage higher than 20 % if the minimum expected was less than 5; (4) the multicollinearity assumption was also investigated, excluding all the predictors that showed results that were highly correlated (values $\geq .10$ or VIF < 4); (5) we then organized all the domains and subdomains considering the maximum number of predictors by participants according to Stevens's (1946) guidelines; (6) finally, we explored potential outliers that would need to be excluded from the final analysis (Zresidual outside the range $-3/+3$ or Cook's <1).

To predict case status (i.e., comparisons between AN, control (PC and NC) and BN groups), we used the logistic regression analysis. Because of the number of comparisons performed, statistical significance for the risk factor subdomain and domain analysis was set at 1 % ($p \leq .01$).

Results

Participants' demographics

Participants with AN had a mean age of 20.95 ± 5.15 years, and the mean age of onset of the first ED symptom was 15.72 ± 3.17. Participants with BN had a mean age of 22.37 ± 5.75 years, and the mean age of onset of the first ED symptom was 14.84 ± 3.41. The diagnoses duration mean was 4.11 ± 4.19 years for AN participants, and 5.28 ± 4.93 years for BN participants. Regarding exhibition of first symptoms, the mean was 5.22 ± 4.56 years and 7.53 ± 5.40 years for AN and BN participants, respectively. Mean body mass index was 15.07 ± 1.56 for AN participants, 21.15 ± 2.19 for BN participants, 20.77 ± 2.56 for non-psychiatric control participants and 21.04 ± 2.56 for psychiatric control participants. AN parental socioeconomic distribution was as follows: high (33, 33.7 %), middle (33, 33.7 %), and low (32, 32.7 %). Parental socioeconomic distribution for BN participants was: high (23, 29.1 %), middle (25, 31.6 %), and low (31, 39.2 %). The results were similar after participants from both control groups were individually matched to AN participants based on age and parental socioeconomic status (see Table 1).

Risk factors in AN versus bulimia nervosa group

Table 2 presents the distribution of putative risk factors in the AN versus BN, AN versus NC, and AN versus PC groups and the results of logistic regression analyses. Table 3 presents the overall level of exposure in each subdomain for these groups. Both tables summarize the

Table 1 Sociodemographic characteristics of the anorexia nervosa group (AN), bulimia nervosa group (BN), non-psychiatric (NC) and other psychiatric disorder (PC) control groups

	AN ($n = 98$) M (SD)	BN ($n = 79$) M (SD)	NC ($n = 86$) M (SD)	PC ($n = 68$) M (SD)
Index age (years)	15.72 (3.17)	14.84 (3.41)	–	–
Current age (years)	20.95 (5.15)	22.37 (5.75)	20.08 (4.24)	19.79 (4.74)
Current body mass index (kg/m^2)	15.07 (1.56)	21.15 (2.19)	20.77 (2.56)	21.04 (2.56)
	N (%)	*N* (%)	*N* (%)	*N* (%)
Education				
9th grade	46 (46.9)	30 (38)	25 (29.1)	28 (41.1)
12th grade	38 (38.8)	38 (48.1)	47 (54.7)	29 (42.6)
College/university	14 (14.2)	11 (13.9)	14 (16.3)	11 (16.2)
Parental socioeconomic status[a]				
High (I or II)	33 (33.7)	23 (29.1)	30 (34.9)	20 (29.4)
Middle (III)	33 (33.7)	25 (31.6)	33 (38.4)	17 (25)
Low (IV or V)	32 (32.7)	31 (39.2)	23 (26.8)	31 (45.6)

AN participants were matched for age and parental socioeconomic status to NC and PC participants

M mean, *SD* standard deviation

[a] Based on an adaptation of the Graffar Schedule

results of the comparisons of the AN group with the BN, NC and PC groups.

Compared to the BN group, participants with AN reported significantly greater levels of exposure to all but one of the 16 subdomains (i.e., sexual, physical and psychological abuse; see Table 3).

Concerning the individual risk factors, the BN group reported significantly greater levels of exposure than the AN group to four risk factors: paternal high expectations, excessive importance about fitness/keeping in shape given by family, negative consequences because of adolescent overweight and adolescent objective overweight (all $p \leq .01$; $2.25 \leq \text{OR} \leq 3.58$; see Table 2).

