ORIGINAL ARTICLE

Changes in Genetic and Environmental Influences on Disordered Eating Across Adolescence

A Longitudinal Twin Study

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Context: Previous research suggests substantial increases in genetic effects on disordered eating across adolescence. Unfortunately, these studies were cross-sectional and focused primarily on early (age 11 years) vs late (age 17 years) adolescence.

Objective: To examine longitudinal changes in genetic and environmental influences on disordered eating across early, mid, and late adolescence.

Design and Setting: Population-based study of female same-sex twins.

Participants: Seven hundred seventy-two female adolescent twins from the Minnesota Twin Family Study assessed at ages 11, 14, and 18 years.

Main Outcome Measures: Disordered eating symptoms (ie, body dissatisfaction, weight preoccupation, binge eating, and the use of compensatory behaviors) were assessed with the total score from the Minnesota Eating Behavior Survey.

Results: Biometric model-fitting indicated significant changes in genetic and shared environmental effects across early to mid adolescence. Although genetic factors accounted for a negligible proportion (6%) of variance at age 11 years, genes increased in importance and accounted for roughly half of the variance (46%) in disordered eating at ages 14 and 18 years. Shared environmental influences decreased substantially across these same ages.

Conclusions: Findings highlight the transition from early to mid adolescence as a critical time for the emergence of a genetic diathesis for disordered eating. The increase in genetic effects during this developmental stage corroborates previous research implicating puberty in the genetic etiology of eating disorders.

Arch Gen Psychiatry. 2007;64(12):1409-1415

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TWIN RESEARCH OVER THE past 20 years has demonstrated that anorexia nervosa (AN),[1-4] bulimia nervosa (BN),[5,6] and disordered eating (DE)[7,8] exhibit significant genetic effects in adults. Heritabilities tend to be greater than 50% with the remaining variance due to nonshared rather than shared environmental influences. These findings clearly underscore the importance of genetic factors for eating disorders in adulthood. However, they tell us little about genetic influences on DE during adolescence, the peak period of risk for eating disorders.[9] Moreover, they ignore potential developmental changes in genetic influences that may provide important clues as to the mechanisms of genetic effects. Indeed, identifying periods during which genetic influences become prominent can inform neurobiological and molecular genetic research about the systems and genes that may contribute to the genetic diathesis of the disorders.

We have previously examined developmental differences in genetic and environmental influences on DE using cross-sectional data from the Minnesota Twin Family Study (MTFS).[7,10,11] Findings were striking in suggesting substantial differences in genetic effects across development. We found essentially no genetic influence on overall levels of DE (ie, levels of body dissatisfaction, weight preoccupation, binge eating, and the use of compensatory behaviors) in 11-year-old twins but found significant genetic effects (>50%) in 17-year-old twins.[7] Importantly, however, genetic influences did contribute to DE (>50%) in 11-year-old twins who had begun puberty.[10] These findings collectively imply that puberty may play a role in the genetic diathesis for DE. A subsequent study of the twins at age 14 years offered additional support for this hypothesis; results indicated that pubertal development moderates genetic effects on DE (ie, genetic influences were

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stronger in twins who were in mid to late as compared with early puberty).[11] Taken together, these findings highlight meaningful developmental differences in genetic influences on DE across adolescence and are consistent with the possibility that pubertal development contributes to these differences.

Nonetheless, 2 key limitations of our previous work limit the conclusions that can be drawn. All of our previous data were cross-sectional, potentially obscuring the origin of observed developmental differences in etiology by confounding cohort with age effects. Our use of cross-sectional data also limited our ability to examine overlap (eg, genetic influences in early adolescence continue influencing DE into late adolescence) vs specificity (eg, new genetic influences emerge during mid adolescence) in genetic and environmental influences on DE across adolescence. The present study addresses these limitations by examining changes in genetic and environmental influences across adolescence using longitudinal twin data. Early, mid, and late adolescence was examined by analyzing data from a single cohort assessed at ages 11, 14, and 18 years. Thus, in addition to providing an opportunity to replicate our cross-sectional results, our longitudinal design also allowed us to examine the extent to which genetic and environmental influences are specific to particular developmental periods.

