BRITISH JOURNAL OF PSYCHIATRY (1998), 173, 75–79

# Genetic epidemiology of binging and vomiting

PATRICK F. SULLIVAN, CYNTHIA M. BULIK and KENNETH S. KENDLER

**Background** Bulimia nervosa is typically defined as the combination of the behaviours of binging and vomiting. We sought to clarify the relationship of these behaviours from a genetic epidemiological perspective.

**Method** Using data on the lifetime history of binging and vomiting from a personally interviewed population-based sample of female twins ($n$=1897), we applied bivariate twin modelling to estimate the sources of variation for these traits.

**Results** The association between having ever binged (23.6%) and having ever induced vomiting (4.8%) was very strong (odds ratio=8.78, $P \ll 0.0001$). The best-fitting model indicated that lifetime binging and vomiting were both heritable (46% and 72%) and influenced by individual–specific environmental factors (54% and 28%). The overlap between the genetic ($r_a$=0.74) and individual-specific environmental factors ($r_e$=0.48) for the two traits was substantial. No violations of the equal environment assumption were evident.

**Conclusions** Including binging and vomiting under the rubric of bulimia nervosa appears to be appropriate. Our data are consistent with the identification of binging and vomiting as complex traits resulting from the interplay of multiple genes and individual-specific environmental influences. In contrast to 'environmentalist' theories, our results suggest that genetic influences may be of particular relevance to the aetiology of binging and vomiting.

The key elements in recent definitions of bulimia nervosa are the presence of eating binges and 'compensatory' behaviours intended to counteract binging and to prevent weight gain. Although these criteria may be useful clinically, their application in studies of the aetiology of bulimic behaviour could be inappropriate. First, studying syndromal cases of bulimia nervosa hinders the capacity to investigate more fundamental aetiological factors. Second, typical definitions of bulimia nervosa implicitly link binging and vomiting behaviour and it is often assumed that binging leads to or causes vomiting. In the present study, we investigated the lifetime history of binging and vomiting in a birth-registry based sample of female twins in order to clarify interrelationship of these essential precursors of syndromic bulimia nervosa. We addressed four questions: (a) How strongly are the lifetime histories of binging and vomiting associated? (b) Using univariate twin models, what are the relative aetiologic contributions of genetic and environmental factors for binging and vomiting? (c) Using bivariate twin models, what is the overlap between the genetic and environmental risk factors for binging and vomiting? (d) Are these violations of the key assumption of the twin design that question the validity of these results?

## METHOD

### Subjects

Subjects were caucasian female twins drawn from the Virginia Twin Registry, a population-based longitudinal study. The registry was formed from a systematic review of all birth records in the Commonwealth of Virginia (USA) after 1918. Twins were eligible to participate if they were born between 1934 and 1971 and both members had previously responded to a postal questionnaire. The individual response rate was 64%, which certainly underestimates the cooperation rate as an unknown proportion of twins did not receive the questionnaire because of incorrect addresses. In the Wave 1 interview (1987–1989), 92% of the eligible individuals ($n$=2163) were assessed. There have been two subsequent waves of telephone interviews completed in 2001 (92.5%, Wave 2) and 1898 (87.7%, Wave 3) of the original sample. The data for this report are from Wave 3 (1992–1995) when the mean age of the sample was 35.1 years (s.d.=7.5, range 22–59). Zygosity was determined blindly by standard questions (Eaves *et al*, 1989), photographs, and, if necessary, by DNA (Spence *et al*, 1988). There were 850 twin pairs of known zygosity (497 monozygotic, 353 dizygotic). These studies were approved by the local ethics committee and informed consent was obtained from all participants.

### Measures and interviewers

Lifetime psychiatric illness was diagnosed with an adapted version of the Structured Clinical Interview for DSM–III–R (SCID; Spitzer, *et al*, 1992). The Wave 3 interview (described in detail elsewhere; Sullivan *et al*, 1998) included the SCID questions for bulimia nervosa and additional questions that gathered more detailed information about bulimic behaviour. All subjects were asked two questions that were the focus of this report: whether they had ever had eating binges ("during which you ate a lot of food in a short period of time") and whether they had ever made themselves vomit ("as a means of controlling your shape and weight"). All interviewers had at least a master's degree in social work or a bachelor's degree plus two years of social work or counselling experience. They received 40–80 hours of initial training plus periodic review sessions. Each member of a twin pair was interviewed by different interviewers.