AN versus non-psychiatric disorder control group

Compared to the NC group, participants with AN reported significantly greater levels of exposure to all except 1 of the 17 subdomains (i.e., behavioral problems; see Table 3). A greater degree of exposure within each subdomain/domain was associated with a greater risk of developing AN. The AN participants reported significantly greater levels of exposure in regard to perfectionism, self-consciousness about appearance, unresolved/unaddressed family disagreements, teasing, parental comments about eating, negative attitudes toward parents shape and weight, feeling fat with significant concern, being teased by peers about shape, weight, eating, and appearance, having a family history of AN or BN and antecedent life events (all $p \leq .01$; $2.43 \leq \text{OR} \leq 3.92$; see Table 2).

AN versus psychiatric control group

Compared to the PC group, participants with AN reported significantly greater levels of exposure to all but two of the 15 subdomains (i.e., parental relationship and father–daughter relationship; see Table 3). A greater degree of exposure within each subdomain/domain was again associated with a greater risk of developing AN. The AN group reported significantly greater levels of exposure than the PC group to eight risk factors: perfectionism, self-consciousness about appearance, unresolved/unaddressed family disagreements, teasing, negative attitudes toward parents' shape and weight, significant concern about feeling fat, a family history of AN or BN and antecedent life events (all $p \leq .01$; $\text{OR} \leq 3.94$; see Table 2).

Discussion

When comparing AN versus BN, we found four retrospective correlates that distinguished AN participants from BN participants: paternal high expectations, family excessive importance about fitness/keeping in shape, adolescent objective overweight and negative consequences because of adolescent overweight. All were associated with the highest risk for BN development. As mentioned earlier, in the case–control study that compared AN risk factors with BN risk factors and two control groups, Fairburn et al. [18] concluded that women who developed BN seem to be vulnerable to become heavier than peers which, in addition to social consequences and some other retrospective correlates such as parental obesity and early menarche, encourage dieting. Considering our results, we further strengthen Fairburn's results about being overweight during a critical developmental period: adolescence. Compared to AN, BN participants seem to be prone to have a development environmental context marked by high expectations, excessive importance of keeping in shape, and all factors combined with overweight have negative consequences on the individual. This picture may predispose adolescents to engage in diets. Diet is commonly one of the first symptoms in AN and BN, and there are various ED risk pathways. We should consider that these results are consistent with risk factors implicated in the onset of threshold, subthreshold and partial ED. Stice et al. [33] found that body dissatisfaction was the strongest predictor of risk in the onset of any ED, with risk being amplified by depressive symptoms. As the authors commented, increasing the effectiveness of prevention programs that target qualitatively distinct risk groups, rather than only individuals with a single risk factor, may be a possible solution. In defining community high-risk groups that may benefit from prevention programs that cover the ED risk spectrum, all etiology factors that are relevant to prevent ED onset should be anticipated.

Because AN retrospective correlates were already presented and discussed in a previous study [24], we focused on AN versus BN comparisons. Briefly, in terms of specific retrospective correlates for AN, we determined that perfectionism, participant's self-consciousness regarding appearance, unresolved family disagreements, teasing, negative attitudes regarding parents' shape and weight, feeling fat, a family history of ED and antecedent life events were associated with the highest risk for AN. Moreover, parental comments about eating and being teased (specifically related to shape, weight, eating, and/or appearance) emerged as retrospective correlates for general psychopathology. These results are consistent with previous research in which general retrospective correlates have been discussed in relation to specific retrospective correlates for AN development [18, 19, 22, 24]. Perfectionism and family history of ED seem to be central in the understanding of AN etiology, specifically if associated with other factors that increase the vulnerability for dieting (such as being self-consciousness about appearance and

Table 2 Distribution of putative risk factors in the anorexia nervosa group (AN) matched for age and parental socioeconomic status to non-psychiatric (NC) and other psychiatric (PC) control groups and risk factors in the bulimia nervosa group (BN) using univariate logistic regression analyses