As noted earlier, information about developmental specificity in genetic effects may enhance efforts to identify neurobiological and genetic substrates. In particular, our examination of longitudinal changes in genetic and environmental influences on DE will provide important, if indirect, information on the possible role of puberty. As puberty commonly occurs in girls by age 14 years, we hypothesized that there would be a significant increase in genetic effects between ages 11 and 14 years. However, we did not expect changes in the magnitude of genetic factors from ages 14 to 18 years. If puberty contributes to increases in genetic effects on DE during adolescence, and puberty is typically complete by age 18 years, genetic influences should be stable across mid to late adolescence.

METHODS

PARTICIPANTS

Participants included 772 same-sex female twins (386 pairs) drawn from the MTFS, a population-based, longitudinal twin study designed to examine genetic and environmental risk factors for substance use and psychopathology. Twins were ascertained from State of Minnesota birth records and located using public databases, including telephone directories and driver license registrations. Although recruitment procedures, exclusion criteria, and zygosity determination are detailed elsewhere,[12] it is notable that the MTFS has been successful in locating more than 90% of twin births in the state of Minnesota in any given birth year and recruiting more than 80% of eligible twin families to participate in the research.

The twins were assessed at 3 time periods, corresponding roughly to early, mid, and late adolescence. At baseline, twins had a mean (SD) age of 11.71 (0.46) years (range, 10-13 years). At the first and second follow-up assessments, twins had a mean (SD) age of 14.78 (0.52) years (range, 13-16 years) and a mean (SD) age of 18.23 (0.68) (range, 17-20 years), respectively. Although the MTFS aims to assess twins every 3 years, the last assessment period averaged 4 instead of 3 years, resulting in a mean age that is slightly higher than other MTFS cohorts.[7] Of the 772 twins who participated, 713 (92%) provided complete data on our eating disorder measure at age 11 years, 687 (89%) provided complete data at age 14 years, and 634 (82%) provided complete data at age 18 years.

All study procedures were reviewed and approved by the institutional review board at the University of Minnesota, where data collection took place. After we provided a complete description of the study to participants, we obtained written informed assent from the twins and written informed consent from the parents.

MEASURES

Similar to our previous developmental twin studies, self-reported DE attitudes and behaviors were assessed with the 30-item total score of the Minnesota Eating Behavior Survey (MEBS).[7,13] The MEBS is a revised version of the Eating Disorders Inventory[14] that was modified for use with girls as young as age 9 years (ie, the language was simplified, items were limited to those assessing DE symptoms, the response format was changed to true/false[13]). The MEBS total score assesses general levels of eating pathology in the areas of body dissatisfaction (dissatisfaction with the size and/or shape of one's body), weight preoccupation (preoccupation with dieting, weight, and the pursuit of thinness), binge eating (the tendency to engage in episodes of overeating as well as having attitudes conducive to binge eating), and the use of compensatory behaviors (the tendency to use or to contemplate using inappropriate compensatory behaviors such as self-induced vomiting and laxatives to control weight). The scale is scored in the traditional "pathological" direction with high scores indicating greater degrees of DE. The MEBS total scores were prorated for twins who were missing 1 to 3 (ie, $<10\%$) items. We coded the MEBS total scores as missing when more than 3 items were missing. To account for positive skew while still allowing for changes in the mean and variance with age, the MEBS total scores were log-transformed.