### Statistical analyses

To quantify the sources of variation in liability to a lifetime history of binging and vomiting, we used Mx (Neale, 1997) to fit a series of nested univariate twin models directly to contingency table data for monozygotic and dizygotic twin pairs. In univariate twin modelling, the sources of variation in liability to developing a trait (e.g. ever binging) are broken down into three parts corresponding to additive genetic influences ($a^2$ or 'heritability'), shared

https://doi.org/10.1192/bjp.173.1.75 Published online by Cambridge University Press

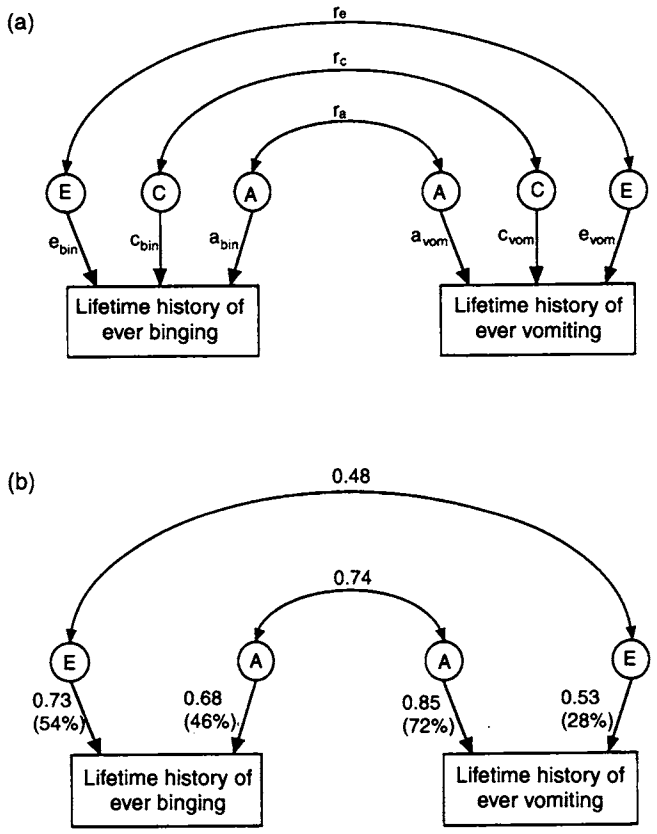


**Fig. 1** (a) Bivariate twin full model containing additive genetic, shared environmental and individual-specific environmental effects for a lifetime history of ever binging and ever inducing vomiting. (b) Bivariate twin best fit model containing additive genetic and individual-specific effects for a lifetime history of ever binging and ever inducing vomiting.

environmental influences common to both members of a twin pair ($c^2$), and environmental influences specific to an individual and thus unshared by members of a twin pair ($e^2$); Neale & Cardon, 1992). In bivariate twin modelling (Fig. 1a), the sources of variation are estimated for ever binging and ever vomiting yielding two separate sets of estimates of $a^2$, $c^2$ and $e^2$ (Neale & Cardon, 1992; Kendler, 1993). In addition, three distinct and independent correlations are estimated corresponding to the degree of overlap between $a^2$, $c^2$ and $e^2$ for the first trait and $a^2$, $c^2$ and $e^2$ for the second trait ($r_a$, $r_c$ and $r_e$ in Fig. 1). We used Mx to perform a bivariate cholesky decomposition (Neale & Cardon, 1992) with the input data in the form of tetrachoric correlation matrices and the associated asymptotic weighted least squares matrices (Jöreskog & Sörbom, 1993). Mx was also used to calculate the 95% confidence intervals for these parameters (Neale & Miller, 1997).

We used the $\chi^2$ difference test (Neale & Cardon, 1992) to compare the improvement in fit between models. Where there was equivocal improvement in fit, we also used Akiake's Information Criterion (AIC; Akiake, 1987). The lowest AIC provides the optimal combination of explanatory power and parsimony.