Risk factors	Group, *n* (%)				Anorexia nervosa subjects vs					
					Bulimia nervosa group		Non-psychiatric control group		Psychiatric control group	
	AN ($n = 98$)	BN ($n = 79$)	NC ($n = 86$)	PC ($n = 68$)	*p*	Odds ratio (95 % CI)	*p*	Odds ratio (95 % CI)	*p*	Odds ratio (95 % CI)
Personal vulnerability domain										
Subdomain 1–childhood characteristics										
Negative self-evaluation	44 (44.9)	47 (59.5)	18 (21.2)	22 (32.4)	.263	1.46 (.75–2.82)	.503	.34 (.34–1.71)	.978	1.01 (.49–2.09)
Shyness	44 (44.9)	36 (45.6)	23 (27.1)	24 (35.3)	–	–	.575	.80 (.37–1.74)	–	–
Perfectionism	65 (66.3)	53 (67.1)	32 (37.6)	30 (44.1)	–	–	**.002**	**.34** (.17–.67)	**.008**	**.40** (.20–.79)
No close friends	15 (15.3)	22 (27.8)	7 (8.1)	10 (14.7)	.214	1.64 (.75–3.58)	–	–	–	–
Anxiety	21 (21.4)	29 (36.7)	10 (11.8)	17 (25)	.101	1.78 (.89–3.56)	.778	1.16 (.41–3.28)	–	–
Self–consciousness about appearance	29 (29.6)	32 (40.5)	1 (1.2)	5 (7.4)	.582	1.21 (.61–2.42)	**.001**	**.03** (.004–.27)	**.003**	**.19** (.07–.56)
Height	23 (23.5)	13 (16.5)	7 (8.2)	8 (11.8)	–	–	.065	2.59 (.94–7.14)	.263	1.70 (.67–4.28)
Subdomain 2: Premorbid psychiatric disorder										
Enuresis	8 (8.2)	14 (17.7)	8 (9.3)	8 (11.8)	.061	2.42 (.96–6.11)	–	–	–	–
Other than mood psychiatric disorders (like anxiety disorders)	9 (9.2)	8 (10.1)	0	1 (1.5)	–	–	–	–	.073	.15 (.02–1.19)
Subdomain 3: Behavioral problems	–	–	–	–	–	–	–	–	–	–
Subdomain 4: Family/parental psychiatric disorder (ever)										
Depression	50 (51)	52 (65.8)	31 (36.5)	35 (51.5)	.487	1.40 (.55–3.50)	.130	.62 (.34–1.15)	–	–
Parental depression	35 (35.7)	41 (51.9)	25 (29.1)	28 (41.2)	.283	.61 (.24–1.51)	–	–	–	–
Obsessive–compulsive disorder	11 (11.2)	12 (15.2)	1 (1.2)	1 (1.5)	–	–	.028	.09 (.01–.78)	.042	.12 (.01–.93)
Alcoholism	31 (31.6)	31 (39.2)	14 (16.5)	29 (42.6)	–	–	.133	.52 (.22–1.22)	.926	1.04 (.44–2.46)
Parental alcoholism	13 (13.3)	10 (12.7)	4 (4.7)	18 (26.5)	–	–	.425	1.76 (.44–7.02)	.117	.43 (.15–1.24)
Drug abuse	12 (12.2)	18 (22.8)	6 (7)	12 (17.6)	.037	2.41 (1.06–5.49)	–	–	–	–
Environmental domain										
Subdomain 1 – Parental problems										
Family dynamic/context										
Low parental contact	34 (34.7)	24 (30.4)	14 (16.3)	30 (44.1)	–	–	.031	.36 (.14–.91)	–	–
Separation from parents	20 (20.4)	10 (12.7)	10 (11.6)	25 (36.8)	–	–	.929	.95 (.33–2.74)	.005	3.05 (1.41–6.58)
Family isolated	35 (35.7)	27 (34.2)	16 (18.6)	20 (29.4)	–	–	.015	.33 (.14–.81)	–	–
Parental arguments	36 (36.7)	43 (54.4)	18 (20.9)	28 (41.2)	.273	1.59 (.70–3.63)	.865	.90 (.27–3.02)	–	–
Parental arguments (subject involved)	24 (24.5)	32 (40.5)	9 (10.5)	23 (33.8)	.496	1.36 (.56–3.30)	.640	.72 (.18–2.83)	–	–

