

Risk Factors for Binge Eating Disorder

A Community-Based, Case-Control Study

Christopher G. Fairburn, DM, MPhil, FRCPsych; Helen A. Doll, MSc; Sarah L. Welch, DPhil, MRCPsych; Philippa J. Hay, DPhil, MD, FRANZP; Beverley A. Davies, BSc; Marianne E. O'Connor, BA

Background: Many risk factors have been implicated for eating disorders, although little is known about those for binge eating disorder.

Methods: A community-based, case-control design was used to compare 52 women with binge eating disorder, 104 without an eating disorder, 102 with other psychiatric disorders, and 102 with bulimia nervosa.

Results: The main risk factors identified from the comparison of subjects with binge eating disorder with healthy control subjects were certain adverse childhood experiences, parental depression, vulnerability to obesity, and repeated exposure to negative comments about shape, weight, and eating. Compared with the subjects with other psychiatric disorders, those with binge eating disorder

reported more childhood obesity and more exposure to negative comments about shape, weight, and eating. Certain childhood traits and pronounced vulnerability to obesity distinguished the subjects with bulimia nervosa from those with binge eating disorder.

Conclusions: Binge eating disorder appears to be associated with exposure to risk factors for psychiatric disorder and for obesity. When compared with the wide range of risk factors for bulimia nervosa, the risk factors for binge eating disorder are weaker and more circumscribed. Premorbid perfectionism, negative self-evaluation, and vulnerability to obesity appear especially to characterize those in whom bulimia nervosa subsequently develops.

Arch Gen Psychiatry. 1998;55:425-432

BINGE EATING disorder is a new diagnostic concept, which is included in the appendix of *DSM-IV*, reserved for categories meriting further study.¹ The cardinal feature is binge eating, but, in contrast to bulimia nervosa, the binge eating is not accompanied by "the regular use of inappropriate compensatory behaviors" such as self-induced vomiting and misuse of laxatives.^{1p5501} The disorder commonly co-occurs with obesity.²

Research on binge eating disorder is still at an early stage,³ and its relationship with other eating disorders (particularly bulimia nervosa) continues to be debated.⁴⁻⁸ Its clinical features are being delineated,^{3,9} and there have been a small number of controlled studies of its treatment.¹⁰⁻¹² There has been little research on its causes or development. Several studies have focused on the timing of its onset,¹³⁻¹⁷ and reports exist of a raised rate of lifetime and family psychiatric disorder in the families of these patients.^{7,18,19} No comprehensive risk factor studies have been conducted.

We herein report the findings of a community-based, case-control study of risk factors for binge eating disorder. It complements a parallel and integrated study of risk factors for bulimia nervosa.²⁰ Our study had the following 3 aims: to identify risk factors for the development of binge eating disorder, to determine which of these risk factors are especially common among subjects with binge eating disorder compared with subjects with other psychiatric disorders, and to compare the risk factors for binge eating disorder with those for bulimia nervosa. On the basis of the clinical characteristics of the disorder, we made the following 2 main predictions: first, there would be 2 broad classes of risk factor for binge eating disorder, those that increase the risk for psychiatric disorder in general and those that increase the risk for obesity (parental and childhood obesity); and, second, compared with subjects with bulimia nervosa, those with binge eating disorder would have less exposure to risk factors for psychiatric disorder in general but more exposure to risk factors for obesity.

From the Department of Psychiatry, Warneford Hospital, Oxford University, Oxford, England.

SUBJECTS AND METHODS

DESIGN

A case-control design was used with 3 related comparisons, corresponding to our 3 aims. Fifty-two women with binge eating disorder were compared with 104 without an eating disorder (healthy control subjects), 102 with other psychiatric disorders (general psychiatric control subjects), and 102 with bulimia nervosa. For the first comparison, both groups were individually matched within 1 year of age and within 3 parental social class bands²¹ (social classes I and II [high], III [middle], and IV and V [low]). Matching for current age reduced the risk for age-related recall bias, and matching for parental social class removed a potential confounding variable. To match the subjects for the time available for exposure, the healthy controls were questioned about their life until the age of onset of disturbed eating (index age) of their particular matched subject with binge eating disorder. The second and third comparisons were unmatched and used subjects who had been recruited for the study of bulimia nervosa.²⁰ These analyses were undertaken after adjusting for current age, parental social class, and index age.

Unless otherwise indicated, data are given as mean \pm SD.

RECRUITMENT

The recruitment and assessment strategy is described in detail in the article on risk factors for bulimia nervosa.²⁰ The

sampling frame was the patient registers of 23 general practices situated in urban and rural parts of Oxfordshire, England. All women aged from 16 to 35 years listed on these registers were sent 2 self-report questionnaires: the Eating Disorder Examination-Questionnaire (EDE-Q), a questionnaire designed to detect eating disorders,²²⁻²⁵ and the 30-item General Health Questionnaire (GHQ),²⁶ an instrument designed to detect psychiatric disorder. Subjects who did not reply were sent a second set of questionnaires. Those whose responses on the EDE-Q suggested that they might have binge eating disorder were invited for an interview to establish their true case status. Subjects who met diagnostic criteria underwent a further interview to identify their age at onset of the eating disorder (see below) and to assess their exposure to putative risk factors for eating disorders. The subjects with bulimia nervosa were recruited in an equivalent manner.²⁰

Potential healthy controls were identified from their responses on the EDE-Q, and potential general psychiatric controls, from their scores on the GHQ, with true case status being established by interview. The subjects had their height and weight measured, and informed consent was obtained.

Subjects with eating disorders had to meet strict operational definitions of *DSM-IV* binge eating disorder or bulimia nervosa based on ratings on the EDE interview.²⁷⁻²⁹ The healthy controls were required to have no current or past eating disorder, established by interview. There were no other exclusion criteria. The general psychiatric controls had to have a current Axis I *DSM-III-R* psychiatric disorder, confirmed by interview using the Structured

RESULTS

CHARACTERISTICS OF THE GROUPS

Subjects with binge eating disorder had a mean age of 24.5 ± 5.7 years and a parental social class distribution as follows: high (social classes I or II), 22 (43.1%); middle (III), 18 (35.3%); and low (IV or V), 11 (21.6%) (social class information was not available for 1 subject). Mean index age was 16.8 ± 4.6 years. Mean body mass index was 26.9 ± 5.5 , and 22.2% (10 of the 45 subjects who provided this information) had a body mass index of more than 30. Few subjects had ever used extreme methods of weight control (4 subjects [7.7%] had induced vomiting; 1 subject [1.9%] had misused laxatives), and none had a history of bulimia nervosa. None were currently in treatment.