Finally, we evaluated the 'equal environment assumption', a central assumption of the twin design. If monozygotic twin pairs are more similar than dizygotic twin pairs in their exposure to an environmental aetiological factor, then estimates of $a^2$ will be artefactually inflated. We tested the equal environment assumption with six logistic regressions (SAS Institute Inc, 1996) for both ever binging and ever inducing vomiting. In each instance, the dependent variable was whether a twin pair was discordant or concordant for the trait under study. The independent variables were zygosity and, in turn, six measures of specified common environment plus the interaction between zygosity and the environmental measure. Regression coefficients for the environmental measure that are significantly different from zero reflect equal environment assumption violations. The assumption measures examined were: childhood treatment, co-socialisation, similitude, physical similarity, degree of adult contact, and parental approach to twin rearing. The first three variables are factor scores from 12 questions of environmental similarity asked of all twins (Kendler & Gardener, 1998). Childhood treatment ($n$=820 twin pairs) referred to how similarly a twin pair was treated as children by their parents (sharing the same room and classroom and being dressed alike). Co-socialisation ($n$=820 pairs) reflected the tendency of a twin pair to socialise together during childhood and adolescence (sharing playmates as children and as teenagers, sharing friends, going around with the same group, and going out together on dates) as well as an item of emotional closeness. Similitude ($n$=820 pairs) reflected the degree to which the twins themselves and those in their social environment (e.g., parents, teachers and friends) emphasised their similarity. Physical similarity ($n$=704 pairs) was based on blinded ratings of colour photographs of a twin pair (Hettema *et al*, 1995). Adult contact ($n$=820 pairs) reflected the frequency with which the twins see or talk to each other as adults. Finally, parental approach to twin rearing ($n$=733 pairs) reflects parental ratings of the degree to which the similarities of their twin offspring were emphasised. The number of twin pairs available for analysis varied because of missing data for some items.

## RESULTS

### Descriptive statistics

Of the 1897 personally interviewed female twins who participated in Wave 3, 23.6% reported having ever binged on food and 4.8% reported having induced vomiting to control body weight or shape. The association between these two behaviours was very strong (odds ratio=8.78, 95% CI 5.52–14.0, $\chi^2$=115.6, d.f.=1, $P \ll 0.0001$). Clearly, binging and vomiting are linked behaviours.

### Univariate twin modelling

To understand the causes of variation for ever binging and ever inducing vomiting, we fit a number of univariate twin models

https://doi.org/10.1192/bjp.173.1.75 Published online by Cambridge University Press

**Table 1** Univariate twin modelling for a lifetime history of ever binging or ever vomiting in 850 pairs of female twins

| Variable | Model | $\chi^2$ | d.f. | AIC | $a^2$ | $c^2$ | $e^2$ |
|---|---|---|---|---|---|---|---|
| Ever binged | ACE | 3.56 | 2 | −0.46 | | | |
| | AE[1] | 3.56 | 3 | −2.46 | 0.46 | – | 0.54 |
| | CE | 12.98 | 3 | 6.98 | | | |
| | E | 40.93 | 4 | 32.93 | | | |
| Ever vomited | ACE | 1.26 | 2 | −2.74 | | | |
| | AE[1] | 1.26 | 3 | −4.74 | 0.70 | – | 0.30 |
| | CE | 5.42 | 3 | −0.58 | | | |
| | E | 36.78 | 4 | 28.78 | | | |

1. Best-fitting model by AIC.
AIC, Akiake's Information Criterion (Akiake, 1987); ACE, additive genetic, shared and individual-specific environmental effects model; AE, additive genetic and individual-specific effects model; CE, shared and individual-specific environmental effects model; E, individual-specific environmental effects model; $a^2$, $c^2$ and $e^2$ refer to the proportion of variance in the best-fitting models due to additive genetic, shared environmental and individual-specific environmental effects.

(Table 1). For ever binging, the full ACE model fits well. The AE model had an identical goodness-of-fit $\chi^2$ as the ACE model and its lower AIC indicated an improvement in parsimony. The CE model had a considerably worse fit to the data ($P=0.002$) as did the E only model ($P<0.00005$). For the best-fitting AE model, the heritability ($a^2$) of ever binging was 46% (95% CI 32–58%) and individual-specific environmental effects ($e^2$) accounted for 54% (95% CI 42–68%) of the variance.