Table 2 continued

Risk factors	Group, *n* (%)				Anorexia nervosa subjects vs					
					Bulimia nervosa group		Non-psychiatric control group		Psychiatric control group	
	AN ($n = 98$)	BN ($n = 79$)	NC ($n = 86$)	PC ($n = 68$)	*p*	Odds ratio (95 % CI)	*p*	Odds ratio (95 % CI)	*p*	Odds ratio (95 % CI)
Arguments within the home	40 (40.8)	34 (43)	17 (19.8)	26 (38.2)	–	–	.912	.94 (.34–2.63)	–	–
Family disagreements not resolved/avoided	51 (52)	39 (49.4)	9 (10.5)	23 (33.8)	–	–	**<.001**	**.12** (.05–.29)	**.013**	**.41** (.21–.83)
Negative self–evaluation compared with siblings	27 (27.6)	28 (35.4)	8 (9.3)	10 (14.7)	–	–	.099	.38 (.12–1.20)	.143	.48 (.18–1.28)
Favoritism (other sibling was favorite)	26 (26.5)	33 (41.8)	16 (18.6)	18 (26.5)	.080	.56 (.29–1.07)	–	–	–	–
Sibling rivalry	19 (19.4)	21 (26.6)	5 (5.8)	5 (7.4)	–	–	.513	.63 (.16–2.49)	.352	.56 (.17–1.89)
Family tension during meals	33 (33.7)	27 (34.2)	10 (11.6)	23 (33.8)	–	–	.770	1.21 (.34–4.36)	–	–
Family tension during meals (related to food/eating)	17 (17.3)	10 (12.7)	2 (2.3)	6 (8.8)	–	–	.049	7.18 (1.01–50.91)	.219	1.94 (.68–5.56)
Parental relationship										
Parental criticism	32 (32.7)	38 (48.1)	14 (16.3)	31 (45.6)	.112	1.67 (.89–3.15)	.175	.58 (.26–1.28)	.136	1.63 (.86–3.11)
Parental underinvolvement	62 (63.3)	55 (69.6)	37 (43)	46 (67.6)	–	–	.165	1.64 (.82–3.30)	–	–
Parental overinvolvement	3 (3.1)	9 (11.4)	0	2 (2.9)	.077	.29 (.08–1.14)	–	–	–	–
Parental minimal affection	52 (53.1)	37 (46.8)	28 (32.6)	33 (48.5)	–	–	.330	.69 (.33–1.16)	–	–
Parental high expectations	54 (55.1)	60 (75.9)	42 (48.8)	33 (48.5)	.016	2.27 (1.16–4.43)	–	–	–	–
Excessive parental control	42 (42.9)	37 (46.8)	28 (32.6)	37 (54.4)	–	–	.584	1.20 (.63–2.28)	.217	.67 (.36–1.26)
Mother–daughter relationship										
Maternal underinvolvement	27 (27.6)	27 (34.2)	15 (17.4)	21 (30.9)	–	–	.195	1.96 (.76–3.76)	–	–
Maternal overinvolvement	11 (11.2)	16 (20.3)	1 (1.2)	4 (5.9)	.164	.55 (.23–1.28)	.028	10.54 (1.30–85.63)	–	–
Maternal minimal affection	23 (23.5)	19 (24.1)	12 (14)	16 (23.5)	–	–	.561	.77 (.32–1.85)	–	–
Maternal high expectations	47 (48)	31 (36)	31 (36)	25 (36.8)	.041	1.92 (1.03–3.58)	.475	.79 (.42–1.50)	–	–
Maternal criticism	23 (23.5)	29 (36.7)	9 (10.5)	20 (29.4)	.158	1.63 (.83–3.19)	.167	.53 (.21–1.31)	–	–
Father–daughter relationship										
Paternal underinvolvement	55 (56.1)	41 (51.9)	31 (36)	38 (55.9)	–	–	.054	1.94 (.99–3.80)	–	–
Paternal minimal affection	46 (46.9)	32 (40.5)	27 (31.4)	24 (35.3)	–	–	.335	.71 (.36–1.42)	.136	.62 (.33–1.17)
Paternal high expectations	48 (49)	54 (68.4)	39 (45.3)	27 (39.7)	**.010**	**2.25** (1.21–4.17)	–	–	–	–
Subdomain 2: Disruptive events										
Parental death	5 (5.1)	7 (8.9)	7 (8.1)	9 (13.2)	–	–	–	–	.125	2.93 (.74–11.60)

