Genetic and Environmental Contributions to Obesity and Binge Eating

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Accepted 6 July 2002

Abstract: **Objective:** *Binge eating is present in a substantial proportion of obese individuals. A tendency toward obesity has been identified as a risk factor for eating disorders such as bulimia nervosa and binge eating disorder. The purpose of this article was to determine the extent of overlap between genetic and environmental factors that contribute to the liability to obesity and binge eating.* **Research Method:** *In a population-based sample of 2163 female twins, we conducted bivariate twin modeling to explore the relation between the genetic and environmental risk factors for obesity and binge eating.* **Results:** *Bivariate twin modeling revealed substantial heritability for obesity (0.86: 95% CI, 0.77–0.94), moderate heritability for binge eating (0.49: 95% CI, 0.38–0.61), and a modest genetic correlation of +.34 (95% CI, 0.19–0.50) between the two traits.* **Conclusions:** *Both binge eating and obesity are heritable conditions, and there seems to be only modest overlap in the genetic risk factors that increase liability to each condition.* © *2003 by Wiley Periodicals, Inc.* Int J Eat Disord 33: 293–298, 2003.

Key words: *obesity; binge eating; twin studies; genetic*

INTRODUCTION

Studies of treatment-seeking populations indicate that 23 to 46% of obese individuals report the symptom of binge eating (Gormally, Black, Daston, & Rardin, 1982; Marcus, Wing, & Lamparski, 1985; Spitzer, Devlin, Walsh, Hasin, Wing, Marcus, Stunkand, Wadden, Yanovski, Agras, Mitchell, & Nonas, 1992; Spitzer, Yanovski, Wadden, Wing, Marcus, Stunkard, Devlin, Mitchell, Hasin, & Horne, 1993). Binge eating and binge-eating disorder (BED) in the context of obesity are relatively equally distributed across gender in treatment-seeking populations (Spitzer et al., 1992; Spitzer et al., 1993) and may be more

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Published online in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/eat.10140

common in individuals from lower socioeconomic classes (Langer, Warheit, & Zimmerman, 1992; Warheit, Langer, Zimmerman, & Biafora, 1993). Community-based risk factor studies suggest that individuals with BED display greater vulnerability to obesity than healthy controls and that a history of childhood obesity is more common in individuals with BED than individuals with bulimia nervosa (Fairburn, Doll, Welch, Hay, Davies, & O'Connor, 1998).

Given the frequently noted associations between binge eating and obesity, the goal of this study was to explore the relation between genetic and environmental risk factors for obesity and binge eating using bivariate twin modeling.

METHOD

Subjects

The data for this report are from a population-based longitudinal study of Caucasian female twins drawn from the Virginia Twin Registry, which 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 mailed questionnaire (individual response rate was conservatively estimated to be 64%). Data for this study are from the first and third interview waves. In wave 1 (1987–1989), we assessed 92% of the eligible individuals ($N =$ 2,163), 90% face-to-face and the remainder by telephone. The average age of the twins was 30.1 years (*SD*, 7.6; range, 17–55 years). The wave 3 interview (1992–1995) occurred when the age of the twins averaged 35.1 years (*SD*, 7.5; range, 22–59 years)—an average of 5.1 years (*SD*, 0.4) after wave 1. Both members of 854 twin pairs (497 monozygotic [MZ] pairs, 354 dizygotic [DZ] pairs, and three pairs of unknown zygosity) were assessed at both wave 1 and wave 3. Interviews were conducted blind to information about the cotwin and to data collected at prior interviews. Written informed consent was obtained before face-to-face interviews and verbal assent before telephone interviews.

Measures and Interviewers

Body mass index (kg/m^2) was determined based on self-reported heights and weights from the wave 1 interview. We defined ''obesity'' using the conventional body mass index (BMI) cutoff of $30.0\,kg/m^2$. Given that we have noted poor reliability of the diagnosis of bulimia nervosa and reporting of the symptom of binge eating, we incorporated information from both the wave 1 and wave 3 interview into our definition of lifetime history of binge eating to improve accuracy and reliability. A lifetime history of binge eating was determined by a positive response to the screening question at either wave 1 or wave 3 for bulimia nervosa. For both interviews, the screening question was: ''Have you ever in your life had eating binges during which you ate a lot of food in a short period of time?'' Interviewer characteristics and training have been described in detail elsewhere (Kendler, MacLean, Neale, Kessler, Health, & Eaves, 1991).

