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## Age Differences in Genetic and Environmental Influences on Weight and Shape Concerns

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### Abstract

**Objective**—Previous research has shown important developmental shifts in genetic and environmental influences for disordered eating. However, little research has examined age differences for weight/shape concerns, two key components of eating disorders. The goal of the present study was to investigate these age differences in pre-adolescent, adolescent, young adult, and mid-adult twins.

**Method**—Participants included 2,618 female twins (ages of 10–41 years) from three large twin registries. Shape and weight concerns were assessed with the Eating Disorders Examination Questionnaire.

**Results**—Genetic influences were modest in pre-adolescent twins, but significant from early-adolescence through middle adulthood. Shared environmental factors showed the opposite pattern, with the largest shared environmental contributions occurring in the youngest age group. Nonshared environmental effects remained relatively constant across age.

**Discussion**—Findings highlight the importance of age differences in genetic and environmental influences. Possible mechanisms include gene × environment interactions and biological changes associated with key developmental stages.

Twin studies have suggested that anorexia nervosa (AN), bulimia nervosa (BN), and eating disorders not otherwise specified (e.g., binge eating disorder) [1–9] are moderately heritable in adulthood. However, heritability for disordered eating symptoms (e.g., weight preoccupation, binge eating, compensatory behaviors) is unlikely to be stable over the lifetime. Disordered eating shows important developmental shifts in etiologic influences across adolescence, with increasing genetic and decreasing shared environmental influences. For example, cross-sectional and longitudinal studies have shown lower heritability of overall levels of disordered eating symptoms (i.e., levels of binge eating, compensatory behaviors, weight preoccupation) in pre- and early adolescent twins as compared to twins in middle and late adolescence [4,10,11]. Moreover, longitudinal data suggest that there are no new genetic factors influencing general disordered eating symptoms from age 14 through age 18 [10]. Explanations for these developmental shifts have frequently focused on puberty, where analyses from two twin registries (MTFS) [12] (Michigan State University Twin Registry; MSUTR) [13] suggest that puberty moderates genetic effects on disordered eating, with no

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genetic influence in early puberty but significant genetic effects in middle- to post-puberty [14-16].

These findings have been important for identifying developmental stages in which genetic effects may become more pronounced. This information has, in turn, narrowed hypotheses regarding regulators of genetic effects (e.g., ovarian hormones) [17] and potential candidate genes [18]. Nonetheless, much more information is needed to fully characterize the developmental trajectories of genetic and environmental influences on disordered eating. For example, we currently have no data beyond the adolescent period. Thus, it is unclear if the magnitude of genetic and environmental influences remains constant from late adolescence (a peak time of eating disorder risk) through young and middle adulthood.

We also have little information about developmental changes in different types of disordered eating. One critical type of disordered eating is weight and shape concerns (i.e., concern about the size or shape of one's body, preoccupation with body weight, etc.). Weight and shape concerns contribute to cognitive symptoms of eating disorders (e.g., fear of fatness, misperceptions of body shape, importance of weight and shape on self-evaluation) [19] and have been shown to be strong prospective risk factors for eating disorders [20]. This strong prospective relationship likely explains the emphasis on weight/shape concerns in etiological [21] and treatment [22] conceptualizations of the disorders.

Despite this emphasis, few studies have examined age differences in genetic and environmental influences for these symptoms. Initial research comparing genetic and environmental effects across age suggested low heritability of weight concerns in pre-adolescent twins [4] but significant heritability (>50%) in adolescent twins [4]. This general pattern has been replicated in studies examining one age group only, which tend to show moderate heritability of weight and/or shape concerns in late adolescence and young adulthood [23-25]. However, others have found lower heritability of weight and shape concerns and related symptoms (i.e., the undue influence of weight and shape on self-evaluation) [9,26,27], particularly during middle adulthood [21,26]. These discrepant results cannot be due entirely to differences in assessment instrument across studies (e.g., weight/shape concerns measured with the Minnesota Eating Behavior Survey versus the Eating Disorders Examination Questionnaire (EDE-Q)), as different results have been obtained when using the same family of assessment instruments (i.e., the EDE and EDE-Q) (see Wade and Bulik, 2007 and Spanos, Burt and Klump, in press) [21,23]. This has led some to speculate that discrepant findings are actually due to age/developmental differences in etiologic effects rather than measurement error per se [23].

The purpose of our study was to directly examine this possibility by investigating age differences in etiologic effects on weight and shape concerns. We combined data across three twin registries (i.e., the MTFs, the MSUTR, and the Australian Twin Registry (ATR) [28]) to investigate cross-sectional age differences in genetic and environmental effects in 2,618 same-sex female twins who were pre-adolescence, adolescence, young adulthood, or middle adulthood. Our study was the largest to date to examine age differences in etiologic effects and was the first to examine middle adulthood.