Table 2 continued

Risk factors	Group, *n* (%)				Anorexia nervosa subjects vs					
					Bulimia nervosa group		Non-psychiatric control group		Psychiatric control group	
	AN ($n = 98$)	BN ($n = 79$)	NC ($n = 86$)	PC ($n = 68$)	*p*	Odds ratio (95 % CI)	*p*	Odds ratio (95 % CI)	*p*	Odds ratio (95 % CI)
Parental chronic illness	15 (15.3)	10 (12.7)	5 (5.8)	4 (5.9)	–	–	.374	1.95 (.45–8.57)	.243	2.04 (.62–6.76)
Family chronic illness	25 (25.5)	13 (16.5)	13 (15.1)	11 (16.2)	–	–	.444	.66 (.22–1.96)	–	–
Change of parent figure	9 (9.2)	12 (15.2)	4 (4.7)	24 (35.3)	–	–	–	–	.004	3.81 (1.53–9.52)
Frequent house moves	61 (62.2)	53 (67.1)	60 (69.8)	52 (76.5)	–	–	–	–	.108	1.87 (.87–4.02)
Severe personal health problems	13 (13.3)	19 (24.1)	3 (3.5)	11 (16.2)	.091	1.97 (.90–4.33)	–	–	–	–
Severe personal health problems (weight/appearance changed)	12 (12.2)	13 (16.5)	1 (1.2)	5 (7.4)	–	–	.029	.09 (.01–.78)	–	–
Teasing (not about shape, weight, eating or appearance)	59 (60.2)	49 (62)	25 (29.1)	28 (41.2)	–	–	**<.001**	.27 (.13–.56)	**.014**	**.42** (.21–.84)
Threatening teasing	20 (20.4)	25 (31.6)	8 (9.3)	9 (13.2)	.123	.58 (.29–1.16)	.844	1.11 (.40–3.09)	–	–
Subdomain 3: Family/parental psychiatric disorder										
Family alcoholism	29 (29.6)	26 (32.9)	14 (16.3)	27 (39.7)	–	–	.142	.53 (.23–1.23)	–	–
Parental alcoholism	11 (11.2)	9 (11.4)	4 (4.7)	18 (26.5)	–	–	.548	1.53 (.39–6.08)	.013	.35 (.15–.80)
Subdomain 4: Teasing and bullying										
Bullying	7 (7.1)	13 (16.5)	1 (1.2)	4 (5.9)	.058	2.56 (.97–6.77)	–	–	–	–
Subdomain 5: Sexual, physical and psychological abuse										
Sexual abuse	20 (20.4)	15 (19)	5 (5.8)	10 (14.7)	–	–	.177	.41 (.11–1.49)	–	–
Physical abuse	17 (17.3)	22 (27.8)	3 (3.5)	18 (26.5)	.462	1.53 (.49–4.79)	.362	.41 (.06–2.77)	–	–
Repeated and/or severe sexual or physical abuse	22 (22.4)	26 (32.9)	4 (4.7)	21 (30.9)	.688	1.24 (.43–3.60)	.550	.57 (.09–3.59)	–	–
Psychological maltreatment	18 (18.4)	17 (21.5)	4 (4.7)	9 (13.2)	–	–	.098	.36 (.11–1.21)	–	–
Dieting vulnerability domain										
Subdomain 1: Dieting risk										
Family weight and eating concerns										
Family member dieting for shape or weight	38 (38.8)	36 (45.6)	19 (22.6)	23 (33.8)	–	–	.902	.94 (.37–2.44)	–	–
Parents' dieting for shape or weight	21 (21.4)	13 (16.5)	2 (2.4)	12 (17.6)	–	–	.024	7.35 (1.30–41.67)	–	–
Critical comments by family about shape or weight	52 (53.1)	56 (70.9)	31 (36.9)	44 (64.7)	.024	2.14 (1.11–4.14)	.507	.79 (.40–1.58)	.083	.52 (.25–1.09)
Parents' underweight	12 (12.2)	4 (5.1)	1 (1.2)	3 (4.4)	.038	3.73 (1.07–12.99)	–	–	.201	2.52 (.61–10.35)
Family shape or weight concern	36 (36.7)	31 (39.2)	19 (22.6)	14 (20.6)	–	–	.931	1.04 (.44–2.67)	.430	.69 (.27–1.74)
Parents shape or weight concern	16 (16.3)	11 (13.9)	3 (3.6)	4 (5.9)	–	–	.336	2.27 (.43–12.03)	.328	2.06 (.49–8.71)