Healthy controls were individually matched to the subjects with binge eating disorder in terms of age and parental social class, with the result that the distribution of these characteristics was very similar.

Subjects with bulimia nervosa had a mean age of 23.7 ± 4.9 years and a parental social class distribution (which was not significantly different from that of the subjects with binge eating disorder) as follows: high (social classes I or II), 47 (46.1%); middle (III), 46 (45.1%); and low (IV or V), 9 (8.8%). Mean body mass index was 24.3 ± 4.6 , and 12% (10 of the 84 subjects who provided this information) had a body mass index of more than 30. Mean

index age was 15.5 ± 3.9 years, and 89% (85 of the 95 subjects who provided this information) were not in treatment.

General psychiatric controls were individually matched to the subjects with bulimia nervosa in terms of age and parental social class. Their diagnoses fell into the following principal categories: major depressive disorder, 83 (81.4%); bipolar disorder, 1 (1.0%); and an anxiety disorder, 18 (17.6%). The overrepresentation of depressive disorder may have been the result of subjects with chronic anxiety disorder being missed by the GHQ.³⁷ Seventy (68.6%) were not receiving treatment.

COMPARISON WITH HEALTHY CONTROLS

Individual Risk Factors

Subjects with binge eating disorder reported greater levels of exposure to the following personal vulnerability factors: negative self-evaluation, parental depression (ever) (for both, $P < .001$), major depression ($P = .003$), marked conduct problems ($P = .002$), and deliberate self-harm ($P = .004$). With regard to the environmental factors, they reported greater levels of exposure to the following parental problems: parental criticism, high expectation, minimal affection (for all 3, $P < .001$); parental underinvolvement; and maternal low care and high overprotection (for both, $P = .002$). In addition, they were more likely to report sexual ($P = .001$) and repeated severe physical abuse ($P = .01$) and bullying ($P = .005$). They reported greater

Clinical Interview for *DSM-III-R*.^{30,31} Those with a current or past eating disorder were excluded.

RISK FACTOR ASSESSMENT

Exposure to putative risk factors for eating disorders was assessed by interviewing the subjects (mostly in their homes). The risk factor interview focused on the period before onset of the eating disorder, with onset (index age) being conservatively defined as the age at which the first significant and persistent behaviors characteristic of an eating disorder began (sustained dieting, regular episodes of overeating, self-induced vomiting, or laxative misuse) rather than the age at which the subject first met full diagnostic criteria for the disorder.²⁰ The presence of 5 risk factors was assessed before and after onset of the eating disorder, since they might have a hereditary influence (**Table 1**). The subjects also completed the Parental Bonding Instrument,³² a self-report measure of subjects' experience of both parents during the first 16 years of life.

The risk factor interview was investigator-based and used behavioral definitions of key concepts to minimize the problems associated with retrospective reporting.³³ Further details are provided in the article on bulimia nervosa²⁰ and a related article on sexual abuse.³⁴ (A copy of the interview schedule may be obtained by writing to C.G.F. A charge will be made to cover the cost of postage and photocopying.) A wide range of putative risk factors was assessed, and these were categorized a priori into domains and subdomains, each reflecting certain types of exposure (Table 1). Additional factors, which did not naturally

belong to any of the domains, were age at onset of menstruation, a history of pregnancy or abortion, and parity.

DATA ANALYSIS

The first comparison used conditional logistic regression analyses appropriate for a case-control design with individual matching.^{35,36} The other 2 comparisons used logistic regression analyses appropriate for an unmatched case-control design.^{35,36} The relationships between individual putative risk factors and case status were first assessed univariately. Each risk factor was considered as a single indicator variable and coded 0 for "no" or 1 for "yes." Statistical significance was assessed using the χ^2 likelihood ratio statistic and was set at the 1% level ($P < .01$) to achieve levels of statistical power comparable with those in the bulimia nervosa case-control study.²⁰ An overall measure of exposure to risk in each subdomain and domain was obtained by summing the numbers of component factors to which each subject had been exposed. The resulting index scores were grouped into categories of as equal size as possible, with varying numbers for the subdomains and 4 for the domains. The relationships between case status and exposure in each subdomain and domain were examined univariately and, to assess the relative importance of different types of exposure, in multiple stepwise logistic regression analyses. To test for the significance of any apparent linear trend, the categorized scores (each assigned a value of average exposure) were entered in factored and unfactored forms. Statistical significance for the subdomain and domain analyses was set at the 5% level ($P < .05$).

exposure to just the following 2 dieting vulnerability factors: critical comments by family about shape, weight, or eating ($P < .001$) and teasing about shape, weight, eating, or appearance ($P = .01$). They were also more likely to have been pregnant before index age ($P < .001$).

Overall Level of Exposure to Each Subdomain

Subjects with binge eating disorder reported a greater level of exposure to the following 7 of the 12 subdomains: parental psychiatric disorder (ever), parental problems, dieting risk (for all 3, $P < .001$), childhood characteristics ($P = .002$), sexual and physical abuse ($P = .003$), obesity risk ($P = .007$), and disruptive events ($P = .04$). In each case, the more exposure, the greater the risk was for developing binge eating disorder. The following 3 subdomains entered the multiple regression model: parental problems ($\chi^2_2 = 26.8$; $P < .001$), parental psychiatric disorder (ever) ($\chi^2_2 = 9.6$; $P = .008$), and dieting risk ($\chi^2_2 = 6.8$; $P = .03$), 1 from each of the 3 domains.