Because of interest in 'objective' binges (Cooper & Fairburn, 1987; Beglin & Fairburn, 1992), we made the definition of 'ever binging' more restrictive by requiring both loss of control during the binge and the consumption of an amount of food that others would regard as unusual (Sullivan *et al*, 1998). The lifetime prevalence of objective binging was 13.5% and the AE model provided the best fit to the data with estimates of $a^2$ (49%, 95% CI 32–63%) and $e^2$ (51%, 95% CI 37–68%) that were statistically indistinguishable from the less restrictive definition, although the confidence intervals were broader. In addition, it is possible to evaluate formally (using the multiple threshold model; Jöreskog & Sörbom, 1993) whether graded definitions of the binging trait (i.e. never binged, subjective binges and objective binges) differ in a quantitative or a qualitative manner. The 'quantitative' multiple threshold model fit well for both monozygotic ($P=0.78$) and dyzygotic twins ($P=0.38$) suggesting that subjective and objective binging lie along a graded spectrum of severity rather than resulting from different aetiological processes.

For ever having induced vomiting, the ACE model also fit well. The goodness-of-fit $\chi^2$ for the AE model was identical but its AIC indicated a more parsimonious explanation of the observed data. The CE ($P=0.04$) and E ($P<0.00005$) models fit significantly worse than the ACE model. For the best-fitting AE model, the heritability ($a^2$) of ever vomiting was 70% (95% CI 50–84%), common environmental effects ($c^2$) were negligible, and individual-specific environmental effects ($e^2$) accounted for 30% (95% CI 16–50%) of the variance.

## Bivariate twin modelling

The next step was to consider jointly the behaviours of ever having binged and ever having induced vomiting. We did this by fitting a series of bivariate twin models to the observed data (Table 2). The procedure was analogous to the univariate model-fitting in Table 1, but required more models given the greater number of parameters (Table 2). The full model fit well; of the nested sub-models, Model V provided the best fit to the data (four AIC units better than Model II) and included additive genetic and individual-specific environmental paths for both having ever binged and vomited and the correlations between these constructs ($r_a$ and $r_e$). Parameter estimates from Model V were similar to the univariate models: for ever binging, $a^2$ was estimated at 46% (95% CI 33–59%), $e^2$ at 54% (95% CI 40–69%); for ever vomiting, $a^2$ was estimated at 72% (95% CI 55–88%), $e^2$ at 28% (95% CI 11–46%); the correlation between additive genetic factors for ever binging and ever vomiting was estimated at 0.74 (95% CI 0.52–0.95); and the correlation between individual-specific environmental factors for ever binging and ever vomiting was estimated at 0.48 (95% CI 0.16–0.83; Fig. 1).

Consistent with the univariate modelling, a lifetime history of ever binging and ever vomiting are moderately to highly heritable behaviours. Moreover, the genes that influence the liability to these behaviours overlap to a considerable extent ($r_a=0.74$). Modestly overlapping environmental influences not shared by members of a twin pair ($r_e=0.48$) also influence the liability to these behaviours. Of note, the additive genetic and individual-specific environmental factors important in the aetiology of these two traits are neither identical nor entirely distinct (i.e. the 95% CI for both $r_a$ and $r_e$ did not contain unity or zero) and shared or common environmental factors could be ignored.

## Validity of the equal environment assumption

The observed heritabilities of ever binging and ever vomiting could be artefactually inflated by violations of the equal environment assumption. We fit 12 logistic regressions (six environmental measures for two dependent variables, see Methods) to test

**Table 2** Bivariate twin modelling for the lifetime history of ever binging or ever vomiting in a population-based sample of 1897 female twins

| Model | $\chi^2$ | d.f. | AIC |
|---|---|---|---|
| I: ACE-$r_a r_c r_e$ | 6.23 | 11 | −15.77 |
| II: ACE-$r_a r_e$ | 6.23 | 12 | −17.77 |
| III: ACE-$r_c r_e$ | 12.66 | 12 | −11.34 |
| IV: ACE-$r_e$ | 39.77 | 13 | 13.77 |
| V: AE-$r_a r_e$[1] | 6.23 | 14 | −21.77 |
| VI: AE-$r_a$ | 13.85 | 15 | −16.15 |
| VII: AE-$r_e$ | 39.77 | 15 | 9.77 |
| VIII: CE-$r_c r_e$ | 19.26 | 14 | −8.74 |
| IX: CE-$r_c$ | 35.33 | 15 | 5.33 |
| X: CE-$r_e$ | 53.48 | 15 | 23.48 |
| XI: E-$r_e$ | 119.87 | 16 | 87.87 |

