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Toward an understanding of risk factors for anorexia nervosa: a case-control study

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Background. Prospective, longitudinal studies of risk factors for anorexia nervosa (AN) are lacking and existing cross-sectional studies are generally narrow in focus and lack methodological rigor. Building on two studies that used the Oxford Risk Factor Interview (RFI) to establish time precedence and comprehensively assess potential risk correlates for AN, the present study advances this line of research and represents the first case-control study of risk factors for AN in the USA.

Method. The RFI was used for retrospective assessment of a broad range of risk factors, while establishing time precedence. Using a case-control design, 50 women who met DSM-IV criteria for AN were compared to those with non-eating disorder DSM-IV psychiatric disorders ($n = 50$) and those with no psychiatric disorder ($n = 50$).

Results. Women with psychiatric disorders reported higher rates of negative affectivity, maternal and paternal parenting problems, family discord, parental mood and substance disorder, and physical and sexual abuse than women with no psychiatric disorder. Women with AN specifically reported greater severity and significantly higher rates of negative affectivity, perfectionism and family discord, and higher parental demands than women with other psychiatric disorders. The role of weight and shape concerns was most salient in the year preceding onset of AN.

Conclusions. Convergent data identifying common risk factors as well as those more severe in the development of AN are emerging to inform longitudinal risk factor and prevention studies for this disorder.

Introduction

Anorexia nervosa (AN) is a psychiatric disorder associated with profound morbidity and dramatically elevated mortality rates. Although recognized for centuries, definitive longitudinal, population-based studies identifying risk factors for AN do not exist and therefore the current knowledge base on the etiology of AN remains limited (Jacobi *et al.* 2004; Commission on Adolescent Eating Disorders, 2005).

A significant challenge in studying the etiology of AN is capturing the complexity of this multi-determined disorder. Potential contributions to increased risk for AN span from the prenatal stage to early adulthood and occur across multiple levels of biology and environment. Another challenge is the low prevalence of the disorder; epidemiological studies indicate that AN afflicts less than 1% of females (Hoek, 2002; Hoek & van Hoeken, 2003; McKnight Investigators, 2003; Commission on Adolescent Eating Disorders, 2005).

The rarity and intrinsic complexities in the development of AN confer methodological challenges. Studies are often compromised by over-reliance on self-report survey data or loosening of diagnostic criteria to generate sufficient samples. Many studies are also limited by narrow focus, failure to establish time precedence, and lack of appropriate comparison groups. Jacobi *et al.* (2004) identified 28 longitudinal studies examining the etiology of eating psychopathology but none of these studies generated a sufficient number of cases to have adequate statistical power to contribute to the specific knowledge base of AN. Thus, cross-sectional studies using epidemiological,

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case-control and family history designs remain crucial to building an initial database and developing hypotheses that can subsequently be tested in longitudinal investigations of the etiology of AN.

With increasingly sophisticated methodologies, the complex relationships among biological, environmental and developmental factors are gradually emerging. Gender and age are associated with increased risk for AN. Females are 10 times more likely than males to develop AN (Jacobi *et al.* 2004), and eating disorders most commonly arise during adolescence and early adulthood. Genetic contribution is best understood in relation to specific traits or symptoms, but specific genes have yet to be identified (Kendler *et al.* 1995; Hinney *et al.* 2004; Commission on Adolescent Eating Disorders, 2005; Mazzeo *et al.* 2006). Mixed findings exist for the role of parenting problems, early childhood experiences, family psychiatric history and health factors as potential retrospective correlates of AN (i.e. retrospectively assessed factors that predate the onset of the disorder; Kraemer *et al.* 1997). Thus, AN is a developmental disorder that targets females; however, the task remains to identify those factors that explain why certain females develop AN while most do not.

Two studies using the Oxford Risk Factor Interview (RFI; Fairburn *et al.* 1999; Karwautz *et al.* 2001) redress many methodological limitations of earlier studies by using an interview to establish diagnosis, time precedence and evaluate a wide range of putative risk factors comprehensively. (The RFI is available in the online Appendix.) Findings from both studies suggest that although family and individual history of weight, shape and dieting concerns are significant in predicting the development of AN when considered in isolation, their relative contribution is less than other personal vulnerability factors when evaluated in comprehensive models. These findings are especially provocative given the centrality of weight and shape concerns described in the clinical presentation of AN. The personal vulnerability factors that appear to be most significant in predicting AN are perfectionism and negative affectivity. Both studies also reported high parental expectations, parental history of depression, and childhood physical and sexual abuse as significant in the etiology of AN; however, the comparisons with control groups with bulimia nervosa and other psychiatric disorders in the Fairburn *et al.* (1999) study suggest that these factors are not unique to AN.