Overall Level of Exposure to Each Domain

On univariate analysis, subjects with binge eating disorder had a significantly greater level of exposure to all 3 domains (**Table 2**). The odds ratios increased linearly from those subjects exposed to the smallest number of factors to those exposed to the largest. Two domains entered the multiple regression model (**Table 3**). Exposure in the en-

vironmental domain was entered first ($\chi^2_3 = 33.6$; $P < .001$), followed by exposure in the dieting vulnerability domain ($\chi^2_3 = 10.1$; $P = .02$). There was no significant statistical interaction between the domains, suggesting that the effect of exposure in any 1 domain did not depend on the degree of exposure in the other and that the effect of combined exposures was additive.

COMPARISON WITH THE GENERAL PSYCHIATRIC CONTROLS

Individual Risk Factors

Subjects with binge eating disorder had a greater level of exposure to 1 factor from the personal vulnerability and environmental domains, low parental contact ($P < .001$), and 2 of the dieting vulnerability factors: critical comments by family about shape, weight, or eating and childhood obesity (for both, $P = .006$).

Overall Level of Exposure to Each Subdomain

Both groups did not differ significantly in their exposure to any of the personal vulnerability or environmental subdomains. Subjects with binge eating disorder, however, had been exposed to a significantly greater extent to all 3 dieting vulnerability subdomains. Only obesity risk, however, entered the multiple regression model ($\chi^2_1 = 8.1$; $P = .004$).

Table 1. Distribution of Putative Risk Factors in the 4 Subject Groups and the Results of Univariate Regression Analyses*

Risk Factors by Subdomains	Subjects, No. (%)				Results of Univariate Regression Analyses†					
					Healthy Controls		General Psychiatric Controls		Subjects With Bulimia Nervosa	
	With Binge Eating Disorder	Healthy Controls	General Psychiatric Controls	With Bulimia Nervosa	Odds Ratio (95% CI)	P	Odds Ratio (95% CI)	P	Odds Ratio (95% CI)	P
	Personal Vulnerability Domain									
Childhood characteristics										
Negative self-evaluation	18 (34.6)	11 (10.6)	31 (30.4)	55 (53.9)	4.4 (1.8-10.6)	<.001	1.0 (0.5-2.1)	.99	0.4 (0.2-0.9)	.02
Shyness	14 (26.9)	20 (19.2)	45 (44.1)	41 (40.2)	1.5 (0.7-3.4)	.28	0.4 (0.2-0.97)	.04	0.6 (0.3-1.2)	.13
Perfectionism	12 (23.1)	19 (18.3)	26 (25.5)	42 (41.2)	1.4 (0.6-3.3)	.46	1.1 (0.5-2.4)	.89	0.5 (0.2-1.1)	.06
Extreme compliance	8 (15.4)	14 (13.5)	27 (26.5)	29 (28.4)	1.2 (0.5-3.1)	.74	0.6 (0.2-1.6)	.29	0.6 (0.2-1.4)	.20
No close friends	7 (13.5)	4 (3.8)	10 (9.8)	18 (17.6)	3.5 (1.0-12.0)	.04	1.5 (0.5-4.3)	.47	0.8 (0.3-2.2)	.66
School absence through anxiety	3 (5.8)	1 (1.0)	4 (3.9)	8 (7.8)	5.2 (0.5-50.4)	.12	1.4 (0.3-6.9)	.66	0.7 (0.2-2.9)	.64
Premorbid psychiatric disorder										
Major depression	10 (19.2)	6 (5.8)	17 (16.7)	30 (29.4)	7.6 (1.6-35.7)	.003	1.0 (0.4-2.5)	.94	0.5 (0.2-1.2)	.12
Drug abuse	4 (7.7)	3 (2.9)	3 (2.9)	3 (2.9)	2.7 (0.6-11.9)	.20	2.3 (0.5-10.8)	.30	2.8 (0.6-13.6)	.21
Alcohol abuse	6 (11.5)	14 (13.5)	14 (13.7)	13 (12.7)	0.8 (0.3-2.4)	.72	0.5 (0.2-1.6)	.22	0.4 (0.1-1.5)	.18
Behavioral problems										
Marked conduct problems	8 (15.4)	2 (1.9)	7 (6.9)	16 (15.7)	8.0 (1.7-37.7)	.002	2.7 (0.8-8.7)	.10	1.1 (0.4-2.8)	.90
School absence (truancy)	4 (7.7)	7 (6.7)	7 (6.9)	16 (15.7)	1.1 (0.3-3.9)	.83	1.0 (0.3-3.6)	.99	0.4 (0.1-1.3)	.09
Deliberate self-harm	6 (11.5)	1 (1.0)	9 (8.8)	13 (12.7)	12.0 (1.5-9.7)	.004	1.1 (0.3-3.3)	.91	0.7 (0.2-2.1)	.54
Parental psychiatric disorder (ever)										
Depression‡	17 (32.7)	9 (8.6)	20 (19.6)	23 (22.5)	5.0 (2.0-12.8)	<.001	1.7 (0.8-3.8)	.19	1.5 (0.7-3.2)	.34
Alcoholism‡	6 (11.5)	5 (4.8)	7 (6.9)	24 (23.5)	2.4 (0.7-7.9)	.15	2.3 (0.6-9.0)	.24	0.3 (0.1-0.9)	.03
Drug abuse‡	1 (1.9)	0 (0)	3 (2.9)	9 (8.8)	... (2.3-142.1)	...	0.9 (0.1-11.6)	.92	0.2 (0.03-1.8)	.10
Environmental Domain										
Parental problems										
Low parental contact	20 (38.5)	21 (20.2)	14 (13.7)	34 (33.3)	2.8 (1.2-6.3)	.01	6.0 (2.4-15.0)	<.001	1.3 (0.6-2.8)	.47
Separation from parents	2 (3.8)	5 (4.8)	10 (9.8)	13 (12.7)	1.3 (0.2-8.0)	.76	0.5 (0.1-2.4)	.33	0.3 (0.1-1.2)	.05
Parental problems										
Arguments	24 (46.2)	27 (26.0)	36 (35.3)	58 (56.9)	2.3 (1.1-4.4)	.02	1.4 (0.7-2.8)	.37	0.6 (0.3-1.2)	.15
Criticism	11 (21.2)	4 (3.8)	17 (16.7)	34 (33.3)	6.9 (1.9-24.8)	<.001	1.2 (0.5-2.9)	.70	0.5 (0.2-1.1)	.09
High expectations	20 (38.5)	16 (15.4)	25 (24.5)	51 (50.0)	5.3 (1.9-14.6)	<.001	2.7 (1.2-6.2)	.01	0.7 (0.4-1.5)	.39
Overinvolvement	9 (17.3)	6 (5.8)	7 (6.9)	15 (14.7)	3.8 (1.2-12.7)	.02	2.0 (0.7-6.2)	.21	1.1 (0.4-2.8)	.87
Underinvolvement	30 (57.7)	32 (30.8)	43 (42.2)	59 (57.8)	2.8 (1.4-5.5)	.002	1.6 (0.8-3.3)	.18	0.8 (0.4-1.7)	.57
Minimal affection	20 (38.5)	8 (7.7)	28 (27.4)	41 (40.2)	10.9 (3.2-37.2)	<.001	1.4 (0.7-3.0)	.33	0.9 (0.4-1.8)	.76
Parental Bonding Instrument (low care and high overprotection)										
Maternal	21 (40.4)	20 (19.2)	39 (38.2)	55 (53.9)	4.0 (1.6-10.4)	.002	1.8 (0.8-4.2)	.16	0.9 (0.4-2.1)	.80
Paternal	16 (30.8)	25 (24.0)	41 (40.2)	44 (43.1)	3.2 (1.2-8.4)	.02	0.8 (0.3-2.0)	.68	1.0 (0.4-2.4)	.96
Disruptive events										
Parental death	4 (7.7)	4 (3.8)	5 (4.9)	2 (2.0)	2.3 (0.5-10.4)	.30	1.4 (0.3-5.7)	.67	3.3 (0.5-20.5)	.18
Change of parent figure	10 (19.2)	14 (13.5)	20 (19.6)	18 (17.6)	1.6 (0.6-4.2)	.97	0.8 (0.3-2.0)	.66	0.9 (0.4-2.4)	.90
Parental chronic illness	17 (32.7)	16 (15.4)	36 (35.3)	28 (27.4)	2.8 (1.2-6.4)	.01	1.0 (0.5-2.2)	.93	1.3 (0.6-2.8)	.46
Frequent house moves	10 (19.2)	18 (17.3)	19 (18.6)	22 (21.6)	1.2 (0.5-2.8)	.76	1.0 (0.4-2.5)	.95	0.8 (0.3-2.0)	.61
Severe personal health problems	8 (15.4)	9 (8.6)	14 (13.7)	12 (11.8)	2.0 (0.7-5.5)	.21	0.9 (0.3-2.7)	.01	1.5 (0.6-4.2)	.41
Parental psychiatric disorder										
Depression	8 (15.4)	6 (5.8)	9 (8.8)	19 (18.6)	2.7 (0.9-7.7)	.07	1.8 (0.6-5.3)	.29	0.8 (0.3-2.0)	.60
Alcoholism	4 (7.7)	5 (4.8)	6 (5.8)	20 (19.6)	1.6 (0.4-6.0)	.49	2.0 (0.5-8.7)	.35	0.3 (0.1-0.96)	.03
Other drug abuse	0 (0)	0 (0)	2 (1.9)	9 (8.8)