We focused on the MEBS total score as our measure of DE rather than *DSM-IV* diagnoses or other measures for several reasons. First, the prevalence of AN and BN diagnoses at age 11 and 14 years was much too low ($<0.1\%$) for statistical analyses. This is not surprising given that preadolescent and early-adolescent onset of these disorders is rare.[9] Second, the MEBS total score shows ample variability and strong psychometric properties across adolescence. The internal consistency of the MEBS total score is similar in twins with ages of 11 (0.86), 14 (0.87), and 18 (0.89) years[13]; the scale shows good stability ($r=0.61$) across a 3-year test-retest[7,13]; and it successfully discriminates between eating disorder cases and controls.[7,13] Third, the magnitude of genetic and environmental influences on eating disorder diagnoses and DE symptoms has been found to be very similar (see Bulik et al[4] and Klump et al[15]). Finally, because the MEBS total score is on a continuous rather than categorical scale, it provides greater power for the Cholesky decomposition models used to examine changes in etiologic effects.

STATISTICAL ANALYSES

Cholesky decomposition models were used to examine the magnitude of age differences in genetic and environmental effects and the degree of age overlap vs specificity in these effects. Cholesky decomposition is a multivariate technique based on the principles of factor analysis that provides estimates of additive

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genetic (A) (genetic influences that add across genes), shared environmental (C) (environmental influences that are shared by reared-together twins and are a source of behavioral similarity), and nonshared environmental (E) (environmental influences that are not shared by reared-together twins and are a source of behavioral dissimilarity) contributions to variance in, and to covariance between, DE scores across time.

The 3-factor ACE Cholesky model used in this study is depicted in **Figure 1**. Genetic and environmental contributions to the variance in DE at age 11 years (which are represented as a_{11}, c_{11}, and e_{11} in the figure) are obtained by simply squaring their respective path estimates. The genetic and environmental variance in DE at age 14 years is decomposed into components attributable to the genetic and environmental effects on DE at age 11 years (represented as the square of a_{21}, c_{21}, e_{21}) as well as residual components that are independent of the genetic and environmental variance at age 11 years (represented as the square of a_{22}, c_{22}, e_{22}). Finally, variance in DE at age 18 years is decomposed into components attributable to the genetic and environmental effects on DE at age 11 years (represented as the square of a_{31}, c_{31}, e_{31}) and 14 years (represented as the square of a_{32}, c_{32}, and e_{32}) and residual components that are independent of the genetic and environmental variance at ages 11 and 14 years (represented as the square of a_{33}, c_{33}, and e_{33}).

To statistically compare the magnitude of genetic effects across age, we compared the fit of a series of nested Cholesky models in which the A, C, and E estimates were constrained to be equal across age vs models in which they were allowed to vary. Using these constraint models, we were able to explicitly evaluate whether, for example, the magnitude of genetic influence at age 11 years was equal to or smaller than those at age 14 and 18 years.

These models were fit to the raw twin observations using the Mx statistical software program.[16] The raw data option in Mx allows for the analysis of all relevant twin data regardless of the number of assessments completed. In addition, the raw data option treats missing data (eg, from participant dropout) as missing at random. This treatment of missing data is expected to produce less biased and more consistent estimates than other techniques (eg, pairwise or listwise deletion).

Models were fit using the maximum likelihood option in Mx. We first fit a baseline, or unrestricted, model that allows variances, covariances, and means of these data to be freely estimated by minimizing minus twice the log-likelihood (–2lnL). Differences in the minimized value of –2lnL between the baseline model and more restrictive models (ie, $-2\ln L_{\Delta}$) yield a likelihood-ratio χ^2 test that is used to test the significance of the more restrictive model. A nonsignificant χ^2 indicates that the more restrictive model provides an appropriate fit to the data. In addition, models that minimize Akaike's information criterion (AIC) ($AIC = \chi^2 - 2*\Delta df$) are preferred as the AIC balances model fit with parsimony.

RESULTS

DESCRIPTIVE STATISTICS AND TWIN CORRELATIONS

As expected, MEBS scores exhibited linear increases across adolescence ($F_{1,562} = 4.28$, $P = .04$) with significantly higher levels of DE at age 18 years than at ages 11 and 14 years. Nonetheless, ample variability in MEBS scores was present at all ages, and a significant minority of twins scored above the MEBS clinical cut-off (ie, score = 11[7]) at ages 11 (10%), 14 (15%), and 18 (16%) years, respectively.