Although the investigations by Fairburn *et al.* (1999) and Karwautz *et al.* (2001) represent significant advances in the study of risk factors for AN, methodological limitations warrant note. Fairburn *et al.* used an unmatched design, and Karwautz *et al.* focused primarily on non-shared environmental factors and therefore only included a control group composed of sisters (not matched for age). The present study moves the field forward by using the RFI in a matched case-control study that includes psychiatric and non-psychiatric control groups. AN and control cases are matched for current age and the developmental period assessed.

The present investigation also contributes to the literature regarding antecedent life events that may be proximal triggers associated specifically with the onset of AN. Some evidence suggests an increased number of adverse life events in the year prior to onset of AN (Rastam & Gillberg, 1992). However, other data suggest no differences in the *number* of adverse life events (Troop & Treasure, 1997) but emphasize the role that specific types and particularly severe life events (e.g. loss of a first-degree relative) play as proximal triggers for onset of AN (Schmidt *et al.* 1997). Limited data suggest that adverse life events that function as proximal triggers for AN may be common factors associated with the onset of a range of psychiatric disorders (Horesh *et al.* 1995; Gowers *et al.* 1996). In studies of binge eating disorder (BED; Pike *et al.* 2006) and bulimia nervosa (BN; Fairburn *et al.* 1997), interpersonal stressors and specific comments about weight and shape were especially salient antecedents to the eating disturbances; whether these antecedent life events are also associated with the onset of AN remains unknown.

We hypothesized that, consistent with earlier findings, parental psychopathology, parenting problems, and physical and sexual abuse are general retrospective correlates common to AN and other psychiatric disorders. We hypothesized that family and personal histories of weight, shape and eating concerns, and personal vulnerability factors of perfectionism and negative affectivity are significant retrospective correlates that are more severe for AN than for other psychiatric disorders. Regarding proximal antecedent life events, we hypothesized that similar stressors are associated with AN and for other eating disorders. Specifically, we hypothesized that critical comments about weight, shape and eating represent specific risk for the onset of AN and these factors, coupled with more general interpersonal events, increase in the year immediately preceding the onset of AN. We also hypothesized that the impact of these stressors is cumulative, an increased number of stressors being associated with an increased risk for onset of AN.

Method

Design and recruitment

Under the auspices of the New England Women's Health Project (NEWHP) and the New York State

Psychiatric Institute Eating Disorders Research Unit (NYSPI-EDRU), a case-control design compared women with AN, women with no psychiatric diagnosis, and women with non-eating disorder DSM-IV psychiatric diagnoses. After completing a telephone screening interview, eligible individuals were invited to participate in diagnostic interviews, a risk factor interview, and several self-report instruments. Height and weight were measured.

Two recruitment strategies were used. The first involved telephone recruitment using a consumer information database of 10 000 women, aged 18–40 years. The second was an advertising campaign consisting of posters, newspaper advertisements, community referrals and public service announcements that invited interested individuals to call a study phone number. The consumer database yielded the majority of the control group with no psychiatric diagnosis. The psychiatric control group was recruited approximately equally from the consumer database and advertising campaign. AN cases were recruited from the NYSPI-EDRU. All individuals who contacted the unit between 1998 and 2002 and met criteria for current AN were invited to participate in the current study. Of this sample, approximately 50% pursued treatment upon study completion.

Institutional review boards at Wesleyan and Columbia Universities approved this study. (For additional methodological details, see Striegel-Moore *et al.* 2005.)

Participants

Participants in this study were 50 women with AN as the primary diagnosis (AN group), 50 women with no psychiatric diagnosis (NC group) and 50 women with non-eating disorder DSM-IV psychiatric diagnoses (PC group). Exclusion criteria for all groups were physical conditions known to influence eating or weight, current pregnancy, or presence of psychotic disorder. Inclusion criteria for the NC group were absence of past or current clinically significant eating disorder symptoms and absence of a current psychiatric disorder. For the PC group, inclusion required a current DSM-IV Axis I psychiatric disorder, but no history of clinically significant eating disorder symptoms. PC group members carried the following primary DSM-IV diagnoses (based on clinical interview as described below): mood disorder ($n=27$, 54.0%), anxiety disorder ($n=17$, 34.0%), substance disorder ($n=2$, 4.0%), and other DSM-IV diagnoses (e.g. body dysmorphic disorder; $n=4$, 8.0%). In the PC group, 28 individuals (56.0%) had at least one co-morbid diagnosis. In the AN group, 39 individuals (78.0%) had at least one co-morbid diagnosis.

