

Genetic and Environmental Influences on Restrained Eating Behavior

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ABSTRACT

Objective: We examined the relative contributions of genetic and environmental influences to restrained eating.

Method: Restrained eating was assessed by the Restraint Scale in a survey mailed to all twins enrolled in the University of Washington Twin Registry. We used structural equation modeling to estimate genetic and nongenetic contributions to restrained eating.

Results: 1,196 monozygotic (MZ), 456 same-sex dizygotic (DZ) twins, and 447 opposite-sex twins were included in analyses. Restraint Scale scores were more closely correlated in MZ twins ($r_{\text{male}} = .55$, $r_{\text{female}} = .55$) than in same-sex DZ twins ($r_{\text{male}} = .31$, $r_{\text{female}} = .19$). Based

on structural equation modeling, the estimated heritability for restrained eating, adjusted for body mass index (BMI) and sex, was 43% (95% confidence interval 35–50%). There was little evidence for common environmental effects.

Discussion: These results indicate an inherited component to restrained eating. Genes could influence restrained eating directly or through inherited mediators such as personality factors or tendencies to gain weight. © 2009 by Wiley Periodicals, Inc.

Keywords: genetics; twin studies; eating behavior; restrained eating

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Introduction

People attempt to manage their weight for many reasons. Overweight and obese people are encouraged to lose weight for health reasons. Many normal-weight people, especially women, attempt weight loss for body image reasons. Restrained eating is one form of attempted weight regulation, characterized by the exertion of cognitive control over food intake. In contrast, unrestrained eaters self-regulate eating based on appetite and satiety. Paradoxically, prospective studies show that restrained eating is associated with a higher risk of developing obesity in preadolescent and adolescent girls,^{1,2} as well as greater weight gain in adults.^{3,4} In addition, restrained eating has been linked to

overeating in experimental conditions⁵ and, prospectively, to the development of disordered eating.^{6–8} Thus, restrained eating appears to be, at minimum, a counterproductive means of weight control, and also possibly an important point of intervention to prevent eating disorders.

Several possible mechanisms may lead restrained eaters to gain weight and/or to develop disordered eating. Restrained eaters may be more likely to engage in binge eating,^{9,10} perhaps because of appetite stimulation by hormonal factors,¹¹ and they may be less sensitive to internal satiety (and hunger) states.¹² Restrained eaters might also be genetically predisposed toward both weight gain and the behavioral response of recurrent dieting.¹³ However, evidence for genetic contributions to restrained eating has been mixed, possibly because of differences in the phenotypes identified by various psychometric measures of restrained eating.¹⁴

The original measure of restrained eating by Herman and Polivy,¹⁵ the Restraint Scale, assesses concerns with dieting and overeating, as well as weight fluctuations. Later restraint measures, such as the Restraint subscales of the Three Factor Eating Questionnaire¹⁶ (TFEQ) and the Dutch Eating Behavior Questionnaire¹⁷ (DEBQ), measure caloric restriction and intentions to restrict caloric intake. Particular instruments appear to categorize particular types of eating restraint. Specifically, the

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“restrained eaters” identified by the Restraint Scale are more likely to overeat or “disinhibit” their eating in a variety of situations, whereas those identified by the TFEQ and DEBQ are less likely to display such binge-type eating behavior.¹⁴

Given this background, it is not surprising that published estimates of the genetic contribution to restrained eating, as assessed by twin studies using the diverse available measures, show wide variation, ranging from 0 to 58%.^{18–20} Other pertinent investigations have observed that binge eating is moderately²¹ to highly²² heritable, and that disinhibited eating patterns have an estimated heritability of 45% (95% confidence interval 32–57%).²⁰ In sum, there is evidence that eating behaviors are genetically influenced, but the degree of influence on restrained eating remains unknown.

We conducted a classical twin study in a community-based sample of twins from Washington State by using the Restraint Scale to measure restrained eating. Our goals were to (1) estimate the proportion of variance in restrained eating that is explained by genetic and environmental factors and (2) determine whether genetic factors that influence restrained eating depend on the effect of body composition. We thereby sought to clarify the relative importance of inherited and noninherited factors to restrained eating and its consequences.