Twin intraclass correlations offer preliminary evidence that there are shifts in etiologic factors across age (**Table 1**). At age 11 years, the monozygotic (MZ) and dizygotic (DZ) correlations were approximately equal and moderately large, suggesting little to no genetic influence but significant shared environmental effects. By contrast, MZ twin correlations were more than double those of the DZ twins at ages 14 and 18 years, primarily as a result of decreases in DZ twin similarity (rather than increases in MZ similarity). These findings are indicative of prominent genetic influences and suggest that despite modest increases in DE from age 11 to 14 years, there are rather large differences in the types of etiological factors contributing to DE at each age.

Differences in MZ and DZ twin correlations were nearly identical at ages 14 and 18 years, suggesting minimal changes in genetic and environmental influences. Consequently, despite increases in mean levels of DE across these 2 ages, little to no change occurs in the etiologic factors contributing to DE in middle to late adolescence.

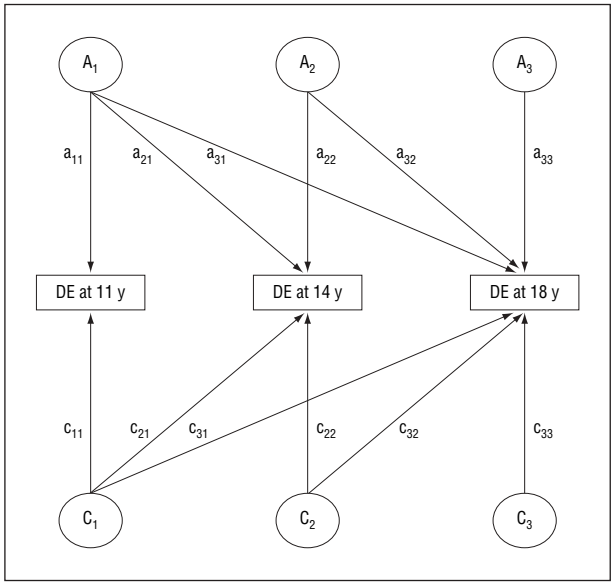

Figure 1. Standardized path diagram of a 3-factor Cholesky decomposition model. The variance in liability to disordered eating (DE) at each assessment is parsed into that which is due to additive genetic effects (A_1, A_2, and A_3) and shared environmental effects (C_1, C_2, and C_3). Although they are estimated in the model, nonshared environmental effects (E_1, E_2, and E_3) are not pictured here for ease of presentation. Similarly, this path diagram represents only one twin in a pair, although the model is identical for the co-twin. Individual paths, which are squared to estimate the proportion of variance accounted for, are represented by lowercase letters followed by 2 numerals (eg, a_{11}, a_{21}, and a_{31}).

MODEL FITTING

Findings from the Cholesky models confirm these impressions. Differences in model fit between the fully constrained and fully unconstrained models (see **Table 2**) indicated that A, C, and E could not be constrained to be equal across all 3 ages without a significant worsening in model fit (ie, a statistically significant $-2\ln L_{\Delta}$ value). We subsequently fit a series of nested models that differentially constrained A, C, or E across ages. Given the pattern of twin correlations described earlier, we were particularly interested in testing a model in which A and C were constrained to be equal across ages 14 and 18 years but were allowed to vary between age 11 years and the 2

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Table 1. Means (SD), Ranges, and Twin Intraclass Correlations for MEBS Total Scores[a]

Subject Age, y	Score, Mean (SD)[b]	Score Range	Intraclass Correlations[c]		
			MZ	DZ	Test of Equality *z*
11	5.63 (4.87)[d]	0-23	0.50	0.47	0.50
14	5.94 (5.69)[d]	0-24	0.54	0.28	3.81[e]
18	6.43 (5.60)	0-27	0.54	0.24	4.20[e]