Matching and establishment of index age

NC and PC participants were matched individually to participants with AN on current age (± 2 years), and were assigned an index age corresponding to the index age of the AN case to which they were matched. The index age represents the age at which clinically significant eating disturbances emerged for the first time for each AN participant, as determined at the beginning of the Oxford RFI (described below). Clinically significant eating disturbance was defined as the age at which at least one of the following dimensions of eating pathology first began: sustained dieting (i.e. dieting for at least 3 consecutive months); sustained overeating (i.e. overeating, on average, at least once a week for at least 3 consecutive months); sustained purging (i.e. efforts at purging at least once a week for at least 3 consecutive months). All AN cases and matched controls were Caucasian; this racial distribution corresponds to epidemiological findings (see Jacobi *et al.* 2004).

Groups did not differ on the matching variables of age at time of study participation or index age ($p > 0.05$; see Table 1). They did not differ on education ($p > 0.05$) but, as expected, AN participants had a significantly lower current body mass index (kg/m²) than NC and PC participants ($p > 0.001$). Half of AN participants met diagnostic criteria for current restricting type AN (AN-R; $n=25$, 50.0%), and half ($n=25$, 50.0%) met criteria for current binge eating/purging type AN (AN-BP). Within this subtype, 19 participants reported both binge eating and purging (38.0% of the AN sample), and six (12.0% of the AN sample) reported purging in the absence of binge eating.

Assessment

Diagnostic assessment

Current and lifetime psychiatric disorders were assessed with the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-IV; First *et al.* 1996). Eating disorder diagnoses and psychopathology were assessed through an abbreviated diagnostic version of the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), a semi-structured investigator-based interview. At the outset of the EDE, the index age was determined in AN cases (Fairburn *et al.* 1998). Assessment of risk factors focused on the period before the index age, that is before onset of clinically significant eating symptoms (or age 18 years if it came first for those questions pertaining to family life and experience of parenting), thereby ensuring precedence of the risk factor to the onset of clinically significant eating disturbances. The index age of each AN case was applied to the matched individuals in the NC and

Table 1. *Sociodemographic characteristics: index age, current age, current body mass index, and education*

	AN ($n=50$)	PC ($n=50$)	NC ($n=50$)
Index age (years), mean (S.D.)[a]	14.66 (3.13)	14.38 (3.56)	14.66 (3.17)
Current age (years), mean (S.D.)[b]	26.70 (6.23)	27.02 (6.05)	26.56 (5.51)
Current body mass index (kg/m²), mean (S.D.)[c]	14.96 (2.02)	24.95 (6.29)	23.16 (4.63)
Education[d]			
High school or less, *n* (%)	7 (14.0)	11 (22.0)	6 (12.0)
Some college, *n* (%)	23 (46.0)	22 (44.0)	17 (34.0)
College graduate or higher, *n* (%)	20 (40.0)	17 (34.0)	27 (54.0)

AN, Anorexia nervosa; PC, psychiatric control group; NC, non-psychiatric control group; S.D., standard deviation.

[a] Repeated measures analysis, $F(1, 73)=2.09$, $p=0.143$.
[b] Repeated measures analysis, $F(2, 92)=1.83$, $p=0.169$.
[c] Repeated measures analysis, $F(2, 81)=65.37$, $p<0.001$; *post hoc* paired t tests: AN<PC, NC, $p<0.001$.
[d] Stuart–Maxwell χ^2 tests of overall marginal homogeneity, all $p>0.05$.

PC groups to ensure assessment of the same developmental windows across groups.

Risk factor assessment

Exposure to putative risk factors was assessed using the RFI (Fairburn *et al.* 1998). The RFI assesses biological, psychological and social factors thought to place a person at risk for the development of an eating disorder. To minimize bias associated with retrospective reporting, the RFI uses behavioral definitions of key concepts and establishes a timeline for sequences of events. Degree of exposure to a potential risk factor is rated on a five-point rating scale ranging from 0=no exposure to 4=high severity, long duration, or high frequency of exposure. A conservative cut-off score of 3 or 4 was used when calculating exposure to life events to reduce the risk of false positives. In addition, the Parental Bonding Instrument (PBI; Parker *et al.* 1979), a self-report questionnaire assessing participants' experience of both parents up to index age, was administered. Measures of parental control, overprotection and low care were extracted from the PBI.