Overall Level of Exposure to Each Domain

The two groups only differed significantly with respect to the dieting vulnerability domain ($\chi^2_3 = 8.74$; $P = .03$), with binge eating disorder 3 times as likely to develop in those subjects who had been exposed to 3 or more factors as those with 2 or fewer exposures (Tables 2 and 3).

COMPARISON WITH SUBJECTS WITH BULIMIA NERVOSA

Individual Risk Factors

There were no statistically significant differences between the 2 groups with respect to individual risk factors.

Table 1. Distribution of Putative Risk Factors in the 4 Subject Groups and the Results of Univariate Regression Analyses* (cont)

Risk Factors by Subdomains	Subjects, No. (%)				Results of Univariate Regression Analyses†					
	With Binge Eating Disorder	Healthy Controls	General Psychiatric Controls	With Bulimia Nervosa	Healthy Controls		General Psychiatric Controls		Subjects With Bulimia Nervosa	
					Odds Ratio (95% CI)	P	Odds Ratio (95% CI)	P	Odds Ratio (95% CI)	P
Environmental Domain										
Teasing and bullying										
Teasing (not shape, weight, eating, or appearance)	6 (11.5)	18 (17.3)	12 (11.8)	15 (14.7)	0.5 (0.2-1.6)	.26	1.0 (0.3-3.1)	.99	0.7 (0.2-2.2)	.58
Bullying	9 (17.3)	4 (3.8)	13 (12.7)	11 (10.8)	5.5 (1.5-20.7)	.005	1.5 (0.6-4.1)	.40	1.7 (0.6-4.6)	.30
Sexual and physical abuse										
Sexual abuse	15 (28.8)	11 (10.6)	26 (25.5)	36 (35.3)	5.7 (1.8-17.6)	.001	1.1 (0.5-2.3)	.90	0.6 (0.3-1.4)	.23
Repeated severe sexual abuse	2 (3.8)	0 (0)	3 (2.9)	9 (8.8)	1.4 (0.2-8.7)	.74	0.4 (0.1-2.2)	.27
Physical abuse	11 (21.2)	10 (9.6)	30 (29.4)	33 (32.4)	2.3 (0.9-5.6)	.07	0.5 (0.2-1.2)	.10	0.5 (0.2-1.1)	.07
Repeated severe physical abuse	5 (9.6)	1 (1.0)	10 (9.8)	8 (7.8)	10.0 (1.2-85.6)	.01	0.8 (0.3-2.6)	.72	1.0 (0.3-3.6)	.98
Repeated severe physical or severe sexual abuse	6 (11.5)	1 (1.0)	12 (11.8)	12 (11.8)	12.0 (1.4-99.7)	.004	0.8 (0.3-2.5)	.73	0.9 (0.3-2.6)	.80
Dieting Vulnerability Domain										
Dieting risk										
Family member dieting										
For any reason	27 (51.9)	46 (44.2)	44 (43.1)	64 (62.7)	1.4 (0.7-2.7)	.36	1.4 (0.7-2.9)	.32	0.8 (0.4-1.6)	.54
For shape or weight	23 (44.2)	36 (34.6)	37 (36.3)	50 (49.0)	1.5 (0.8-2.8)	.26	1.3 (0.7-2.7)	.42	1.0 (0.5-2.1)	.95
Critical comments by family about shape, weight, or eating	28 (53.8)	26 (25.0)	32 (31.4)	66 (64.7)	3.7 (1.7-7.8)	<.001	2.7 (1.3-5.4)	.006	0.7 (0.3-1.4)	.31
Repeated comments by others about shape or weight	17 (32.7)	17 (16.3)	23 (22.5)	34 (33.3)	2.3 (1.1-4.9)	.03	1.5 (0.7-3.3)	.32	1.1 (0.5-2.2)	.90
Teasing about shape, weight, eating, or appearance	24 (46.2)	26 (25.0)	43 (42.3)	50 (49.0)	2.4 (1.2-5.0)	.01	1.2 (0.6-2.5)	.57	1.0 (0.5-2.0)	.99
Parental history of anorexia nervosa or bulimia nervosa	2 (3.8)	0 (0)	1 (1.0)	5 (4.9)	4.3 (0.4-51.4)	.23	1.0 (0.2-5.5)	.99
Parental obesity	10 (19.2)	13 (12.5)	20 (19.6)	34 (33.3)	1.8 (0.7-4.8)	.22	1.0 (0.4-2.3)	.93	0.5 (0.2-1.0)	.06
Childhood obesity	16 (30.8)	20 (19.2)	13 (12.7)	41 (40.2)	1.7 (0.8-3.6)	.13	3.3 (1.4-7.9)	.006	0.8 (0.4-1.7)	.49
Obesity risk										
Childhood obesity	16 (30.8)	20 (19.2)	13 (12.7)	41 (40.2)	1.7 (0.8-3.6)	.13	3.3 (1.4-7.9)	.006	0.8 (0.4-1.7)	.49
Any parental obesity‡	16 (30.8)	24 (23.1)	29 (28.4)	44 (43.1)	1.5 (0.7-3.4)	.29	1.1 (0.5-2.4)	.78	0.5 (0.3-1.1)	.09