Abbreviations: DZ, dizygotic twins; MEBS, Minnesota Eating Behavior Survey; MZ, monozygotic twins.
[a] Although log-transformed scores were used for twin correlations and model-fitting analyses, raw means, standard deviations, and ranges are presented for ease of interpretation.
[b] Sample sizes for mean comparisons were, for age 11 years, 713; for age 14 years, 687; and for age 18 years, 634.
[c] Sample sizes for correlations were, for age 11 years, 680 (MZ, 410; DZ, 270); for age 14 years, 666 (MZ, 412, DZ, 254); and for age 18 years, 588 (MZ, 366; DZ, 222). Smaller sample sizes for the correlations are due to the necessary exclusion of pairs for whom one co-twin was missing the MEBS score at the indicated age.
[d] Significantly different ($P<.05$) from the 18-year-old score in post hoc contrasts conducted within repeated-measures analyses of variance.
[e] $P<.001$.

Table 2. Test Statistics for Cholesky Decomposition Models

Model	–2lnL	*df*	$-2\ln L_{\Delta}$ (*df*)	*P* Value	AIC
1. Fully unconstrained	1110.40	2005	-	-	0
2. Fully constrained	1133.22	2011	22.82 (6)	<.001	–2888.78
3. Constrain A across age	1117.12	2007	6.72 (2)	.03	–2896.89
4. Constrain C across age	1113.70	2007	3.30 (2)	.19	–2900.30
5. Constrain E across age	1113.18	2007	2.78 (2)	.43	–2900.82
6. Constrain A: ages 14 and 18 years C: all ages E: all ages	1119.12	2010	8.72 (5)	.12	–2900.88
7. Constrain[a] A: ages 14 and 18 years C: ages 14 and 18 years E: all ages	1114.83	2009	4.43 (4)	.35	–2903.17

Abbreviations: A, additive genetic effects; AIC, Akaike's information criteria; C, shared environmental effects; E, nonshared environmental effects; –2lnL, –2 times the log likelihood; $-2\ln L_{\Delta}$, differences in –2lnL values between the fully unconstrained model and each reduced model.
[a] The best-fitting model.

older adolescent ages (ie, model 7 in the table). Indeed, this model provided a better fit to the data (as indicated by the lowest AIC and nonsignificant $-2\ln L_{\Delta}$ value) than the models that constrained A, C, or E to be equal across all 3 ages.

Standardized parameter estimates from model 7 are shown in **Figure 2**. Because shifts in the unstandardized A, C, and E parameters looked essentially identical (data not shown), only standardized parameter estimates are presented for ease of interpretation. As shown in Figure 2, shifts in the magnitude of genetic, shared environmental, and nonshared environmental influences are present across age. At age 11 years, shared and nonshared environmental influences predominate while genetic factors account for only a very small proportion of variance (ie, 6%). By contrast, at ages 14 and 18 years, genetic factors account for roughly half of the variation in levels of disordered eating with nonshared environmental influences accounting for the majority of the remaining variance. Shared environmental influences have little impact on disordered eating at ages 14 and 18 years.

Despite changes in the magnitude of etiologic effects across age, a significant degree of overlap was observed in the types of genetic and environmental influences that are important at each age. Genetic influences on disordered eating at age 14 years are primarily due to genetic factors that were present at age 11 years (45%) rather than those that were new at age 14 years (ie, 1%). Likewise, genetic influences on disordered eating at age 18 years were due entirely to genetic effects present at ages 11 (28%) and 14 years (18%). Importantly, there were no genetic influences on disordered eating that were new at age 18 years. (Note: in addition to the Cholesky models, we fit a series of common pathway models to confirm that the same genetic factors were influencing DE across adolescence. Results revealed that age-specific genetic factors could be dropped from the model without a significant worsening of fit, but genetic factors common to each age could not be dropped. These results confirm that the genetic influences operating on DE at age 11 years are likely the same as those operating at ages 14 and 18 years.)