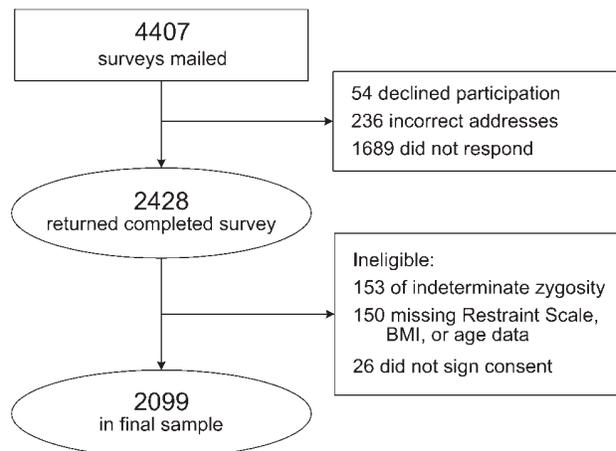
Method

Sample Population and Survey Methods

All twins were participants in the University of Washington Twin Registry, a community-based registry of twin pairs derived from applications for drivers' licenses in Washington State. The construction and characteristics of the Registry and its sample population are described in full elsewhere.²³ Briefly, because drivers' license numbers in Washington State are assigned on the basis of an individual's name and date of birth, the Department of Licensing asks each new applicant whether he or she is a member of a twin pair to avoid issuing duplicate license numbers. The University of Washington receives lists of applicants who are twins, and each member of the pair is invited to join the Registry. Twin pairs who complete an initial intake questionnaire are enrolled in the Registry. All Registry and data collection procedures involved in this study were approved by the University of Washington Institutional Review Board. Informed consent was obtained from all twins.

In August 2006, a written health survey was mailed to 4,407 twins (60% female) enrolled in the University of Washington Twin Registry, along with a consent form; a

FIGURE 1. Flow diagram of University of Washington Twin Registry health survey respondents.



self-addressed, postage-paid envelope; and a description of the study incentive. Twins who returned completed surveys were entered in a raffle for prizes including digital music players and online gift certificates. After 3 weeks, twins who had not responded were mailed a reminder letter and a second survey. Nonresponders were subsequently contacted by telephone, and could choose to complete the survey at that time. Optical scanning was used for initial data entry, followed by manual verification. Twins were excluded from the analyses if zygosity could not be determined or if they were missing a signed consent form or Restraint Scale, BMI, and/or age data (see Fig. 1).

Measures

Restraint Eating. The Restraint Scale is a 10-item self-report questionnaire designed to identify individuals with chronic dieting and weight concerns.¹⁵ The Restraint Scale identifies a population with high concern over their weight, who are prone to overeating in experimental situations where self-control is challenged,⁵ and to “unsuccessful” dieting, as exhibited by weight fluctuations and overeating.²⁴ The Restraint Scale was chosen because of our interest in a potential biological basis of the overeating observed in restrained eaters identified by this scale. The Scale's internal reliability, test-retest reliability, and predictive validity are well established.²⁵ However, the scale may overestimate restraint in overweight²⁶ and obese⁵ populations.

Zygosity Assignment. As part of the Registry's intake questionnaire, all twins were asked questions about childhood similarity. Responses to the similarity questions were used in a multistep process to assign zygosity. Studies in both U.S. and Scandinavian twin registries have repeatedly demonstrated that questions about childhood similarity in twin pairs correctly classify zygo-

sity with an accuracy of 95–98% compared with zygosity determined by biological indicators.^{27–30}

Weight, Height, and Demographic Variables. Current weight and height were self-reported (How much do you weigh without clothes or shoes? and How tall are you without shoes?). Highest grade of school completed was also self-reported. BMI was calculated (weight/height²). Age was determined from Registry records.