Parental eating disorder										
Any parental history of anorexia nervosa or bulimia nervosa‡	4 (7.7)	0 (0)	2 (2.0)	6 (5.9)	6.3 (1.0-38.8)	.04	1.8 (0.5-6.8)	.40
Additional Risk Factors										
Age at menarche, y										
14-18	20 (38.5)	34 (32.7)	30 (29.4)	19 (18.6)	1.0	.39	1.0	.26	1.0	.02
9-13	31 (59.6)	69 (66.3)	71 (69.6)	82 (80.4)	0.7 (0.4-1.5)		0.7 (0.2-1.1)		0.4 (0.2-0.9)	
Pregnancy	14 (26.9)	7 (6.7)	7 (6.9)	13 (12.7)	11.3 (2.5-50.7)	<.001	5.4 (1.3-22.7)	.02	1.5 (0.5-4.8)	.46
Parity										
1	8 (15.4)	2 (1.9)	3 (2.9)	7 (6.9)	17.3 (2.1-142)	<.001	6.1 (1.2-30.7)	.06	1.7 (0.5-6.0)	.73
2-5	6 (11.5)	5 (4.8)	4 (3.9)	6 (5.9)	7.1 (1.0-48.3)		4.5 (0.7-29.3)		1.3 (0.3-6.4)	
Abortion	2 (3.8)	0 (0)	5 (4.8)	5 (4.8)	0.4 (0.1-2.7)	.31	0.6 (0.1-3.8)	.58

*Analyses are conditional for the matched comparison with healthy control subjects. The statistical significance of the exposure (likelihood ratio statistic, χ^2), odds ratio (adjusted in comparisons 2 and 3), and 95% confidence interval (CI) are given for each factor. All exposures, except those noted, predate the onset of the eating disorder. Ellipses indicate not applicable.

†Comparisons with subjects with binge eating disorder.

‡Before or after onset of the eating disorder.

Overall Level of Exposure to Each Subdomain

Subjects with binge eating disorder reported less exposure to 2 of the subdomains: childhood characteristics

($P = .001$) and obesity risk ($P = .02$). Childhood characteristics entered the multiple regression model first ($\chi^2_2 = 12.9$; $P = .002$), followed by obesity risk ($\chi^2_1 = 5.24$; $P = .02$).

Table 2. Factors in Each Domain to Which Subjects Were Exposed and the Results of Univariate Regression Analyses

Domain, No. of Factors	Subjects, No. (%)				Results of Univariate Regression Analyses*					
	With Binge Eating Disorder	Healthy Controls	General Psychiatric Controls	With Bulimia Nervosa	Healthy Controls		General Psychiatric Controls		Subjects With Bulimia Nervosa	
					Odds Ratio (95% CI)	P	Odds Ratio (95% CI)	P	Odds Ratio (95% CI)	P
Personal vulnerability										
0	17 (13.5)	37 (35.6)	19 (18.6)	5 (4.9)	1.00		1.00		1.00	
1	12 (23.1)	34 (32.7)	27 (26.5)	16 (15.7)	1.80 (0.6-5.5)	<.001	1.09 (0.3-3.5)	.14	0.67 (0.2-2.9)	.05
2	11 (21.2)	21 (20.2)	13 (12.7)	15 (14.7)	2.75 (0.9-8.8)		3.63‡ (1.0-13.1)		0.60 (0.1-2.6)	
3-9	22 (42.3)	12 (11.5)	43 (42.2)	66 (64.7)	7.25† (2.5-20.9)		1.22 (0.4-3.6)		0.26‡ (0.1-0.9)	
Environmental										
0-1	5 (9.6)	34 (32.7)	20 (19.6)	8 (7.8)	1.00		1.00		1.00	
2-3	10 (19.2)	40 (38.5)	21 (20.6)	8 (7.8)	1.99 (0.6-7.0)	<.001	1.46 (0.4-5.3)	.58	2.51 (0.5-11.6)	.14
4-5	12 (23.1)	18 (17.3)	24 (23.5)	25 (24.5)	5.62‡ (1.4-22.7)		1.66 (0.5-5.9)		0.84 (0.2-3.3)	
6-18	25 (48.1)	12 (11.5)	37 (36.3)	61 (59.8)	21.4† (5.1-89.4)		2.16 (0.7-6.9)		0.68 (0.2-2.4)	
Dieting vulnerability										
0	6 (11.5)	26 (25.0)	16 (15.7)	4 (3.9)	1.00		1.00		1.00	
1-2	19 (36.5)	54 (51.9)	58 (56.9)	36 (35.3)	1.25 (0.4-3.6)	<.001	1.03 (0.3-3.3)	.03	0.57 (0.1-2.5)	.63
3	12 (23.1)	12 (11.5)	13 (12.7)	20 (19.6)	3.98‡ (1.1-13.9)		3.04 (0.8-11.4)		0.64 (0.1-3.1)	
4-8	15 (28.8)	12 (11.5)	15 (14.7)	42 (41.2)	5.98§ (1.8-19.9)		3.02 (0.8-10.9)		0.41 (0.1-1.9)	