Findings for shared environmental influences were similar in that the majority of shared environmental influences at ages 14 and 18 years were due to shared environmental factors that were present at earlier ages. By

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contrast, there were several new sources of nonshared environmental influences at ages 14 and 18 years (ie, residual nonshared environment was 38% at both ages). Some of these residual effects are likely due to measurement error as estimates of nonshared environment include these sources of variation. Nonetheless, given the increased autonomy of girls in mid to late adolescence, at least some of the residual nonshared environmental influence likely reflects new environmental experiences that are unique to co-twins and differentially influence their levels of disordered eating.

COMMENT

To our knowledge, this is the first longitudinal twin study of developmental shifts in etiologic influences on DE across adolescence. Consistent with our hypotheses, genetic factors increased substantially from early to mid adolescence. Indeed, genetic effects were essentially nonexistent in early adolescence but increased to almost 50% of the variance by mid adolescence. These results confirm cross-sectional findings[7] and significantly extend them by identifying mid adolescence as the critical time for changes in etiologic effects. Indeed, no new genetic influences on DE were identified after early and mid adolescence. By contrast, shared environmental effects decrease across adolescence, accounting for 0% of the variance by late adolescence. Nonshared environmental factors appear to contribute to DE throughout adolescence. Taken together, these findings highlight critical developmental shifts in etiologic influences on DE and contribute to a growing literature supporting changes in the magnitude of genetic and environmental influences across development for several psychiatric phenotypes (eg, Burt et al[17]).

What might account for the significant increase in genetic effects on DE during early to mid adolescence? Theorists have argued that increases in pressures to be thin[18] and changes in sexual maturity[19,20] may contribute to increased risk for eating disorders in adolescent girls in Western cultures. Although potentially relevant to overall DE development, these psychosocial factors cannot easily account for changes in genetic effects observed in this study. As discussed earlier, MZ twins remain similar in their DE patterns across age, whereas DZ twins become much less similar after middle adolescence. The differential change in DZ relative to MZ twin similarity cannot be attributable to the broad effects of culture alone as all of the co-twins reside in a Western culture. These changes also cannot be easily attributable to psychosocial influences alone as it is unclear how or why psychosocial influences would act to make DZ twins dissimilar but not affect MZ twin similarity. One possibility in this regard would be if the increased environmental and/or physical similarity of MZ twins make them more similar for eating pathology relative to DZ twins (ie, if the equal environments assumption is violated[21]). However, previous studies from this and other data sets have confirmed that the increased environmental[4,22] and physical similarity (in terms of general appearance or body weight)[21] of MZ twins does not influence their similarity for disordered eating. Thus, although these psychosocial risk factors are likely to be important in the development of eating pathology, they are not sufficient for explaining the changes in genetic effects across early to middle adolescence observed in this study.