1. Best-fitting model by AIC. The full "ACE-$r_a r_c r_e$" model (Model I) contains additive genetic, shared environmental, and individual-specific environmental effects for both binging and vomiting and their intercorrelations (see Fig. 1a). As other examples, Model II differs from Model I in that $r_c$ was set to zero and Model V differs from Model II in that the $c^2$ paths for both binging and vomiting were also set to zero.

https://doi.org/10.1192/bjp.173.1.75 Published online by Cambridge University Press

the assumption. For ever binging, the interaction between zygosity and each environmental measure was not significant ($P>0.10$ in all instances). For ever vomiting, the interaction between zygosity and the environmental measures was not significant in four instances and approached statistical significance for childhood treatment and similitude ($P=0.02$ for each; adjusting for multiple comparisons, the appropriate Type I error level is 0.004). These analyses did not reveal important violations of the equal environment assumption beyond chance expectations for these two dependent variables.

## DISCUSSION

We have attempted to provide an assessment of the validity of the DSM–III–R/DSM–IV (American Psychiatric Association, 1987, 1994) conceptualisation of bulimia nervosa as consisting of two phenomenologically and aetiologically linked behaviours. A lifetime history of ever binging and ever inducing vomiting (3.4%) were strongly associated (odds ratio=8.78, $P\ll0.0001$). This overlap was not complete as substantial proportions of the sample binged but did not vomit (20.3%) or had induced vomiting without binging (1.4%).

Consistent with prior results for a broad definition of bulimia (Kendler *et al*, 1991), the lifetime history of ever binging was fairly heritable with negligible shared environmental influences. The lifetime history of ever inducing vomiting was highly heritable – although the 95% confidence interval was broad (50–84%), the heritability of this behaviour may be comparable with that of schizophrenia (Kendler, 1983). Shared environmental influences for ever inducing vomiting were also negligible.

The central analyses of this report are in Table 2 and Fig. 1. Given that a lifetime history of both binging and inducing vomiting are substantially heritable, to what extent do the sources of liability to each behaviour overlap? In considering these behaviours jointly in bivariate twin models, a portrait consistent with the univariate models emerged; the behaviours are moderately to highly heritable with no impact of common environmental factors. Moreover, the genetic factors overlapped considerably but not completely ($r_a$=0.74, 95% CI 0.52–0.96) and environmental factors specific to an individual overlapped to a lesser degree ($r_e$=0.48, 95% CI 0.16–0.83).

### Validity of the equal environment assumption

Before commenting on the implications of these analyses, we must consider the extent to which the apparently significant heritabilities from these results may be artefactual. If the critical assumption was violated, then the heritabilities reported are suspect. Moreover, there was *a priori* reason to expect equal environment assumption violations for bulimia: in a prior report from the Virginia Twin Registry, Hettema *et al* (1995) found that a blinded rating of the physical similarity of a twin pair essentially negated the apparent heritability of bulimia. Consequently, it was important to test the equal environment assumption in a comprehensive fashion. We found no apparent violations of the assumption, even with the physical similarity rating used by Hettema *et al*, supporting the validity of these results.

### Implications

First, our results are broadly consistent with the DSM–III–R/DSM–IV (American Psychiatric Association, 1987, 1994) definition of bulimia nervosa that include binging and vomiting under the same rubric. These two behaviours are strongly associated and the rather high genetic correlation ($r_a$) between them suggests that they result from substantially overlapping genetic aetiologic factors. However, the overlap, particularly for environmental influences specific to an individual, was not perfect, indicating aetiologic as well as phenotypic heterogeneity of these behaviours.

Second, additive genetic factors are important in the aetiology of binging and, especially, of vomiting along with environmental factors specific to an individual. These estimates are inferred from the patterns of correlations in genetically identical monozygotic twins compared with dizygotic twins. Our study could not identify the precise genes involved nor could it illuminate the particular environmental factors; instead, we identified and quantified the latent factors underlying the observed data. Our data are consistent with the identification of binging and vomiting as complex traits, that is, behavioural phenomena that result from the interplay of multiple genes and environmental influences.