## Methods

### Participants

Participants included 2,618 female twins (1,624 MZ, 994 DZ) from the MSUTR, the MTFs, and the ATR. Data from all three registries were collected during a similar time period (i.e., 1996-2007), particularly the MSUTR and ATR data (2000-2007). Although recruitment procedures vary somewhat by registry, all are population-based with the majority of twins being Caucasian (>82%) and of middle socioeconomic class [12,28,29]. The MSUTR and

MTFS twins are broadly representative of their respective regions in terms of ethnicity and socioeconomic class [12,29]. Likewise, only limited sampling bias has been noted in the ATR for self-report questionnaires [28]. Although the MSUTR and MTFS have been described in detail elsewhere [12,13] the cohort of ATR [28] twins included in this study have not been reported upon previously. Participation rates (77%) and demographic characteristics were excellent for this new cohort.

Twins were between the ages of 10 and 41 years ( $M = 21.99$  years;  $SD = 7.76$  years). Given the wide range of ages, we divided the sample into 6 age groups for analysis (see Table 1). We divided pre-adolescent and adolescent twins into three groups comprised of 10-12 year-old, 13-15 year-old, and 16-19 year-old twins that were identical to twin groups examined in previous developmental studies of pre-adolescent and adolescent twins [4,10]. This similarity allowed us to directly examine whether weight/shape concerns show similar developmental differences to those observed for overall disordered eating. We then divided the remaining twins into three additional groups comprised of 20-25 year-old, 26-30 year-old, and 31-41 year-old twins. These groupings were based on previous research examining peak periods for eating disorder symptoms [30-32] and sample size considerations. The peak period of onset for eating disorder symptoms extends to age 25 and then levels off [30-32]; thus, we used 25 years as the cut-off for twins in their early 20s. We grouped ages 26-30 and 31-41 years separately to capture theoretically different developmental stages (e.g., late 20s versus mid-30s). Moreover, sample sizes were largest for twins in their early-to-mid 30s and relatively small for twins over 35 years ( $n = 55$  twins). Thus, we used 30 years as the cut-off for the oldest group. Notably, results remained unchanged when twins over the age of 35 were excluded from analyses (data not shown), suggesting that our inclusion of twins in their early and late 30s in the same group did not unduly influence results.

## Measures

**Demographic Questionnaire**—Twin age was assessed via self-report using standard demographic questionnaires.

**Zygosity Determination**—In all three registries, twin zygosity was established using standard physical similarity questionnaires that show accuracies of 95% or better (see Lykken, Bouchard, McGue and Telegen, 1990 and Peeter, Van Gestel, Vlietinck, Derom and Derom, 1998) [33,34]. These questionnaires were completed by either the twins and/or the mother, depending upon the age of the twin. In the MTFS and MSUTR, research assistants also frequently completed the questionnaire, and responses were compared for accuracy. Disagreements were resolved through either: 1) a review of questionnaire data and twin photographs (when available) by one of the principal investigators (MSUTR); or 2) by DNA markers (MTFS).

**Weight and Shape Concerns**—Weight and shape concerns were assessed with the Eating Disorders Examination Questionnaire (EDE-Q) [35]. The EDE-Q is a 36-item self-report questionnaire derived from the Eating Disorders Examination (EDE) [36] that assesses eating pathology over the past 28 days. The EDE-Q was the only measure of disordered eating that was shared among all three registries.

The EDE-Q includes a 7-item shape concerns subscale (i.e., assessing dissatisfaction and discomfort with body shape) and a 5-item weight concerns subscale (i.e., assessing preoccupation with weight and a desire to lose weight) whose items are rated on a 6-point scale (0 = not at all, 2 = slightly, 4 = moderately, 6 = markedly) and then averaged to create subscale scores. In adult populations, these EDE-Q scales correlate highly with scores from the EDE ( $r = .77-.80$ ) [37,38] and exhibit good internal consistency [39] and long-term (~315 days) test-

retest reliability [40]. Although the EDE-Q weight and shape concerns subscales have been studied less extensively in children and adolescents, initial data suggest that correlations with the EDE are high [37,41-44]. Moreover, in the MSUTR adolescent sample (ages 10-17 years) included in this report, the scales showed excellent internal consistency (alphas > .85) and exhibited high correlations ( $r = .66 - .69$ ) with the weight preoccupation and body dissatisfaction subscales from the Minnesota Eating Behavior Survey (MEBS) [45]. The MEBS was designed for use in girls ages 9 and older, and thus provides good evidence of convergent validity for the EDE-Q weight/shape concerns scales in our pre-adolescent and young adolescent twins.