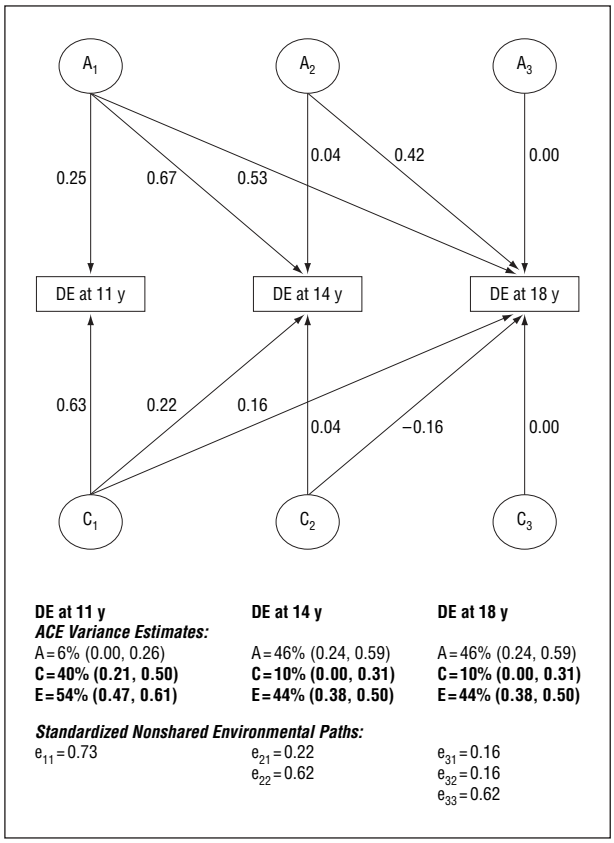

Figure 2. Standardized path diagram of the best-fitting Cholesky decomposition model of disordered eating (DE). The proportion of variance in DE accounted for by genetic (A) and environmental (C and E) factors at each age are listed below the figure; statistically significant variance estimates are noted in bold text. Individual paths within the figure are squared to estimate the proportion of variance accounted for in DE. The standardized nonshared environmental paths (e_{11}, e_{21}, etc) are included at the bottom of the figure for ease of presentation.

However, interactions between psychosocial factors and genetic risk might contribute. Gene-environment interactions are present when psychosocial risk factors have differential effects on disease outcome depending on the level of genetic risk.[23,24] Specifically, individuals with genetic predispositions are at increased risk for a disorder when environmental risk factors are present. Applied to the current study, increases in genetic effects from age 11 to 14 years may be the result of the potentiation of genetic risk by psychosocial risk factors (ie, cultural pressures for thinness, increased family conflict) that may emerge or increase between early and middle adolescence. Importantly, the putative environmental risk factors must be shared by co-twins as nonshared environmental factors would decrease MZ and DZ twin correlations across age. To date, no studies have examined the role of gene-environment interactions in eating disorders or their developmental trajectories. Future twin research should examine this possibility more closely using assessments of shared

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psychosocial and genetic risk factors for DE during early and middle adolescence.

As noted earlier, puberty is also likely to contribute to the observed changes in genetic effects. In previous analyses of MTFS twins,[10,11] puberty was found to moderate genetic influences on DE. We found that genetic influences increased significantly across pubertal development, accounting for 0% of the variance in prepubertal twins but 46% of the variance in postpubertal twins.[11] Given that puberty occurs between the ages of 10 and 14 years in most girls, it may be that puberty accounts for the increases in genetic effects from age 11 to 14 years in the current study.

Puberty may also account for the large degree of overlap in genetic effects across all adolescent ages. In our previous analysis of the 11-year-old cohort, we found no genetic influence on DE in prepubertal twins but significant genetic effects (>50%) in the small number of 11-year-old twins who had begun puberty.[10] Thus, in the present study, genetic effects that are present at age 11 years and overlap with those at ages 14 and 18 years are genetic effects related to puberty as only twins who have begun puberty exhibit genetic effects at age 11 years. In aggregate, findings across our developmental twin studies suggest that genetic influences on DE become prominent at puberty and then continue to influence DE across adolescence. Differences in the magnitude of genetic effects across adolescence likely reflect differences in the proportion of twins who have begun puberty rather than differences in the types of genetic factors.

If puberty does account for developmental shifts in genetic effects, then it will be important to identify the mechanisms by which puberty influences the genetic diathesis of eating pathology. Gene-environment interactions described here may play a role, given that many psychosocial risk factors (ie, family conflict[25,26]) appear to increase during the pubertal period. Significant increases in body weight and anxiety may also contribute as these characteristics are heritable[27,28] and higher body mass index (BMI) and anxiety are significant risk factors for eating disorders.[29,30] Thus, increases in genetic effects on DE may be due to increases in genetic influences on these other characteristics. To examine these possibilities, we re-ran our Cholesky models after partialling out the effects of BMI and trait levels of anxiety (assessed with the State-Trait Anxiety Inventory[31]) from each twin's DE score. The results remained unchanged, suggesting that changes in BMI and/or anxiety across age cannot account for changes in genetic effects on DE.