Third, the missing piece in the binging/vomiting puzzle was common or shared environmental effects. Given that 'familial' environmental effects figure prominently in many aetiological theories of bulimia (Johnson *et al*, 1984; Polivy & Herman, 1985; Schmidt *et al*, 1993; Striegel-Moore *et al*, 1986; Yates, 1989; Zerbe, 1996) and were particularly pronounced ($c^2$=40%) in this sample in a joint analysis of six psychiatric disorders including bulimia (Kendler *et al*, 1995) why was the influence of common environment negligible? There are several conceptually plausible reasons why. First, common environmental effects may, in fact, not be important in the liability to binging and vomiting. Second, twin studies are known to possess relatively low statistical power to detect common environmental effects in the presence of genetic effects (Neale *et al*, 1994). For a trait like ever binging (prevalence=23.6%, $a^2$=50%), the power to detect common environmental effects of 30% was only 0.44. For a rarer (prevalence=4.8%) but more heritable ($a^2$=70%) trait like ever vomiting, the power to detect common environmental effects of 30% was only 0.16. Thus, potentially important common environmental effects may have been present but were beneath the detection threshold. Third, familial factors may, in fact, be important in the aetiology of binging and vomiting but may exert their influence only in concert with an individual's genetic makeup. In this scenario, the independent main effect of familial environmental factors might be small but their effects *via* one of several types of gene–environment interaction (Kendler & Eaves, 1986) could be profound. Again, twin studies have low power to detect these effects.

Finally, perhaps the main implication of these results is to highlight the importance of familial genetic factors in the aetiology of behaviours that are precursors to bulimia nervosa. While many aetiological theories of bulimia at least briefly refer to biological or genetic factors, most emphasise common environmental factors (i.e. socio-cultural and family environmental indices; Johnson *et al*, 1984; Polivy & Herman, 1985; Schmidt *et al*, 1993; Striegel-Moore *et al*, 1986; Yates, 1989; Zerbe, 1996). In our sample, genetic influences may be of particular relevance to the aetiology of binging and vomiting.

https://doi.org/10.1192/bjp.173.1.75 Published online by Cambridge University Press

## REFERENCES


**Akiake, H. (1987)** Factor analysis and AIC. *Psychometrika*, **52**, 317–332.

**American Psychiatric Association (1987)** *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM–III–R). Washington, DC: APA.

**—— (1994)** *Diagnostic and Statistical Manual of Mental Disorders* (4th edn) (DSM–IV). Washington, DC: APA.

**Beglin, S. J. & Fairburn, C. G. (1992)** What is meant by the term "binge"? *American Journal of Psychiatry*, **149**, 123–124.

**Cooper, Z. & Fairburn, C. G. (1987)** The Eating Disorder Examination: a semi-structured interview for the assessment of the specific psychopathology of eating disorders. *International Journal of Eating Disorders*, **6**, 1–8.

**Eaves, L. J., Eysenck, H. G. & Martin, N. G. (1989)** *Genes, Culture, and Personality: An Empirical Approach*. London: Oxford University Press.

**Hettema, J. M., Neale, M. C. & Kendler, K. S. (1995)** Physical similarity and the equal-environment assumption in twin studies of psychiatric disorders. *Behavior Genetics*, **25**, 327–335.

**Johnson, C., Lewis, C. & Hagman, J. (1984)** The syndrome of bulimia. *Psychiatric Clinics of North America*, **7**, 247–273.

**Jöreskog, K. G. & Sörbom, D. (1993)** *PRELIS 2: User's Reference Guide*. Chicago, IL: Scientific Software International.

**Kendler, K. S. (1983)** Overview: a current perspective on twin studies of schizophrenia. *American Journal of Psychiatry*, **140**, 1413–1425.

**—— (1993)** Twin studies of psychiatric illness: current status and future directions. *Archives of General Psychiatry*, **50**, 905–915.

**—— & Eaves, L. J. (1986)** Models for the joint effect of genotype and environment on liability to psychiatric illness. *American Journal of Psychiatry*, **143**, 279–289.

**——, MacLean, C., Neale, M., *et al* (1991)** The genetic epidemiology of bulimia nervosa. *American Journal of Psychiatry*, **148**, 1627–1637.