*Comparisons with subjects with binge eating disorder. CI indicates confidence interval. Statistical significance was assessed using the χ^2 likelihood ratio statistic.

†P<.001.

‡P<.05.

§P<.01.

Table 3. Results of Stepwise Regression Analyses*

Domain, No. of Factors	Healthy Controls†			General Psychiatric Controls†			Subjects With Bulimia Nervosa†		
	Odds Ratio (95% CI)	P		Odds Ratio (95% CI)	P		Odds Ratio (95% CI)	P	
Personal vulnerability									
0			1.00		
1							0.67 (0.2-2.9)		.05
2							0.60 (0.1-2.6)		
3-9							0.26‡ (0.1-0.9)		
Environmental									
0-1	1.00		<.001						
2-3	1.40 (0.4-5.2)			...					
4-5	5.20‡ (1.3-20.8)								
6-18	16.8§ (3.9-73.0)								
Dieting vulnerability									
0	1.00			1.00					
1-2	1.71 (0.5-5.9)	.02		1.03 (0.3-3.3)		.03			
3	4.36 (1.0-19.5)			3.04 (0.8-11.4)					
4-8	6.85‡ (1.6-29.9)			3.02 (0.8-10.9)					

*The significance of the exposure (likelihood ratio statistic, χ^2), odds ratios, and 95% confidence intervals (CI) are given for each domain that had an independent relationship with case status (listed in order of entry to the regression model). Ellipses indicate that the variable did not enter the regression model.

†Comparison with subjects with binge eating disorder.

‡P<.05.

§P<.001.

Overall Level of Exposure in Each Domain

On univariate analysis, the two groups differed with respect to just 1 of the 3 domains, personal vulnerability, the level of exposure being lower in those with binge eating disorder ($\chi^2_3 = 7.72$; $P = .05$) (Table 2). The odds ratios decreased in a linear fashion, from those subjects exposed to the smallest number of factors to those exposed to the greatest. On multiple regression analysis, after adjusting for exposure in the personal vulnerability do-

main, no other domain had an independent relationship with case status (Table 3), although the subdomain of obesity risk ($\chi^2_1 = 4.58$; $P = .03$) remained significantly associated with case status.

COMMENT

To our knowledge, this is the first study of risk factors for the development of binge eating disorder. Certain as-

pects of our methods are of note. First, recruitment was direct from the community, thereby avoiding the sampling bias that is likely to arise from focusing on subjects who are seeking treatment. Such bias applies in bulimia nervosa³⁸ and is likely to apply in binge eating disorder. Second, we assessed a large number of putative risk factors, with the result that we were able to assess their relative contributions and possible interactions. Third, we had 3 comparison groups. The use of healthy controls made it possible to identify which of the putative risk factors were risk factors for binge eating disorder; the general psychiatric controls made it possible to determine which risk factors were particularly common among subjects with binge eating disorder compared with those with other psychiatric disorders; and the comparison with subjects with bulimia nervosa meant that we were able to compare the risk factors for these 2 eating disorders.

The comparison between subjects with binge eating disorder and healthy controls suggested that only certain of the risk factors implicated for eating disorders are risk factors for binge eating disorder. The factors with a major effect were negative self-evaluation; parental depression (ever); adverse childhood experiences (sexual and physical abuse and a range of parental problems); repeated exposure to negative comments from family members about shape, weight, or eating; and pregnancy before onset. One difference of note emerged from the comparison with the general psychiatric controls. This concerned factors belonging to the dieting vulnerability domain, most especially childhood obesity and negative comments from the family about shape, weight, and eating, with exposure being higher among subjects with binge eating disorder. Taken together, these findings support the prediction that binge eating disorder would be associated with exposure to 2 broad classes of risk factor: those that increase the risk for psychiatric disorder in general and those that increase the risk for obesity. Similarly, the findings of our bulimia nervosa risk factor study implicated exposure to the combination of general psychiatric risk factors and risk factors for dieting.²⁰

The comparison between subjects with binge eating disorder and those with bulimia nervosa revealed no differences with respect to individual risk factors, although 2 findings of note emerged from the subdomain and domain analyses. The main finding concerned the personal vulnerability domain, and most especially the childhood characteristics subdomain, where the exposure rates were higher among subjects with bulimia nervosa. Obesity risk (childhood and parental obesity) was also more common among subjects with bulimia nervosa, but the difference was less marked.