Statistical Analyses

To quantify the sources of variation in liability to obesity and binge eating, we used Mx (Neale, 1997) to conduct bivariate twin modeling (Figure 1a). The sources of variation are estimated for obesity and binge eating, yielding two sets of estimates of a^2, c^2,

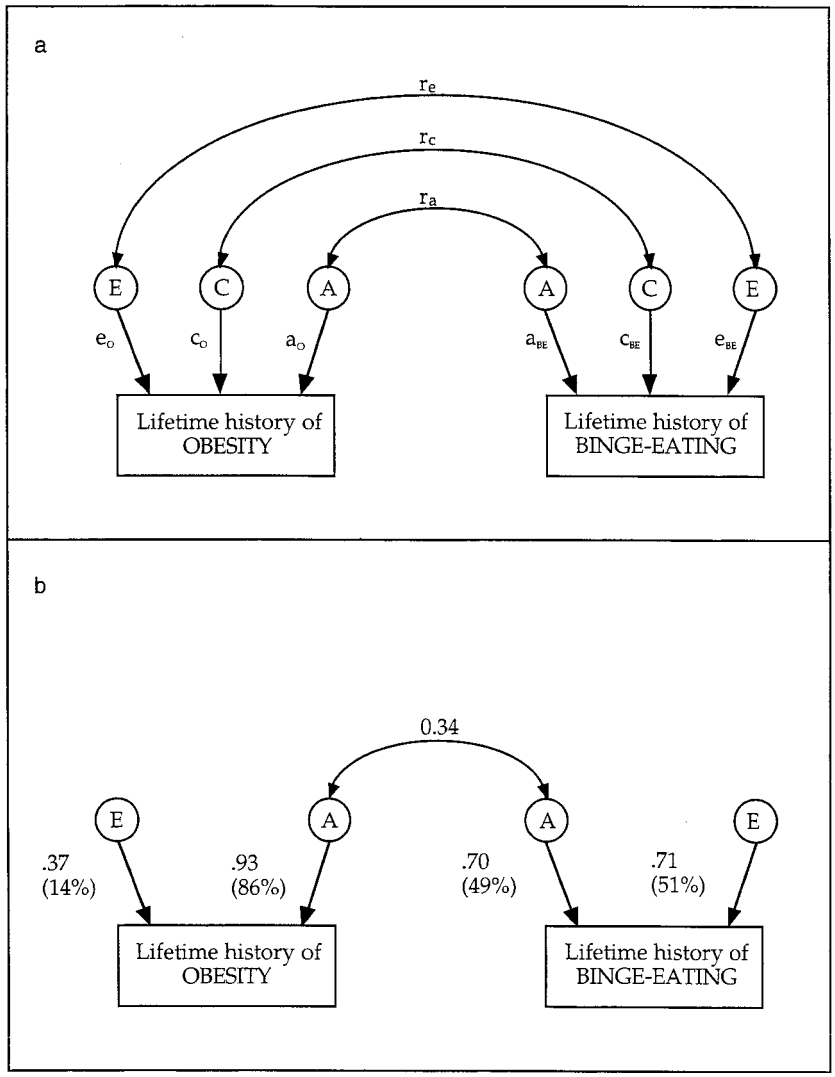

Figure 1. Graphical depiction of the bivariate twin model for a lifetime history of obesity and binge eating. (a) Full model; (b) path coefficients and parameter estimates for the best fitting model. See text for details.

and e^2 (Kendler, 1993; Neale & Cardon, 1992). We then calculated three distinct and independent correlations 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 Figure 1). These correspond to the correlation between additive genetic factors, common environmental factors, and unique environmental factors. 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, 1997). To select the most parsimonious model, we used

Table 1. Bivariate twin modeling of obesity and binge eating

Model	Obesity			Binge Eating			Correlations			Fit Statistics			
	a^2	c^2	e^2	a^2	c^2	e^2	r_a	r_c	r_e	χ^2	df	p	AIC
I. Full model	0.65	0.20	0.15	0.28	0.19	0.53	0.04	1.0	0.05				
ACE-ACE $r_a r_c r_e$*	(0.22–0.91)	(0–0.61)	(0.04–0.25)	(0–0.56)	(0–0.48)	(0.39–0.67)	(−1–0.1)	(−1–1)	(−0.37–0.45)	6.14	11	.86	−15.86
II. ACE-ACE $r_c r_e$	0.63	0.22	0.15	0.26	0.20	0.53		1.0	0.06				
	(0.23–0.83)	(0.05–0.60)	(0.05–0.25)	(0–0.28)	(0.05–0.48)	(0.39–0.67)	—	(0.42–1)	(−0.28–0.42)	6.16	12	.91	−17.84
III. ACE-ACE $r_a r_e$	0.74	0.12	0.15	0.33	0.14	0.53	0.47		−0.03				
	(0.27–0.94)	(0–0.56)	(0.05–0.25)	(0.03–0.60)	(0–0.44)	(0.40–0.67)	(0.19–1.0)	—	(−0.46–0.35)	8.37	12	.76	−15.63
IV. AE-ACE $r_a r_e$	0.86		0.14	0.33	0.15	0.53	0.43		−0.04				
	(0.77–0.94)	—	(0.04–0.25)	(0.02–0.60)	(0–0.44)	(0.38–0.67)	(0.18–1.0)	—	(−0.47–0.36)	8.64	13	.80	−17.36
V. AE-AE $r_a r_e$	0.86		0.14	0.49		0.52	0.35		−0.04				
	(0.77–0.94)	—	(0.04–0.25)	(0.38–0.61)	—	(0.39–0.67)	(0.16–0.54)	—	(−0.49–0.36)	9.35	14	.81	−18.65
VI. AE-AE r_a	0.86		0.14	0.49		0.51	0.34						
(Best fit)	(0.77–0.94)	—	(0.04–0.25)	(0.38–0.61)	—	(0.37–0.64)	(0.19–0.50)	—	—	9.39	15	.86	−20.61
VII. CE-CE $r_c r_e$		0.80	0.20		0.41	0.59		0.41	0.06				
	—	(0.72–0.88)	(0.10–0.30)	—	(0.31–0.51)	(0.47–0.71)	—	(0.23–0.59)	(−0.23–0.35)	17.27	14	.24	−10.73