Table 2 continued

Risk factors	Group, *n* (%)				Anorexia nervosa subjects vs					
					Bulimia nervosa group		Non-psychiatric control group		Psychiatric control group	
	AN ($n = 98$)	BN ($n = 79$)	NC ($n = 86$)	PC ($n = 68$)	*p*	Odds ratio (95 % CI)	*p*	Odds ratio (95 % CI)	*p*	Odds ratio (95 % CI)
Family history of eating disorders	16 (16.3)	13 (16.5)	6 (7.1)	1 (1.5)	–	–	.548	.70 (.22–2.25)	.057	.13 (.02–1.06)
Family overeating	17 (17.3)	17 (21.5)	8 (9.5)	10 (14.7)	–	–	.551	.73 (.27–2.03)	–	–
Family excessive importance about fitness/keeping in shape	10 (10.2)	24 (30.4)	2 (2.4)	4 (5.9)	**.007**	**3.58** (1.42–9.04)	.131	.29 (.06–1.45)	–	–
Family excessive importance about appearance	15 (15.3)	22 (27.8)	7 (8.1)	2 (2.9)	.706	1.18 (.49–2.84)	–	–	.139	.29 (.06–1.49)
Parents' repeated comments about eating	55 (56.1)	38 (48.1)	25 (29)	25 (36.8)	–	–	**.011**	**2.43** (1.23–4.80)	.057	1.94 (.98–3.84)
Subject's weight and eating concerns										
Negative attitude towards parents' shape and weight	17 (17.3)	20 (25.3)	1 (1.2)	1 (1.5)		–	**.007**	**.05** (.001–.43)	**.014**	**.07** (.01–.59)
Encouraged to diet by family member	17 (17.3)	22 (27.8)	6 (7)	7 (10.3)	.978	1.01 (.44–1.56)	.970	.98 (.30–3.24)	–	–
Childhood underweight	13 (13.3)	11 (13.9)	19 (22.1)	9 (13.2)		–	.734	.85 (.32–2.21)	–	–
Adolescent overweight	22 (22.4)	40 (50.6)	7 (8.1)	8 (11.8)		–	.451	2.01 (.33–12.32)	–	–
Negative consequences of childhood overweight	13 (13.3)	17 (21.5)	3 (3.5)	6 (8.8)		–	.405	1.89 (.42–8.51)	–	–
Negative consequences of adolescent overweight	18 (18.4)	38 (48.1)	4 (4.7)	7 (10.3)	**.002**	**.30** (.14–.64)	.124	5.38 (.63–45.99)	.772	.85 (.28–2.59)
Diet with friends	10 (10.2)	14 (17.7)	3 (3.5)	4 (5.9)	–	–	.130	.33 (.08–1.39)	–	–
Feeling big with concern	26 (26.5)	29 (36.7)	8 (9.3)	7 (10.3)	.939	1.03 (.49–2.16)	.413	1.55 (.55–4.38)	.550	1.39 (.47–4.08)
Feeling fat with concern	54 (55.1)	60 (75.9)	17 (19.8)	15 (22.1)	.111	.55 (.26–1.15)	**.006**	**3.08** (1.39–6.80)	**.001**	**3.94** (1.72–8.99)
Sisters' shape: slimmer with concern	18 (18.4)	24 (30.4)	4 (4.7)	3 (4.4)	.411	.73 (.34–1.56)	.998	.99 (.09–10.49)	.833	1.31 (.11–15.45)
Sisters' shape: more attractive with concern	17 (17.3)	19 (24.1)	4 (4.7)	3 (4.4)	–	–	.308	3.38 (.33–35.19)	.356	3.25 (.27–39.75)
Others' weight and eating related behaviors										
Teasing about shape, weight, eating or appearance	46 (46.9)	45 (57)	15 (17.4)	19 (27.9)	–	–	**<.001**	**3.92** (1.96–7.85)	.015	2.28 (1.18–4.42)
Repeated comments by others about shape and weight	43 (43.9)	47 (59.5)	31 (36)	25 (36.8)	.079	1.73 (.94–3.20)	–	–	–	–
Repeated comments by others about eating	20 (20.4)	25 (31.6)	6 (7)	8 (11.8)	.192	1.60 (.79–3.21)	.036	.34 (.13–.93)	–	–
Parents and subjects obesity before index age										
Parental obesity	43 (43.9)	41 (51.9)	26 (30.2)	27 (39.7)	–	–	.09	.59 (.32–1.09)	–	–
Maternal overweight	28 (28.6)	32 (40.5)	17 (19.8)	16 (23.5)	.097	1.70 (.91–3.19)	–	–	–	–
Childhood overweight	24 (24.5)	23 (29.1)	9 (10.5)	8 (11.8)	–	–	.024	.38 (.17–.88)	.045	.41 (.17–.98)