Despite the strong psychometric properties of the EDE-Q, recent factor analyses in non-clinical samples have failed to identify two separate EDE/EDE-Q shape and weight concerns subscales [39,46-48]. Indeed, factor analytic work tends to identify one factor that includes most all of the items from these two subscales<sup>a</sup>. Not surprisingly then, the intercorrelation between the two subscales was very high ( $r = .87 - .95$ ) across all of our age groups. Given these data, we opted to combine items on the two subscales into a single weight/shape concerns scale for all analyses. This combined scale was scored the same way as the individual subscales, i.e., items were added then averaged to create the final score. Similar to findings for the separate weight/shape concerns scales, the combined scale showed excellent internal consistency across all twin registries and ages in our sample (see Table 2).

### Statistical Analyses

Intraclass twin correlations were used to provide initial indications of genetic and environmental influences on the weight/shape concerns subscale both within and across age. Cross-sectional, univariate twin models were then fit to the raw twin sum scores using the maximum likelihood option in Mx [49]. The raw data option in Mx treats missing data as missing at random. This treatment of missing data is expected to produce less biased and more consistent estimates than other techniques (e.g., pairwise or listwise deletion).

We used the cross-sectional models to estimate the relative contribution of additive 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) factors to EDE-Q scales across age. Because of the potentially large number of models that could be fit across the six age groups, we initially fit the fully unconstrained model that allows A, C, and E to vary across age. We then examined parameter estimates from this model to identify appropriate submodels that differentially constrained parameters across groups. This approach allowed us to test theoretically relevant submodels without unduly increasing the number of statistical tests. In addition to these submodels, we also fit the fully constrained model that constrains parameters to be equal across all ages, as this model is the most parsimonious and directly tests the null hypothesis (i.e., that there are no differences in A, C, and E across age). We present standardized (or proportional) parameter estimates for all models in the data tables. Following previous recommendations [50], however, we also report the unstandardized parameter estimates for the best-fitting model. These estimates more accurately depict absolute differences in genetic and environmental influences than standardized estimates which represent these differences as proportions of the total variance.

<sup>a</sup>Notably, in the current sample, we conducted an exploratory factor analysis and also obtained a one-factor solution that contained nearly all of the weight and shape concerns items (data not shown). These findings supported our decision to use a single combined scale and partially guided the focus of the current work on weight and shape concerns rather than other disordered eating constructs assessed by the EDE-Q (e.g., restraint, eating concerns, binge eating, purging, etc.). These other constructs appear to be less well-defined by the EDE-Q for our age groups, limiting their usefulness for the twin model-fitting analyses.

Model fit was determined by examining the difference in the minimized value of minus twice the log-likelihood ( $-2\ln L$ ) between nested models. This difference yields a likelihood-ratio  $\chi^2$  test that is used to test the significance of the more restrictive model. A nonsignificant  $\chi^2$  indicates that the more restrictive model provides an appropriate fit to the data. In addition, models that minimize Akaike's information criterion (AIC;  $AIC = \chi^2 - 2 * \Delta df$ ) [51] are preferred, as the AIC balances model fit with parsimony.

Importantly, before conducting the analyses described above, we first tested for twin registry differences in mean levels of disordered eating and genetic and environmental effects to ensure that findings reflect differences in age rather than twin registry. We used age regressed, standardized EDE-Q scores in these analyses to account for the fact that twin registry was somewhat confounded with age (i.e., the 10-12 group was from the MSUTR; the 13-25 group was from the MSUTR and MTFs; and the oldest two age groups were from the ATR; see Table 1). Mean differences in weight and shape concerns across registry were uniformly of small effect ( $d = .03-.12$ ; see Table 1 for raw means and SD), suggesting minimal differences in weight/shape concerns after controlling for age. Likewise, biometric model-fitting indicated no significant differences in additive genetic (.55; 95% CI = .35-.60), shared environmental (.00; 95% CI = .00-.18), or nonshared environmental (.45; 95% CI = .40-.50) influences on age-regressed weight and shape concern scores across the three twin registries (Unconstrained model AIC = 1617.35; Constrained model AIC = 1612.85). These findings strongly support our inclusion of data from all three registries and confirm that any observed differences in etiologic factors across age are not due to other differences across twin registries.

## Results

### Descriptive Statistics

Descriptive statistics are presented in Table 2. Although variability in weight/shape concerns were present for all age groups, a one-way analysis of variance (ANOVAs) with Tukey's B post hoc t-tests revealed lower mean scores in the two youngest age groups (10-12; 13-15) as compared to the older groups (i.e., 16-19, 20-25, 26-30, and 31-41). These findings corroborate those previously showing the largest increase in eating disorder symptoms during adolescence [30-32] and relative stability in symptoms from adolescence on [30-32]. Notably, these mean differences do not affect our ability to examine age differences in genetic and environmental influences, as the later uses the covariation between co-twins (i.e., similarity between twin 1 and twin 2) to estimate etiologic effects rather than individual twin means.