Consistent with Fairburn *et al.* (1997), this study examined seven *a priori* risk domains: subject's mental health, subject's physical health, other environmental experiences, family weight and eating concerns, quality of parenting, parental psychopathology, and childhood abuse (see Table 2). Within each of these risk domains, multiple items were examined. For the purpose of data reduction, 22 risk factor composite scales were constructed using factor analytic procedures for six of the *a priori* risk domains (see Striegel-Moore *et al.* 2005). These risk factor scales had low overlap and loaded onto one of the six *a priori* risk domains. The seventh risk domain of childhood abuse,

as described in a separate report (Striegel-Moore *et al.* 2002), contains two risk factor items, sexual abuse and physical abuse.

Assessment of antecedent life events

In addition to the assessment of risk factors that occurred at any time prior to the index age, the RFI identifies a subset of factors that represent exposure to life events in the 12 months immediately preceding the index age (e.g. major house move; see Table 4). Following the analytic approach described by Pike *et al.* (2006), two composite variables were computed based on content for data reduction purposes: 'major stress from school, work or other source' (from two items) and 'concerns about safety' (from four items), resulting in a total of 13 items. Initially rated on a four-point scale, items were dichotomized for the statistical analyses: 0=no event occurred (initially coded 0, 1, 2 or 3) *versus* 1=definitely occurred (initially coded 4).

Data analysis

Because each control subject was 'yoked' to a specific AN case, the three groups (AN, PC and NC) were compared using repeated measures analyses of variance (ANOVAs) for each risk factor scale. Planned contrasts were analyzed for all significant F ratios. The first contrast combined the AN and PC cases into a single group representing cases with an Axis I disorder and compared these to the NC group; a significant group difference here indicated general risk factors for a mental disorder (hypothesis: AN/PC >NC). The second contrast compared the AN group to the PC group to determine risk factors more severe in the development of AN compared to other

psychiatric disorders (hypothesis: AN>PC). For analysis of planned contrasts, testing directed hypotheses, one-tailed comparisons were performed (paired *t* tests).

For determination of the impact of cumulative risk on psychiatric status, repeated measures analyses and planned contrasts were conducted on mean scores for each risk factor domain. Stepwise logistic regression analyses were used to identify general risk factors predicting the development of any psychiatric disorder (combined AN/PC groups) *versus* no psychiatric disorder (NC group) and risk factors more significant in predicting AN *versus* other psychiatric disorders (PC group). All risk factor scales for which planned contrasts were statistically significant were entered into the regression analyses. Antecedent life events were analyzed using repeated measures ANOVAs on single antecedent life event items and, in order to determine the overall degree of exposure to significant life events, on a categorical variable of 0, 1, 2, and 3 or more events. Significance level for main statistical analyses was set at $\alpha < 0.05$ and for planned contrasts at $\alpha < 0.01$. Partial η^2, describing the proportion of total variability attributable to a factor, was displayed for estimation of effect sizes (partial η^2: small, 0.01; medium, 0.06; large, 0.14; see Cohen, 1988).

Results

Exposure to risk factors

Table 2 summarizes the results of comparisons of the AN group with the matched PC and NC groups on exposure to risk factors. Significant group differences were found on a number of individual risk factor scales from the domains of subject's mental health, quality of parenting, and parental psychopathology ($p < 0.05$). Large effect sizes were found for maternal problem parenting, paternal problem parenting, family discord, and negative affectivity (all partial $\eta^2 > 0.16$). Additionally, exposure to sexual and physical abuse differed significantly between groups (both $p < 0.05$).

Risk factors common to both the AN and PC groups were determined by planned contrasts comparing these two groups of women with a current psychiatric diagnosis (combined AN/PC group) with the NC group. These analyses found that negative affectivity, family discord, maternal and paternal problem parenting, parental mood and substance disorder and sexual and physical abuse were associated with a generally increased risk for current psychiatric disorder (all $p < 0.01$).

For determination of risk factors that are more severe in AN compared to other psychiatric disorders,

planned contrasts revealed significantly higher negative affectivity, perfectionism, family discord, and higher parental demands in the AN group than the PC group (all $p < 0.01$).