Statistical Analysis

Descriptive statistics for participant characteristics were calculated by using means and standard deviations for continuous variables and percentages for categorical variables. Generalized estimating equation regression models were used to assess differences in survey responders and nonresponders. Structural equation modeling for twin data³¹ was used to estimate genetic and environmental effects. The method builds on the assumption that monozygotic (MZ) pairs share 100% of their genetics and dizygotic (DZ) pairs share, on average, 50%. Before formal modeling, we computed the intraclass correlation estimates for Restraint Scale scores stratified by zygosity and sex. We then used structural equation modeling to estimate the genetic and environmental contributions to Restraint Scale scores. Models were fit to the raw data to estimate the amount of phenotypic variance due to additive genetic (A), common environmental (C), and unique environmental (E) factors. Additive genetic effects are the cumulative impact of multiple genes (as opposed to a single-gene inheritance model) that are estimated to contribute twice as much to the MZ as to the DZ twin correlation. Common environmental factors are assumed to be shared 100% by both MZ and DZ pairs. Unique environmental effects reflect experiences that are not shared by both members of a twin pair.

We used maximum likelihood estimation to calculate parameter estimates, 95% confidence intervals, and goodness of fit statistics for a series of structural equation models. Our full model was a general sex-limitation model that included all effects (ACE), separate parameter estimates for males and females, and a correlation estimate for opposite-sex pairs to assess sex-specific genetic effects. We then fit a common effects sex-limitation model that constrained the opposite-sex genetic correlation to .5. Our next model forced equal ACE parameter estimates in males and females. We then fit a model in which all variance was attributable to genetic and unique environmental factors (AE), and, finally, a model in which all variance was attributable to common and unique environmental factors (CE). Reduced models were constructed by constraining or removing appropriate parameters. Using likelihood ratio tests, we compared the goodness of fit for each model to a saturated model that allowed means and variances to differ by zygosity. We report both χ^2 -distributed differences in $-2(\log \text{likelihood})$ values for each reduced model com-

TABLE 1. Characteristics of 2,099 twins from the University of Washington Twin Registry

Characteristic	
Age, mean years (SD)	37 (16)
Zygosity (%)	
Monozygotic male–male	19
Dizygotic male–male	6
Monozygotic female–female	38
Dizygotic female–female	16
Dizygotic opposite sex	21
4-Year college degree or higher (%)	42
Caucasian (%)	90
Body mass index (%)	
Underweight	2
Normal weight	55
Overweight	26
Obese	16
Restraint Scale score, mean (SD)	13 (6)

n = 9 twins missing education.

pared to the saturated model and accompanying *p*-values. Models were also evaluated by using Akaike's Information Criterion.³² The model with the lowest Akaike's Information Criterion was judged to be the best-fitting and most parsimonious. To determine the components of the final adjusted model, likelihood ratio tests were used to investigate whether the removal of the covariates sex, age, and BMI resulted in a degradation of model fit. An alpha level of .05 was the criterion for a significant degradation of model fit. Because the Restraint Scale can overestimate restraint in overweight persons,²⁶ we also conducted a sensitivity analysis restricted to pairs in which both twins had normal weight.

Descriptive statistics and correlations were computed by using Stata 10.0 for Windows (StataCorp LP, 2008). Structural equation models were fit by using MxGUI version 1.4.06 (Department of Psychiatry, Virginia Commonwealth University, 2003).

Results

Participant Characteristics

We achieved an overall response rate of 55% (Fig. 1). Thirty-eight percent of twins never responded, and an additional 7% declined participation or had undeliverable addresses. Survey responders were older ($p < .01$), and more often female and MZ ($p < .01$, $p = .05$, respectively) than nonresponders. BMI measured at entry into the registry did not differ between responders and nonresponders ($p = .66$).

A total of 2,099 individual twins met inclusion criteria for the study. Twins were predominantly female and MZ (Table 1). The mean age was 37 years (range 19–81 years), and 89% of the sample was less than 60 years of age. Ninety percent were

Caucasian, and most twins had not completed a 4-year college degree. Forty-two percent of participants were overweight or obese.

Restraint Scale Scores and Covariates

The overall mean Restraint Scale score was 13. Female twins had higher scores than male twins (14 ± 5 versus 11 ± 6 , $p < .01$). The mean Restraint Scale score did not differ between MZ and DZ twins ($p = .57$).