**——, Walters, E. E., Neale, M. C., *et al* (1995)** The structure of the genetic and environmental risk factors for six major psychiatric disorders in women: phobia, generalized anxiety disorder, panic disorder, bulimia, major depression, and alcoholism. *Archives of General Psychiatry*, **52**, 374–383.

**—— & Gardener, C. O. (1998)** Twin studies of adult psychiatric and substance dependence disorders: are they biased by differences in the environmental experiences of mono- and dizygotic twins in childhood and adolescence? *Psychological Medicine*, in press.

**Neale, M. C. (1997)** *Mx: Statistical Modeling* (2nd edn). Richmond, VA: Medical College of Virginia, Department of Psychiatry.

**—— & Cardon, L. R. (1992)** *Methodology for the Study of Twins and Families*. Dordrecht: Kluwer Academic Publisher Group.

**——, Eaves, L. J. & Kendler, K. S. (1994)** The power of the classical twin study to resolve variation in threshold traits. *Behavior Genetics*, **24**, 239–258.

**—— & Miller, M. B. (1997)** The use of likelihood-based confidence intervals in genetic models. *Behavior Genetics*, **27**, 113–120.

**Polivy, J. & Herman, C. P. (1985)** Dieting and binging: a causal analysis. *American Psychologist*, **40**, 193–201.

**SAS Institute Inc (1996)** *SAS/STAT® Software: Changes and Enhancements through Release 6.11*. Cary, NC: SAS Institute Inc.

**Schmidt, U., Tiller, J. & Treasure, J. (1993)** Psychosocial factors in the origins of bulimia nervosa. *International Review of Psychiatry*, **5**, 51–60.

**Spence, J. E., Corey, L. A., Nance, W. E., *et al* (1988)** Molecular analysis of twin zygosity using VNTR DNA probes. *American Journal of Human Genetics*, **43**, 159.

**Spitzer, R. L., Williams, J. B. W., Gibbon, M., *et al* (1992)** The Structured Clinical Interview for DSM–III–R (SCID) I: history, rationale, and description. *Archives of General Psychiatry*, **49**, 624–629.

**Striegel-Moore, R. H., Silberstein, L. R. & Rodin, J. (1986)** Toward an understanding of risk factors for bulimia. *American Psychologist*, **41**, 246–263.

**Sullivan, P. F., Bulik, C. M. & Kendler, K. S. (1998)** The epidemiology and classification of bulimia nervosa. *Psychological Medicine*, in press.

**Yates, A. (1989)** Current perspectives on the eating disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, **28**, 813–828.

**Zerbe, K. J. (1996)** Feminist psychodynamic psychotherapy of eating disorders: theoretical integration informing clinical practice. *Psychiatric Clinics of North America*, **19**, 811–827.


### CLINICAL IMPLICATIONS

- Genetic influences may be important in the aetiology of binging and vomiting.
- Socio-cultural and environmental influences may be less important than previously thought.

### LIMITATIONS

- We considered only the vomiting form of purgation. Our findings may not apply to other forms of compensatory behaviours (laxative misuse, fasting or excessive exercise).
- Although our six measures of environmental similarity provided a reasonably comprehensive screen for equal environment assumption violations, we may not have detected violations of the assumption at other developmental periods important in the aetiology of binging or vomiting.
- Our results are constrained by the limited test–retest reliability of these behaviours: heritability is perforce limited by reliability. When the modest reliability of lifetime binging ($\kappa$=0.34) is taken into account, the heritability of ever binging (82%) was substantially higher than that reported here.


PATRICK F. SULLIVAN, FRANZCP, CYNTHIA M. BULIK, PhD, KENNETH S. KENDLER, MD, Virginia Institute for Psychiatric and Behavioral Genetics, Department of Psychiatry, Virginia Commonwealth University

Correspondence: Dr Sullivan, Department of Psychiatry, Virginia Institute for Psychiatric and Behavioral Genetics, PO Box 980126, Richmond, VA, 23298-0126, USA. Tel: 804 828 8129; Fax: 804 828 1471


(First received 18 July 1997, final revision 16 February 1998, accepted 16 February 1998)

https://doi.org/10.1192/bjp.173.1.75 Published online by Cambridge University Press