Exposure to risk factor domains

For the analysis of cumulative risk within risk factor domains, repeated measures analyses revealed significant group differences and large effect sizes on subject's mental health, quality of parenting, and childhood abuse (all $p \leqslant 0.001$; $0.15 \leqslant \eta^2 \leqslant 0.30$; see Table 2). Concerning risk factors common to both AN and other psychiatric disorders, both psychiatric groups reported greater problems in the subject's mental health domain, the quality of parenting domain, and childhood abuse than the NC group (all $p < 0.01$). Concerning risk factors more severe in the AN group, planned contrasts revealed greater problems in the quality of parenting domain for the AN group than the PC group ($p < 0.01$).

Prediction of case status

For the prediction of any psychiatric disorder (combined AN/PC groups) *versus* no psychiatric disorder from risk factor scales, the final logistic regression equation retained four general risk factors (family discord, sexual abuse, negative affectivity, and maternal problem parenting) with positive β coefficients [χ^2 (4, $n = 150$) = 47.3, $p < 0.001$], accounting for a total of 50.0% of the variance (see Table 3). This analysis correctly classified 82.0% of the AN/PC group and 80.0% of the NC group.

For the prediction of AN *v.* PC on the basis of risk factor scales, the final logistic regression equation retained two variables (high parental demands and perfectionism), accounting for a total of 19.0% of the variance [χ^2(2, $n = 100$) = 15.1, $p < 0.001$] and correctly classifying 64.0% of the AN group and 68.0% of the PC group.

As noted earlier, the AN group reported a significantly higher rate of co-morbidity than the PC group ($p = 0.027$). As it is technically not possible to include co-morbidity rate as a covariate in the repeated measures analyses, we included it as a covariate in the logistic regression analysis for prediction of AN *v.* PC status. Although co-morbidity significantly predicted AN *v.* PC case status when entered in a first step ($B = 0.98$, S.E. = 0.45, Wald = 4.78, df = 1, $p = 0.029$, $R^2 = 0.07$), it became insignificant in the second step of the model ($p > 0.05$). Only high parental demands and perfectionism were retained as significant predictors in the final model, suggesting that they are stronger predictors of case status than co-morbidity.

Table 2. *Repeated measures analyses for risk factors items and domains by group, planned contrasts, means[a] and standard deviations*

Composite risk factor scales (no. of variables per scale)[b]	n	AN Mean	S.D.	PC Mean	S.D.	NC Mean	S.D.	F	df	p	Partial η^2	Contrasts[c] (paired t tests)
Subject's mental health domain	50	0.53	1.08	−0.07	1.28	−0.46	0.65	11.25	2, 87	<0.001	0.19	d
Conduct problems (2)	50	0.24	2.32	0.15	2.24	−0.39	0.00	1.59	2, 77	0.043	0.12	
Negative affectivity (5)	50	1.44	3.15	−0.20	3.21	−1.24	2.01	11.58	2, 95	<0.001	0.19	d,e
Substance abuse (2)	50	−0.02	1.23	0.22	2.10	−0.20	0.00	1.10	1, 69	0.319	0.02	
Perfectionism (2)	50	0.46	1.59	−0.42	1.39	−0.04	1.53	4.50	2, 98	0.013	0.08	d
Subject's physical health domain	50	0.34	2.04	−0.25	0.36	−0.09	1.48	2.15	2, 76	0.135	0.04	
Pregnancy history (3)	50	0.15	2.96	−0.28	0.00	0.14	2.96	0.51	2, 80	0.368	0.04	
Severe childhood obesity (3)	50	0.54	2.93	−0.22	0.72	−0.32	0.00	3.79	1, 54	0.053	0.07	
Other environmental experiences domain												
Disruptions and deprivation (3)	50	0.43	2.00	−0.40	1.58	−0.02	1.97	2.71	2, 98	0.072	0.05	
Family weight and eating concerns domain	50	0.11	1.16	0.06	1.14	−0.18	1.13	0.97	2, 97	0.381	0.02	
Family dieting (4)	50	0.27	3.16	0.12	3.04	−0.39	2.49	0.75	2, 80	0.449	0.02	
Maternal overweight (2)	50	−0.29	1.78	0.28	1.80	0.01	2.02	1.38	2, 98	0.255	0.03	
Family history of anorexia nervosa (3)	50	0.41	3.21	−0.29	1.66	−0.12	2.17	1.08	2, 89	0.339	0.02	
Paternal overweight (2)	50	−0.04	1.82	0.16	2.13	−0.12	1.70	0.29	2, 97	0.747	0.01	
Family history of bulimia nervosa (3)	50	0.10	2.41	0.12	2.71	−0.22	2.12	0.32	2, 96	0.723	0.01	
Family overeating (2)	50	0.23	1.93	−0.01	1.81	−0.22	1.65	0.71	2, 98	0.492	0.01	
Quality of parenting domain	50	0.73	1.45	−0.06	1.12	−0.63	1.09	14.66	2, 66	<0.001	0.30	d,e
Maternal problem parenting (3)	36	1.12	2.40	0.25	2.63	−1.06	2.09	6.61	2, 70	0.002	0.16	e
Family discord (4)	50	1.33	3.07	−0.11	2.24	−1.22	1.83	13.74	2, 90	<0.001	0.22	d,e
Paternal problem parenting (3)	36	0.84	2.58	0.42	2.48	−1.02	1.99	9.96	2, 68	<0.001	0.22	e
Separations from parent (2)	50	0.20	2.28	−0.26	1.67	0.06	1.92	0.77	2, 98	0.466	0.02	
Parental absence or death (2)	50	0.33	1.43	−0.31	1.06	−0.02	1.63	2.76	2, 94	0.070	0.05	
High parental demands (3)	50	0.86	2.10	−0.36	1.76	−0.50	1.74	6.73	2, 98	0.002	0.12	d
Parental psychopathology domain												
Parental mood and substance disorder	50	0.24	2.24	0.39	2.33	−0.63	1.69	3.52	2, 97	0.034	0.07	e
Childhood abuse	50	0.36	2.73	0.82	2.72	−1.17	1.51	8.35	2, 84	0.001	0.15	e
Sexual abuse	50	0.61	3.44	0.56	3.90	−1.17	1.43	4.48	2, 83	0.014	0.09	e
Physical abuse	50	0.10	3.35	1.07	3.92	−1.17	2.82	4.79	2, 98	0.010	0.09	e