Twin Correlations

Twin correlations by zygosity and sex were used to assess the similarity in Restraint Scale scores within twin pairs (Table 2). Restraint Scale scores were highly correlated in males and females among MZ pairs ($r_{\text{male}} = .55$, $r_{\text{female}} = .55$), but less so among same-sex DZ twin pairs ($r_{\text{male}} = .31$, $r_{\text{female}} = .19$). The stronger correlation in MZ compared to DZ pairs suggests a genetic component to restrained eating behavior. The correlation of

Restraint Scale scores was lower in opposite-sex DZ pairs ($r = .07$) compared to same-sex DZ pairs, providing some evidence that sex-specific genetic effects may play a role in trait variation, although confidence intervals among DZ pairs were wide and overlapping.

Structural Equation Modeling

Using structural equation modeling, we found that removing sex and BMI as covariates in our models resulted in a significant degradation of fit ($p < .01$), whereas age was not an important contributor ($p = .32$). We began analyses with a sex-limitation ACE model including a sex-specific genetic effect. Although it provided a reasonable fit to the data, the overall best-fitting and most parsimonious model equated the male and female parameter estimates and did not include a sex-specific genetic effect (Table 3), but was adjusted for confounding by sex and BMI. Additive genetic and unique environment factors made significant contributions to the overall variance in restrained eating, while common environment effects were dropped from the best-fitting model (Table 3). The adjusted heritability estimate for restrained eating was 43% (95% confidence interval 35–50%). The unique environment component, encompassing environmental effects specific to the individual and random error, accounted for the remainder of the variance (57%, 95% confidence interval 50–65%).

In the sensitivity analysis restricted to normal weight twins ($n = 1,210$ twins), the AE common

TABLE 2. Twin correlations for Restraint Scale score by zygosity and sex

Zygosity/Sex	N pairs	Intraclass Correlation (95% CI) ^a
Monozygotic male–male	148	0.55 (0.44, 0.65)
Dizygotic male–male	36	0.31 (0.00, 0.61)
Monozygotic female–female	290	0.55 (0.47, 0.63)
Dizygotic female–female	105	0.19 (0.00, 0.38)
Dizygotic opposite sex	141	0.07 (0.00, 0.26)

^a Correlations are based on data from 720 twin pairs and 659 individual twins.

CI, confidence interval.

TABLE 3. Model fit statistics for structural equation models of Restraint Scale score

Model	Model Parameters ^a			Model Fit Statistics			
	A	C	E	χ^2 ^b	df	p	AIC ^c
Sex limitation ACE model with sex-specific genetic effect				9.17	7	.24	−4.83
Male	0.41 (0.00–0.53)	0.00 (0.00–0.43)	0.59 (0.47–0.73)				
Female	0.46 (0.20–0.54)	0.00 (0.00–0.24)	0.54 (0.46–0.63)				
Common effects sex limitation ACE model				10.08	8	.26	−5.92
Male	0.27 (0.00–0.49)	0.13 (0.00–0.45)	0.60 (0.48–0.75)				
Female	0.42 (0.16–0.53)	0.03 (0.00–0.27)	0.54 (0.46–0.64)				
ACE model with equal male and female parameters				13.76	11	.25	−8.24
All twins	0.43 (0.31–0.50)	0.00 (0.00–0.09)	0.57 (0.50–0.65)				
AE model with equal male and female parameters				13.76	12	.32	−10.24
All twins	0.43 (0.35–0.50)	—	0.57 (0.50–0.65)				
CE model with equal male and female parameters				36.01	12	<.01	12.01
All twins	—	0.31 (0.24–0.37)	0.69 (0.63–0.76)				

^a Adjusted for body mass index and sex; ACE refers to a model that includes additive genetics (A), common environment (C), and unique environment (E); AE model includes only additive genetics and unique environment; CE model includes only common and unique environment.

^b Chi-square distributed likelihood ratio test statistic comparing each model to a saturated model that allowed means and variances to differ by zygosity.