Table 2 continued

Risk factors	Group, *n* (%)				Anorexia nervosa subjects vs					
					Bulimia nervosa group		Non-psychiatric control group		Psychiatric control group	
	AN ($n = 98$)	BN ($n = 79$)	NC ($n = 86$)	PC ($n = 68$)	*p*	Odds ratio (95 % CI)	*p*	Odds ratio (95 % CI)	*p*	Odds ratio (95 % CI)
Subdomain 2: Obesity risk										
Parental obesity (ever)	49 (50)	38 (48.1)	27 (31.4)	27 (39.7)	–	–	.048	.53 (.29–.99)	–	–
Maternal overweight (ever)	35 (35.7)	31 (39.2)	16 (18.6)	17 (25)	–	–	–	–	.184	.62 (.31–1.25)
Childhood overweight	24 (24.5)	23 (29.1)	9 (10.5)	8 (11.8)	–	–	.153	.52 (.21–1.28)	.166	.51 (.20–1.32)
Adolescent overweight	22 (22.4)	40 (50.6)	7 (8.1)	8 (11.8)	**<.001**	**3.54** (1.85–6.77)	.126	.46 (.17–1.24)	.339	.62 (.24–1.64)
Subdomain 3: Family/parental eating disorders										
Family history of anorexia nervosa or bulimia nervosa (ever)	26 (26.5)	24 (30.4)	8 (9.3)	3 (4.4)	–	–	**.004**	**.28** (.12–.67)	**.001**	**.13** (.04–.44)
Additional risk factors										
Age at menarche: 12	29 (29.6)	24 (30.4)	26 (30.2)	30 (44.1)	–	–	–	–	.120	1.79 (.86–3.75)
Negative feelings about menstruation	26 (26.5)	21 (26.6)	12 (14)	4 (5.9)	–	–	.043	.42 (.18–.98)	**.003**	**.168** (.05–.54)
Breasts embarrassment with disguise	27 (27.6)	22 (27.8)	15 (17.4)	13 (19.1)	–	–	.285	1.53 (.70–3.34)	–	–
Antecedent life events	92 (93.9)	75 (94.9)	61 (70.9)	56 (82.4)	–	–	**.010**	**.23** (.08–.71)	**.006**	**.208** (.07–.64)
More than one antecedent life event	73 (74.5)	68 (86.1)	39 (45.3)	44 (64.7)	.060	.47 (.22-1.03)	.103	1.87 (.88–3.96)	–	–
Importance of religion during childhood	12 (12.2)	16 (20.3)	17 (19.8)	26 (38.2)	–	–	–	–	.000	5.10 (2.19–11.94)

The significance of the exposure (likelihood ratio statistic, χ^2) and odds ratios with their significance levels and 95 % confidence intervals (CI) are given for each factor. All exposures, except those labeled "ever", predate the onset of the eating disorder; '–' indicates that risk factor failed to meet relevant statistical assumptions for the analysis; values ≤.01 are in bold

Table 3 Overall level of exposure in each risk factor subdomain in the four groups[†]