AN, Anorexia nervosa; PC, psychiatric control group; NC, non-psychiatric control group; S.D., standard deviation.

[a] Group means represent the average sum of the standardized scores for the variables included in the factor. They can be interpreted as deviations from the mean.

[b] All variables reflect exposure before the participant's index age.

[c] One-tailed t tests, p<0.01.

[d] AN>PC.

[e] Combined AN/PC >NC.

Table 3. *Summary of the final logistic regression models for prediction of the AN v. PC case status and of the AN/PC v. NC case status*

Variable	B	S.E.	Wald	df	p	R^{2a}
Prediction of AN *v.* PC						
High parental demands	0.29	0.12	6.12	1	0.013	0.12
Perfectionism	0.33	0.15	5.13	1	0.024	0.19
Constant	−0.06	0.22				
Prediction of AN/PC *v.* NC						
Family discord	0.33	0.14	5.59	1	0.018	0.26
Sexual abuse	0.50	0.20	6.62	1	0.010	0.39
Negative affectivity	0.28	0.12	5.37	1	0.021	0.46
Maternal problem parenting	0.27	0.12	5.06	1	0.025	0.50
Constant	0.53	0.31				

AN, Anorexia nervosa; PC, psychiatric control group; NC, non-psychiatric control group; S.E., standard error.
[a] Cumulative R^2.

Antecedent life events

In the 12 months prior to the participant's index age, the combined AN/PC group revealed a significantly greater number of antecedent life events than the NC group, and in particular, greater exposure to physical abuse (both p's <0.01). In addition, the AN group reported significantly more frequent exposure to critical comments about shape, weight or eating than the PC group (p<0.01; see Table 4). Groups did not differ on any other antecedent life event (all p>0.05).

Comparison of AN-R with AN-BP

An exploratory comparison of exposure to risk factors items, domains and antecedent life events by AN subtype revealed a few significant differences. AN-BP participants showed significantly greater exposure to disruptions and deprivation than AN-R participants [$F(1, 48) = 9.98$, $p = 0.003$, $\eta^2 = 0.17$]. Medium effect sizes were found for greater maternal problem parenting and parental mood and substance disorder, and lower perfectionism in the AN-BP group than in the AN-R group (all $\eta^2 \geqslant 0.06$). During the 12 months prior to index age, the AN-BP group reported significantly greater exposure to sexual abuse and change in family structure, lower exposure to physical illness (df = 1, 48; $F = 5.14, 4.79$, and 5.74; all p<0.04; all $\eta^2 \geqslant 0.90$), and a tendency to lower exposure to major house move than the AN-R group ($\eta^2 = 0.06$).