^c Akaike's information criterion (AIC) is a global measure of goodness of fit; the best-fitting and most parsimonious model is shown in bold.

effects model again provided the best fit. The sex- and BMI-adjusted heritability was 47% (95% confidence interval 37–56%).

Discussion

We found compelling evidence for a genetic contribution to restrained eating. This contribution was independent of the influence of BMI, which is both highly heritable³³ and associated with Restraint Scale scores.²⁶ Unique environmental experiences and events specific to the individual were also important contributors. However, common environmental factors, such as family environment, did not substantially contribute to restrained eating. In addition, we found that the genetic contributions to Restraint Scale scores did not differ by sex. Moreover, we found no evidence of a sex-specific genetic effect, indicating that the same set of genes may influence Restraint Scale scores in both males and females. To our knowledge, this work represents the largest twin study of restrained eating performed in the U.S. and the largest to include both males and females.

Data from prior twin and family studies provide mixed evidence for genetic contributions to restrained eating, perhaps because the phenotypes identified by the various psychometric scales have different underlying risk factors. A study using the Restraint Scale to assess 149 twin pairs in the Minnesota Twin Registry found the heritability of restrained eating to be 58%, decreasing to 30% after controlling for BMI.¹⁸ The authors also estimated a heritability of 44% for the Cognitive Restraint subscale¹⁸ of the Three-Factor Eating Questionnaire (TFEQ).¹⁶ However, this was a relatively small study, and it is difficult to gauge the precision of the estimates, as confidence intervals were not reported. In another study of 456 MZ and 326 DZ Scandinavian male twin pairs, the Cognitive Restraint subscale of the TFEQ-R21 had an estimated heritability of 59% (95% confidence interval 52–66%), with a contribution of both additive and nonadditive genetic factors.³⁴ In contrast, a study using a modified 36-item TFEQ among 129 MZ and 81 DZ female twin pairs from the Virginia Twin Registry found the heritability for the Cognitive Restraint subscale to be 0% (95% confidence interval 0–30%), with a contribution from common environmental factors.²⁰ These disparate findings may reflect sex differences in influences on restrained eating as assessed by the TFEQ, a finding that we did not replicate in our study using the Restraint Scale. Finally, two family studies using the TFEQ Cognitive Restraint subscale produced generalized heritability estimates (estimates that include

both genetic and common environmental effects) for restrained eating of 6%¹⁹ and 28 (± 9)%.³⁵

Taken together, these data suggest that genetic inheritance contributes to restrained eating behavior, but the extent of phenotypic variance due to genetic factors remains unclear.³⁶ In addition, multiple methods of assessing restraint have been used and available measures appear to identify different populations of restrained eaters.^{5,14,24} These disparate phenotypes may reflect different relative contributions of genetic and environmental influences. For example, the cognitive restraint subscale of the TFEQ tends to identify individuals who successfully maintain restraint, and heritabilities tend to be lower when this scale is used, except for one study limited to males only.³⁴ In the population identified by the Restraint Scale, for whom overeating and weight fluctuations may accompany attempted restraint, our estimate of heritability is higher and more consistent with findings for disinhibition,^{20,35} as is the only prior estimate that used the same scale.¹⁸ One possible implication of these findings is that difficulty maintaining restraint, overeating, and tendencies to gain weight have stronger genetic components than successful restriction of calories. In fact, studies have documented potential pathways by which genes promote overeating and/or weight gain through the choice of more energy-rich foods³⁷ or through altered neural responses to the taste of food.³⁸

Genes might influence the restrained eating pattern identified by the Restraint Scale either directly or through other inherited pathways, including personality factors, body weight, body composition, or additional metabolic factors. The genetic pathway appears to be the same in males and females, although we examined a relatively small sample of males and cannot rule out sex-specific traits. There is evidence for genetic linkage of TFEQ Restraint subscale scores to genes located in two chromosomal regions,³⁵ but to our knowledge no genome-wide association or linkage studies aimed at delineating specific genetic vulnerabilities to restrained eating have been performed using the Restraint Scale. Nevertheless, our data strongly suggest that additive genetics underlies the behavior pattern, implying the possibility of a direct effect from multiple genes inherited together.