The findings from the first and third of the case-control comparisons, taken in conjunction with those from our study of risk factors for bulimia nervosa,²⁰ support the prediction that, compared with subjects with bulimia nervosa, those with binge eating disorder would report less exposure to risk factors for psychiatric disorder in general. When subjects with binge eating disorder were compared with their matched healthy controls, far fewer differences in exposure emerged than in the equivalent comparison involving subjects with bulimia nervosa,²⁰ and

the strengths of the associations were in general weaker. This was true of risk factors for psychiatric disorder in general and dieting vulnerability factors. The findings of the direct (but statistically less powerful) comparison of both groups with eating disorders suggest that 2 classes of risk factor in particular differentiate the 2 disorders. The first consists of certain childhood traits, particularly negative self-evaluation, perfectionism, shyness, and extreme compliance, all of which appear to be especially common antecedents of bulimia nervosa. Whereas shyness and extreme compliance were also common antecedents of general psychiatric disorder, negative self-evaluation and perfectionism appeared to be especially characteristic of subjects in whom bulimia nervosa subsequently developed, which is in accord with cognitive accounts of the development of the disorder.^{39,40} The second distinctive risk factor is vulnerability to obesity, which, although increased in binge eating disorder, seems to be even more pronounced in bulimia nervosa. This finding is in contradistinction to our prediction, based on the observed clinical association between binge eating disorder and obesity,² that exposure to risk factors for obesity would be more marked in binge eating disorder. The strong association between bulimia nervosa and vulnerability to obesity may be disguised by the following 2 processes: the intensity of these subjects' efforts to avoid fatness, resulting in unremarkable body weight,⁴¹ and the influence of a sampling bias that results in subjects who are particularly vulnerable to obesity being underrepresented among those subjects who present for treatment.³⁸

Certain limitations of our study should be noted. First, we relied exclusively on information provided by the subjects. Other informants could not be used, since secrecy characterizes binge eating disorder and bulimia nervosa.^{42,43} Few of these community-based subjects would have given us permission to contact other informants. Second, it was not feasible for the risk factor interview to be conducted by assessors who were unaware of the subjects' case status, since this would have necessitated separate interviews with 2 different people, an arrangement that few subjects would have accepted. Third, the study was retrospective in nature and therefore vulnerable to the problems associated with retrospective reporting. In this context, it should be noted that the level of concern about the retrospective reports of subjects with psychiatric disorders is probably excessive; as Brewin et al^{33(p82)} note, "there is little reason to link psychiatric status with less reliable or less valid recall of early experiences." Furthermore, in our study, many such problems are likely to have been minimized by the use of an investigator-based assessment interview in which concepts were defined in behavioral terms. Nevertheless, a prospective study would have been preferable although extremely difficult, given the low incidence of eating disorders. The last point of note concerns the nature of the subjects with binge eating disorder who, although likely to be representative of many subjects in the community, differed in 2 main ways from those seen in clinics. First, they were younger, most patients with binge eating disorder being in their 40s,³ and second, fewer had comorbid obesity.³ Finally, like many other investigators, we focused on women, whereas it has been reported

that a substantial minority of those with binge eating disorder are men.^{44,45} Future studies will need to determine whether our findings can be generalized to men with the disorder and to those who present for treatment.

Accepted for publication November 18, 1997.

Funded by program grant 18157 from the Wellcome Trust; Wellcome Principal Research Fellowship 046386 (Dr Fairburn); grants 030583 and 13123 from the Wellcome Trust (Drs Doll and Welch), London England; a Nuffield Medical Fellowship, University of Oxford, Oxford, England (Dr Hay); and a Girdlers' Junior Research Fellowship, University of Oxford (Dr Hay).

We thank George Brown, PhD, Bedford College, London, England, and Emily Simonoff, MD, Institute of Psychiatry, London, for their advice on the design of the risk factor interview; David Jones, MD, Park Hospital, Oxford, for advice on the assessment of sexual abuse; the participating general practitioners; and Faith Barbour, Jenny Burton, Valerie Dunn, Jan Francis, and Sue Shaw for their work as interviewers.

Corresponding author: Christopher G. Fairburn, DM, MPhil, FRCPsych, Department of Psychiatry, Warneford Hospital, Oxford University, Oxford OX3 7JX, England.