Discussion

To our knowledge, this is the first age-matched, case-control study of risk factors for AN. Using standardized assessments, 50 women with AN were compared to 50 age-matched women with non-eating disorder DSM-IV diagnoses and 50 women without psychiatric diagnosis on their exposure to a wide range of potential risk factors. Building on earlier studies, this investigation used more rigorous methodology to examine comprehensively the role of physical and mental health, family and personal history of eating, weight and dieting concerns, parent–child relationships, parental psychopathology, and childhood abuse in the development of AN.

Exposure to risk factors and prediction of case status

The results of this study suggest several retrospective correlates for psychopathology in general and several that are more salient for AN. Negative affectivity, maternal and paternal parenting problems, family discord, parental mood and substance disorder, and physical and sexual abuse emerged as general risk correlates for psychiatric disorders. The factors that emerged as more severe risk correlates for AN as compared to other psychiatric disorders were negative affectivity, perfectionism, family discord, and high parental demands.

Consistent with the findings from other studies (Fairburn *et al.* 1999; Karwautz *et al.* 2001), the relative contribution of family history of dieting, weight problems or eating disorder was not significant when considered in comprehensive models. These data correspond to other studies suggesting that these family history correlates may be less salient for AN than BN (Fairburn *et al.* 1997; Stice & Agras, 1998; Field *et al.* 1999; Stice, 2001). However, it is important to note that these family history data rely solely on the report of the individual with AN. By contrast, studies of genetic liability suggest that such variables may be etiologically important (Mazzeo *et al.* 2006). Further studies comparing eating disorders directly, gathering data from other informants, and advances in genetics research will bring greater clarity.

The findings from this case-control study offer further support for the centrality of both negative affectivity and perfectionism in the etiology of AN. They are consistent with clinical presentation, theoretical writings (Vitousek & Hollon, 1990; Vitousek & Ewald, 1993; Bruch 2001) and previous empirical investigations (Bastiani *et al.* 1995; Srinivasagam *et al.* 1995; Fairburn *et al.* 1999; Halmi *et al.* 2000; Karwautz *et al.* 2001; Anderluh *et al.* 2003; Bulik *et al.* 2003). It is possible that a high degree of perfectionism

Table 4. *Life events occurring within the year before onset of disordered eating in women with anorexia nervosa and within the equivalent year in general psychiatric or non-psychiatric control women respectively*

Antecedent life events[a]	n	AN		PC		NC		F	df	p	Partial η^2	Contrasts[b] (paired t tests)
		Mean	S.D.	Mean	S.D.	Mean	S.D.					
Major house move	50	−0.02	0.99	−0.02	0.98	0.04	1.05	0.05	2, 98	0.947	0.00	
Significant episode of physical illness	50	−0.06	0.93	0.06	1.08	0.00	1.00	0.17	2, 98	0.843	0.00	
Pregnancy	50	−0.08	0.00	0.16	1.73	−0.08	0.00	1.00	1, 49	0.322	0.02	
Bereavement (close relative/ friend/partner)	50	−0.08	0.84	0.08	1.14	0.00	1.00	0.30	2, 98	0.740	0.01	
Major episode of illness in close relative/friend/partner	50	0.01	1.01	0.10	1.07	−0.11	0.93	0.50	2, 98	0.608	0.01	
Change in family structure (member leaving or joining)	50	0.23	1.21	0.04	1.05	−0.26	0.59	3.19	2, 89	0.050	0.06	
End of relationship with boyfriend/partner	50	0.27	1.22	−0.13	0.86	−0.14	0.85	3.37	2, 97	0.039	0.06	
Sexual abuse	50	−0.02	0.98	0.12	1.16	−0.10	0.85	0.58	2, 98	0.564	0.01	
Physical abuse	50	0.25	1.26	0.10	1.13	−0.35	0.00	5.35	2, 86	0.009	0.10	d
Major stress from school, work or other source	50	0.16	1.00	−0.01	1.01	−0.15	0.99	1.42	2, 98	0.246	0.03	
Critical comments about weight, shape or eating	50	0.38	1.07	−0.15	0.94	−0.23	0.89	5.86	2, 98	0.004	0.11	c
Safety concerns	50	0.08	1.12	0.00	1.01	−0.08	0.88	0.27	2, 98	0.763	0.01	
Anything else significant	50	0.21	1.14	−0.07	0.94	−0.13	0.89	1.51	2, 98	0.225	0.03	
0, 1, 2 or 3 or more life events	50	0.37	0.90	−0.01	0.97	−0.36	1.01	7.37	2, 98	0.001	0.13	d

AN, Anorexia nervosa; PC, psychiatric control group; NC, non-psychiatric control group; S.D., standard deviation.