Alternatively, inherited factors associated with restraint might mediate the relationship between genes and restrained eating. One possibility is neuroticism, a genetically linked personality trait^{39,40} that is associated with disordered eating⁴⁰ and higher Restraint Scale scores.⁴¹ Twin studies have also shown genetic contributions to weight gain in

adulthood,⁴² lifetime maximum weight,⁴² metabolic efficiency,⁴³ diet-induced weight loss,⁴⁴ response to overfeeding,^{45,46} and body shape.⁴⁷ Although we controlled for BMI in our analyses, it is unknown if these additional inherited metabolic characteristics correlate with Restraint Scale scores, especially the weight fluctuation subscale.²⁶ If they do, our heritability estimates might reflect the inherited nature of weight fluctuations more so than cognitive control of eating. This is another possible explanation for discrepancies in the findings of twin studies using the TFEQ and Restraint Scale. Similarly, others have proposed that restrained eating scales identify individuals who are inherently prone to gain weight and to the behavioral response of attempted restriction,⁴⁸ so that our heritability estimates would reflect genetic factors underlying the variability in individual susceptibility to weight gain within an obesogenic environment,⁴⁹ rather than restrained eating per se. Finally, because high scores on the Restraint Scale are associated with body dissatisfaction and dieting concerns,²⁶ genetically determined body shapes that deviate from cultural ideals might predispose twins who are dissatisfied with their appearance to attempt cognitive control of eating. Future work should elaborate on these potential mechanisms.

This study has several potential limitations. First, the survey could have been subject to response bias, thereby affecting the ability of our Restraint Scale scores to accurately reflect underlying population means. Unfortunately, no data are available on the eating styles of nonresponders, limiting conclusions that can be drawn regarding the introduction of bias into the sample. Second, although twin studies are well-suited to examine hypotheses of polygenic or recessive-gene contributions to restrained eating, we cannot verify the underlying assumptions of polygenic inheritance or equal environments for MZ and DZ twin pairs. This is a limitation shared by most twin studies. Yet, other researchers of disordered eating have not documented substantial bias in heritability results, even when violations of the equal environment assumption were found.⁵⁰ Third, we used conventional epidemiologic methods of ascertaining zygosity without DNA verification. Random misclassification of zygosity, however, would tend to decrease the strength of associations. Fourth, we had a relatively small number of DZ pairs, fewer than we expected on the basis of population data. As a result, confidence intervals for estimates of twin correlations were large, prohibiting the detection of small sex-specific effects, and systematic differences in eating behavior between DZ pairs who did and did not participate may have occurred. Never-

theless, our overall sample was much larger than the one used in the only other classical twin analysis of Restraint Scale scores.¹⁸ Fifth, we used self-reported measures of weight and height, which can result in misclassification of BMI, especially in overweight and obese persons.⁵¹

Finally, although we regard our use of a community-based registry as a strength in deriving our study sample, the twins were primarily from Washington State, predominantly Caucasian, and included a wide age range of adult ages. Therefore, our findings cannot necessarily be generalized to other racial groups or to pediatric populations. Although the inclusion of a range of ages from young adult to elderly may have introduced variability due to differing influences on restrained eating across the lifespan, we did not find that age was a significant confounder in our analyses.

Despite these limitations, we feel our work substantiates the inherited nature of eating behavior. We have demonstrated that cognitive attempts to control eating, and/or tendencies toward overeating and weight fluctuation in conjunction with such attempts, are related to genetic factors, independent of the effects of sex and BMI. In addition, we have interpreted our findings within the context of previous genetic studies that used different constructs of restrained eating. Further studies should focus on elucidating the specific genes, gene-environment interactions, and/or inherited factors that mediate restrained eating, paying special attention to defining and refining the particular phenotype under study. Such data will clarify the genetic, physiological, cognitive, and environmental factors that influence human eating behavior, weight gain, and disordered eating. With these data, interventions to reduce the risk of disordered eating could be targeted to the appropriate risk groups.

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