Domain/subdomains	Anorexia nervosa subjects vs								
	Bulimia nervosa group			Non-psychiatric control group			Psychiatric control group		
	χ^2	*df*	*p*	$\chi 2$	*df*	*p*	χ^2	*df*	*p*
Personal vulnerability domain									
Subdomain 1: childhood characteristics	9.73	4	<.05	52.47	6	**<.001**	23.29	4	**<.001**
Subdomain 2: premorbid psychiatric disorder	3.66	1	**<.10**	–	–	–	5.02	1	<.05
Subdomain 3: behavioral problems	–	–	–	1.81	1	ns	–	–	–
Subdomain 4: family/parental psychiatric disorder (ever)	9.50	3	<.05	17.98	4	**<.01**	11.47	3	**<.01**
Environmental domain									
Subdomain 1: parental problems									
Family dynamic/context	9.47	3	<.05	68.51	11	**<.001**	21.06	5	**<.01**
Parental relationship	14.80	3	**<.01**	12.76	4	<.05	4.37	2	ns
Mother-daughter relationship	10.08	3	<.05	17.03	5	**<.01**	–	–	–
Father-daughter relationship	6.81	1	**<.01**	8.40	2	<.05	2.25	1	ns
Subdomain 2: disruptive events	5.81	2	<.10	31.30	5	**<.001**	28.39	5	**<.001**
Subdomain 3: family/parental psychiatric disorder	–	–	–	4.10	2	<.10	6.37	1	<.05
Subdomain 4: teasing and bullying	3.78	1	<.10	–	–	–	–	–	–
Subdomain 5: sexual, physical and psychological abuse	2.96	2	ns	19.52	4	**<.01**	–	–	–
Dieting vulnerability domain									
Subdomain 1: dieting risk									
Family weight and eating concerns	20.97	4	**<.001**	34.74	9	**<.001**	27.38	7	**<.001**
Subject's weight and eating concerns	21.88	5	**<.01**	51.79	12	**<.001**	36.70	6	**<.001**
Others' weight and eating related behaviors	5.99	2	<.10	23.54	2	**<.001**	6.20	1	<.05
Parents' and subjects' obesity before index age	2.77	1	<.10	9.20	2	<.05	4.39	1	<.05
Subdomain 2: Obesity risk	15.38	1	**<.001**	13.84	3	**<.01**	7.18	3	<.10
Subdomain 3: Family/parental eating disorders	–	–	–	9.49	1	**<.01**	15.82	1	**<.001**
Additional risk factors	3.73	1	<.10	28.22	4	**<.001**	38.02	4	**<.001**

The significance of the exposure (likelihood ratio statistic, χ^2) is given for each subdomain. All exposures, except those labeled "ever", predate the onset of the eating disorder; '–' indicates that risk factor failed to meet relevant statistical assumptions for the analysis; values ≤.01 are in bold

feeling fat). Prospective studies to confirm this hypothesis are needed.

Putting together the results obtained about AN vs. BN participants and AN vs. NC and PC groups we seem to have two potential pathways of risk. For AN development we confirmed perfectionism and ED in family and for BN, when compared with AN participants, we sustained being overweight during adolescence with negative consequences. Both sets of risk factors placed young women at risk to engage in diets. Commonly, diet is one of the first symptoms for AN and BN despite the confirmed presence of distinct risk factors between both ED.

Moreover, the discussed risk factors were determined by all female participants, and could be different for males, who also exhibit ED but are underrepresented in the literature.

As we already reflected [24], the current study has several limitations. The most important limitation is inherent in retrospective case–control designs, namely potential biases associated with recall. Although we made every effort to maximize the accuracy of recall, bias is unavoidable. We did not involve other informants, such as relatives or significant others, and the methodology concerning family issues was based on family history reported by the participants who were being evaluated (in contrast to a family study design [20].

However, the convergence of our findings with previous reports on the clarification of the specificity of AN risk factors [18, 22, 34], in addition to the differences between AN and BN risk pathways, clarifies the characteristics that should be considered in targeting high-risk groups and improve the effectiveness of tailored prevention programs for ED and their specific pathology.

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Compliance with ethical standards

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

Ethical standard This study was reviewed and approved by the Institutional Review Board and conformed to both Portuguese and European regulations on conducting research with human participants and on the management of personal data.

Informed consent All participants gave written informed consent, and in the case of minors, child assent and parental consent for research participation were obtained.

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