*For each model, the first set of parameters (ACE) refer to obesity and the second set to binge eating. Values represent standardized parameter estimates with 95% confidence intervals in parantheses below.

Akiake's Information Criterion (AIC) (Akaike, 1987). The lowest AIC provides the optimal combination of explanatory power and parsimony.

RESULTS

Bivariate Twin Modeling

We considered the presence of obesity and lifetime history of binge eating by fitting a series of bivariate twin models to the observed data (Table 1). In the full sample, 29 monozygotic twin pairs and 9 dizygotic twin pairs were concordant for obesity, and 56 monozygotic twin pairs and 40 dizygotic twin pairs were concordant for binge eating. The full bivariate model fit the data well. Of the nested submodels, model V provided the best fit to the data as determined by AIC. This model included additive genetic and individual-specific environmental paths to both obesity and binge eating and the correlation between the additive genetic paths for each trait. Parameter estimates for obesity and binge eating were similar to previously published univariate models for these phenotypes (Maes, Neale, & Eaves, 1997; Sullivan, Bulik, & Kendler, 1998) and suggested a substantial contribution of additive genetic effects to obesity and a moderate contribution to binge eating. Moreover, the proportion of shared genetic variance between obesity and binge eating was estimated to be +0.34 (95% CI, 0.19–0.50), indicating a modest overlap in the genes that influence liability to these two traits (Figure 1b).

DISCUSSION

Our results support prior studies, which have shown a substantial contribution of additive genetic effects to both obesity and binge eating and further reveal a modest overlap of genetic factors that contribute to each of these two traits.

The fact that obesity exists in the absence of binge eating and that binge eating exists in the absence of obesity provides evidence that the genetic and environmental factors that contribute to liability to these two traits are not identical. Our bivariate twin analysis confirmed prior reports that obesity is a highly heritable condition with little influence from common environmental factors (Maes et al., 1997). Binge eating is also a moderately heritable trait, with little contribution from common environment (Sullivan et al., 1998). Of particular interest was the genetic correlation ($r_a = 0.34$: 95% CI, 0.19–0.50) between the two traits. This suggests some limited overlap in genetic etiological factors. Whereas there are genetic factors that contribute uniquely to obesity and genetic factors that contribute uniquely to binge eating, there also seems to be a relatively modest subset of genetic factors that are of etiological relevance to both traits. In addition, the best fitting model did not include a correlation between the unique environmental paths (e^2) for each trait, suggesting little overlap in the unique environmental factors that contribute to liability to obesity and binge eating. The fact that we were able to drop the common environmental and unique environmental correlations from the model without effecting a deterioration in fit suggests that the correlation between these two traits is primarily genetic. If obesity is caused binge eating (or vice versa), one would expect the causal effect to pass from both genetic and environmental causes of obesity to binge eating through the phenotype of obesity. The best-fitting model in this case would include both

genetic and environmental correlations (Neale & Kendler, 1995). Thus, our model argues against a direct causal pathway between these two phenotypes.

Limitations

Although our study is strengthened by our use of a genetically informative population-based design, our definition of binge-eating was broad and our assessment limited. Despite this limitation, the prevalence of binge eating in obese women in this sample was comparable to that reported in other studies.

Second, our study focused solely on the presence of binge eating and our results are therefore not automatically applicable to the proposed diagnostic criteria for BED.

Third, our findings are entirely limited to white women. This is particularly relevant, because both the prevalence of obesity (Mokdad, Serdula, Dietz, Bowman, Marks, & Koplan, 1999) and possibly the prevalence of binge eating (Warheit et al., 1993) differ between African-American and European-American populations. Similar studies in men and in non-white populations are necessary to determine whether the genetic and environmental contributors to these traits are constant across genders and ethnic groups.

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