REFERENCES

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*. Washington, DC: American Psychiatric Association; 1994.
2. Marcus MD. Binge eating and obesity. In: Brownell KD, Fairburn CG, eds. *Eating Disorders and Obesity: A Comprehensive Handbook*. New York, NY: Guilford Press; 1995:441-449.
3. Castonguay LG, Eldredge KL, Agras WS. Binge eating disorder: current state and future directions. *Clin Psychol Rev*. 1995;15:865-890.
4. Fairburn CG, Welch SL, Hay PJ. The classification of recurrent overeating: the "Binge Eating Disorder" proposal. *Int J Eating Disord*. 1993;13:155-159.
5. Spitzer RL, Stunkard AJ, Yanovski S, Marcus MD, Wadden T, Wing R, Mitchell J, Hasin D. Binge eating disorder should be included in *DSM-IV*: a reply to Fairburn et al's The classification of recurrent overeating: the "Binge Eating Disorder" proposal. *Int J Eating Disord*. 1993;13:161-169.
6. Fichter MM, Quadflieg N, Brandl B. Recurrent overeating: an empirical comparison of binge eating disorder, bulimia nervosa and obesity. *Int J Eating Disord*. 1993;14:1-16.
7. Hay PJ, Fairburn CG, Doll HA. The classification of bulimic eating disorders: a community-based cluster analysis study. *Psychol Med*. 1996;26:801-812.
8. Hay PJ, Fairburn CG. The validity of the *DSM-IV* scheme for classifying bulimic eating disorders. *Int J Eating Disord*. 1998;23:7-15.
9. Mitchell JE, Mussell MP. Comorbidity and binge eating disorder. *Addict Behav*. 1995;20:725-732.
10. Hudson JI, Carter WP, Pope HG. Antidepressant treatment of binge-eating disorder: research findings and clinical guidelines. *J Clin Psychiatry*. 1996;57:73-79.
11. Devlin MJ. Assessment and treatment of binge-eating disorder. *Psychiatr Clin North Am*. 1996;19:761-772.
12. Wilson GT, Fairburn CG. Treatment of eating disorders. In: Nathan PE, Gorman JM, eds. *A Guide to Treatments That Work*. New York, NY: Oxford University Press; 1998:501-530.
13. Wilson GT, Nonas CA, Rosenblum GD. Assessment of binge eating in obese patients. *Int J Eating Disord*. 1993;13:25-33.
14. Marcus MD, Moulton MM, Greeno CG. Binge eating onset in obese patients with binge eating disorder. *Addict Behav*. 1995;20:747-755.
15. Mussell MP, Mitchell JE, Weller CL, Raymond SJ, Crosby R. Onset of binge eating, dieting, obesity and mood disorders among subjects seeking treatment for binge eating disorder. *Int J Eating Disord*. 1995;17:395-401.
16. Mussell MP, Mitchell JE, de Zwaan M, Crosby RD, Seim HC, Crow SJ. Clinical characteristics associated with binge eating in obese females: a descriptive study. *Int J Obes*. 1996;20:324-331.
17. Spurrell EB, Wilfley D, Tanofsky MB, Brownell KD. Age of onset for binge eating: are there different pathways to binge eating? *Int J Eating Disord*. 1997;21:55-65.
18. Brody MJ, Walsh BT, Devlin MJ. Binge eating disorder: reliability and validity of a new diagnostic category. *J Consult Clin Psychol*. 1994;62:381-386.
19. Yanovski SZ, Nelson JE, Dubbert BK, Spitzer RL. Association of binge eating disorder and psychiatric comorbidity in obese subjects. *Am J Psychiatry*. 1993;150:1472-1479.
20. Fairburn CG, Welch SL, Doll HA, Davies BA, O'Connor ME. Risk factors for bulimia nervosa: a community-based case-control study. *Arch Gen Psychiatry*. 1997;54:509-517.
21. Office of Population Censuses and Surveys. *Classification of Occupations 1980*. London, England: Her Majesty's Stationery Office; 1980.
22. Fairburn CG, Beglin SJ. Assessment of eating disorders: interview or self-report questionnaire? *Int J Eating Disord*. 1994;16:363-370.
23. Beglin SJ, Fairburn CG. Evaluation of a new instrument for the detection of eating disorders in community samples. *Psychiatry Res*. 1992;44:191-201.
24. Black CMD, Wilson GT. Assessment of eating disorders: interview versus questionnaire. *Int J Eating Disord*. 1996;20:43-50.
25. Wilfley DE, Schwartz MB, Spurrell EB, Fairburn CG. Assessment of binge eating disorder. *Behav Res Ther*. 1997;35:1151-1159.
26. Goldberg D, Williams P. *A User's Guide to the General Health Questionnaire*. Windsor, Ontario: NFER Publishing Company; 1988.
27. Cooper Z, Fairburn CG. The Eating Disorder Examination: a semi-structured interview for the assessment of the specific psychopathology of eating disorders. *Int J Eating Disord*. 1987;6:1-8.
28. Cooper Z, Cooper PJ, Fairburn CG. The validity of the Eating Disorder Examination and its subscales. *Br J Psychiatry*. 1989;154:807-812.
29. Fairburn CG, Cooper Z. The Eating Disorder Examination (12th edition). In: Fairburn CG, Wilson GT, eds. *Binge Eating: Nature, Assessment and Treatment*. New York, NY: Guilford Press; 1993:317-360.
30. Spitzer RL, Williams JBW, Gibbon M, First MB. The Structured Clinical Interview for *DSM-III-R* (SCID), I: history, rationale and description. *Arch Gen Psychiatry*. 1992;49:624-629.
31. Williams JBW, Gibbon M, First MB, Spitzer RL, Davis M, Borus J, Howes MJ, Kane J, Pope HG, Rounsaville B, Wittchen HU. The Structured Clinical Interview for *DSM-III-R* (SCID), II: multisite test-retest reliability. *Arch Gen Psychiatry*. 1992;49:630-636.
32. Parker G, Tupling H, Brown LB. A parental bonding instrument. *Br J Med Psychol*. 1979;52:1-10.
33. Brewin CR, Andrews B, Gotlib IH. Psychopathology and early experience: a reappraisal of retrospective reports. *Psychol Bull*. 1993;113:82-98.
34. Welch SL, Fairburn CG. Sexual abuse and bulimia nervosa: three integrated case control comparisons. *Am J Psychiatry*. 1994;151:402-407.
35. Breslow NE, Day NE. *Statistical Methods in Cancer Research: The Analysis of Case-Control Studies*. Lyon, France: International Agency for Research on Cancer; 1980:1.
36. Statistics and Epidemiology Research Corporation. *Epidemiological, Graphics, Estimation, and Testing Package (EGRET)*, 1.02. 10th ed. Seattle, Wash: Statistics and Epidemiology Research Corporation; 1995.
37. Koeter M, van den Brink W, Ormel J. Chronic psychiatric complaints and the General Health Questionnaire. *Br J Psychiatry*. 1989;155:186-190.
38. Fairburn CG, Welch SL, Norman PA, O'Connor ME, Doll HA. Bias and bulimia nervosa: how typical are clinic cases? *Am J Psychiatry*. 1996;153:386-391.
39. Fairburn CG. Eating disorders. In: Clark DM, Fairburn CG, eds. *Science and Practice of Cognitive Behaviour Therapy*. New York, NY: Oxford University Press; 1997:209-241.
40. Vitousek KM. The current status of cognitive-behavioural models of anorexia nervosa and bulimia nervosa. In: Salkovskis P, ed. *Frontiers of Cognitive Therapy*. New York, NY: Guilford Press; 1996:383-418.
41. Fairburn CG, Cooper PJ. The clinical features of bulimia nervosa. *Br J Psychiatry*. 1984;144:238-246.
42. Fairburn CG, Cooper PJ. Self-induced vomiting and bulimia nervosa: an undetected problem. *BMJ*. 1982;284:1153-1155.
43. Herzog DB. Bulimia: the secretive syndrome. *Psychosomatics*. 1982;23:481-484.
44. Spitzer RL, Devlin M, Walsh BT, Hasin D, Wing R, Marcus M, Stunkard A, Wadden T, Yanovski S, Agras S, Mitchell J, Nonas C. Binge eating disorder: a multisite field trial of the diagnostic criteria. *Int J Eating Disord*. 1992;11:191-203.
45. Spitzer RL, Yanovski S, Wadden T, Wing R, Marcus MD, Stunkard A, Devlin M, Mitchell J, Hasin D, Horne RL. Binge eating disorder: its further validation in a multisite study. *Int J Eating Disord*. 1993;13:137-153.