[a] Standardized scores. All variables reflect exposure during the year prior to the participant's index age.

[b] One-tailed t tests, $p < 0.01$.

[c] AN > PC.

[d] Combined AN/PC > NC.

and negative affectivity are primary retrospective correlates for AN, whereas weight and shape issues may instead intensify in closer proximity to the emergence of the disorder.

Consistent with other studies, parental psychopathology and childhood physical and sexual abuse emerged as retrospective correlates common to the AN and PC groups (Pope & Hudson, 1992; Wonderlich & Mitchell, 1997). These data suggest that prevention interventions for children of parents with mental illness and prevention of abuse could contribute to reducing risk across a wide range of disorders that commonly afflict women.

The logistic regression model conducted to predict case status successfully classified the vast majority of the individuals with psychiatric disorders as compared to those with no psychiatric disorder and successfully classified the majority of AN and PC cases. More risk correlates common to both AN and PC were identified as compared to risk correlates that were more significant specifically for AN, offering support to the view that psychiatric disorders share a common base of risk factors. However, it is important to pursue identification of additional risk factors that might be especially linked to AN given that the identified risk factors accounted for a low to moderate degree of explained variance.

The differences identified in the exploratory comparison of the restricting and binge/purge subtype of AN suggest a pattern of greater disturbance among the binge/purge subtype. These findings are consistent with other reports of higher rates of impulse control problems for this subtype (Jacobi *et al.* 2004; Commission on Adolescent Eating Disorders, 2005). Additional studies are needed to further understand the differences between the two subtypes in terms of both risk factors and clinical features.

Antecedent life events

The findings regarding proximal triggers for AN are provocative and only partially support our hypotheses. It is noteworthy that critical comments about weight, shape or eating constitute the only variable that emerged as a specific proximal trigger for AN. This is especially notable against the longer-term picture, where family weight problems and eating disorders among first-degree relatives were not identified as retrospective correlates for AN. Taken together, these data suggest that weight and shape concerns contribute to AN in proximity to its actual onset and in the context of a longer-term backdrop of retrospective correlates that are unrelated to weight and shape. Our hypothesis that other interpersonal experiences are proximally associated with the specific development

of AN was, however, not supported. Physical abuse was identified as a proximal antecedent life event but it was associated with both the AN and PC groups. Nevertheless, cumulative exposure to antecedent life events was associated with the onset of AN. Thus, it appears that in the year preceding onset, critical comments regarding weight and shape are most clearly associated with increased risk, and interpersonal stresses are cumulative but variable, such that specific interpersonal stressors do not emerge as antecedents for AN.

Limitations

Several limitations of this study are noteworthy. The RFI sets an upper limit of 18 years for assessment of family and parenting variables. Because nine of the AN subjects had index ages greater than 18 years, the results may fail to capture some experiences of risk exposure. In addition, although procedures were followed to maximize accuracy of reporting, potential biases due to recall are intrinsic to retrospective case-control designs. The findings represent the subjective experience of respondents, which would not necessarily be consistent with the views of other family members or significant others, for example, when reporting family psychiatric history. Given the extraordinary undertaking of a longitudinal risk factor study, it would be of enormous benefit to gather multiple perspectives on putative risk factors.

Conclusions

The convergent findings from this study and those of Fairburn *et al.* (1999) and Karwautz *et al.* (2001) move the field closer to defining the factors that should be examined using longitudinal models in the next generation of studies on the etiology and prevention of AN. Defining high-risk groups based on the identified retrospective correlates from these and other studies will increase the focus and feasibility of longitudinal and prevention studies of AN. The assessment of perfectionism, negative affectivity, parenting problems, family discord, and childhood abuse should contribute to delineating high-risk target groups. Such initiatives are urgently needed to advance our understanding of the etiology of AN and improve the efficacy of prevention programs.

Note

Supplementary information accompanies this paper on the Journal's website (http://journals.cambridge. org).

Acknowledgments

Declaration of Interest

None.

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