By contrast, ovarian hormones that are activated at puberty and drive pubertal changes in girls may contribute.[32,33] Estrogen and progesterone serve as transcription factors to regulate messenger ribonucleic acid expression within neurotransmitter systems important for eating disorders (eg, serotonin[34]). Ovarian hormones also show robust influences on food intake (see Asarian and Geary[35]) and predict menstrual cycle changes in DE in women with BN.[36] Taken together, these findings highlight the potential role for ovarian hormones in the genetic diathesis of DE during puberty and suggest a need for future studies to more closely examine their phenotypic and genetic effects.

In addition to shifts in genetic effects, we also observed decreases in the influence of shared environmental factors across adolescence. These findings corroborate those within the larger behavioral genetic literature showing a general decrease in these influences across development.[37] Waning shared environmental effects could be due to gene-environment interactions as these interactions contribute to genetic rather than shared environmental components of variance in twin models.[23] However, decreases may also reflect the growing independence of adolescence. During this time, shared family influences decrease as adolescents become more independent and involved with peers and extracurricular activities. These experiences frequently result in increases in the influence of nonshared environmental factors as more of an adolescent twin's environment is independent of the family environment. Our data confirm these impressions: the best-fitting model indicated that there are new, age-specific nonshared environmental influences on DE at ages 14 and 18 years. Thus, environmental factors that are unique to each co-twin (eg, participation in dance classes by one co-twin but not the other) and unique to specific ages (eg, entrance into middle vs high school) may have more significant effects on DE than those that are shared by twins.

Several limitations of our study should be noted. First, we focused on disordered eating symptoms rather than AN and BN. The low prevalence of these disorders in early adolescence, coupled with the low incidence of twin births, makes it very difficult to examine these clinical syndromes in developmental twin research. Our continuous measure of DE has been shown to successfully discriminate women with AN and BN from controls.[13] Nonetheless, the extent to which our findings extend to women with AN and BN remains unknown.

Second, the Cholesky decomposition models were unable to parse out gene-environment interactions or epistatic genetic effects. Future research should examine whether these processes impact developmental changes in etiologic effects for eating disorders. Third, the MEBS true/false response format may have affected parameter estimates through decreased variability in scores. Additional research is needed to compare our results with studies using instruments with more diverse response formats (ie, Likert scales).

Finally, we examined DE at 3-year intervals rather than more frequent (eg, annual) assessments. These protracted assessments limited our ability to identify when during early to middle adolescence changes in etiologic factors occur. For example, we could not definitively determine whether longitudinal changes occur during puberty since the vast majority of twins were prepubertal (more than 90%) at age 11 years and postpubertal at age 14 years (more than 90%). Thus, changes in pubertal status were significantly confounded with changes in age. Indeed, when longitudinal analyses were re-run (data not shown) using only those twins whose pubertal status changed from age 11 to 14 years, we obtained essentially identical results to those reported for age. These findings highlight the need for additional longitudinal research that incorporates more frequent assessments to identify developmental shifts in etiologic effects. This research would benefit from including measures of

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puberty, psychosocial risk factors, ovarian hormones, and their interactions to provide the most comprehensive picture of DE development during the critical adolescent period.

Submitted for Publication: March 3, 2007; final revision received June 5, 2007; accepted June 28, 2007.
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Author Contributions: All authors had full access to the data in the study.
Financial Disclosure: None reported.
Funding/Support: The research presented herein was supported by grants from the National Institute on Drug Abuse (DA 05147) and the National Institute of Alcohol Abuse and Alcoholism (AA 09367) awarded to Drs McGue and Iacono for study design and data collection. Data analyses and manuscript preparation were supported by a grant from the National Institute on Mental Health (MH 65447) awarded to Dr Klump.
Previous Presentation: Parts of the manuscript were presented at the 2006 International Conference on Eating Disorders; June 9, 2006; Barcelona, Spain.
Additional Information: The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory [M-EDI]) was adapted and reproduced by special permission of Psychological Assessment Resources, Inc, 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, Polivy, Copyright 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

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