### Twin Intraclass Correlations

Twin intraclass correlations are presented in Table 3. The pattern of correlations suggests developmental differences in genetic and environmental effects. In the youngest (i.e., ages 10-12) age group, the MZ twin correlation was not significantly different from the DZ correlation, indicating little genetic influence but significant shared and nonshared environmental effects. By contrast, in ages 13-30, the MZ/DZ twin correlation differences tended to be larger and reach or almost reach significance. These results suggested greater heritability of weight and shape concerns from mid-adolescence through young adulthood. Finally, in the oldest age group, MZ twin correlations were larger than DZ correlations, but differences were more modest, suggesting that heritability may be somewhat lower in this age group.

### Biometric Model-Fitting

Standardized parameter estimates from the fully unconstrained model suggested substantial changes in genetic and shared environmental effects across age (see Table 4), with modest genetic influence at ages 10-12 years, but significant genetic effects from ages 13-30 years.



Genetic effects were somewhat lower again at ages 31-41, although the decrease was not as dramatic as that observed in the 10-12 year-old age group. Overall, shared environmental factors tended to show the opposite pattern of effects (highest at the youngest age), while nonshared environment changed much less across age.

Based on these results, we fit three submodels to the data: 1) a model that constrained A, C, and E across ages 13-30; 2) a model that constrained A, C, and E across ages 13-41; and 3) the fully constrained model that constrained A, C, and E across all ages. Model fit comparisons indicated that the best fitting model was one in which A, C, and E were constrained to be equal across ages 13-41, but were allowed to vary in the 10-12 year old group. This model had the lowest AIC value, and the chi-square difference tests comparing the model to the fully unconstrained model was non-significant (see Table 4). Standardized (or proportional; see Table 4) and unstandardized (or absolute; see Figure 1) parameter estimates from this model indicated that shifts in additive genetic and shared environmental influences primarily account for these age differences. In the 10-12 year old age group, genetic influences were nominal, while shared and nonshared environmental factors accounted for substantial proportions of variance. By contrast, at ages 13-41, additive genetic and nonshared environmental influences predominated with little-to-no shared environmental influence.

## Discussion

Our study is the first to examine age differences in genetic and environmental effects on weight and shape concerns from pre-adolescence through middle adulthood. We found evidence for modest genetic influences in pre-adolescence and significant genetic effects from early adolescence through middle adulthood. Shared environmental factors tended to show the opposite pattern, with the largest shared environmental contributions occurring in the youngest age groups. Finally, nonshared environmental factors remained relatively constant across age groups. Overall, our findings underscore the likely importance of developmental changes in etiologic effects for weight and shape concerns and their potential role in the development of eating disorders.

Our data are significant in showing that the pre-adolescent period is marked by low genetic risk for core weight and shape concerns of eating disorders. These findings extend previous work by showing that in addition to developmental changes in general disordered eating (i.e., sum scores that include binge eating, compensatory behavior, body dissatisfaction, and weight concerns) (see Klump, McGue and Iacono, 2000 and Klump, Burt, McGue and Iacono, 2007) [4,10], key symptoms of eating disorders also show nominal genetic effects in pre-adolescence. Previous explanations for these effects have focused on puberty as an activating event for genetic risk for eating pathology [15-18]. This focus partially stems from extant data showing that ovarian hormones regulate gene transcription within several neurotransmitter systems known to be disrupted in eating disorders (e.g., serotonin) [18,52]. Thus, the activation of ovarian hormones at puberty may activate genetic risk for eating disorders through differential transcription of risk genes. New data indirectly implicate this process, as estrogen levels moderate genetic effects on disordered eating during puberty, with little-to-no genetic effects in girls with low estrogen levels, and significant genetic effects (>50%) in girls with moderate-to-high estrogen levels [53]. Unfortunately, pubertal stage information was not available for many of our 10-12 year-old twins and thus, we were unable to directly examine the effect of puberty on age differences. Nonetheless, it is likely that puberty may partially account for observed differences in genetic effects, and that puberty and ovarian hormones will be critically important mechanisms to examine in future twin and genetic research.

The lack of genetic effects for weight and shape concerns in pre-adolescent twins was countered by the effects of shared environment during this time period. This finding replicates those

previously for overall levels of disordered eating [4,10,15,16] and highlights shared environment as a risk factor for disordered eating during pre-adolescence. Shared environment has generally been ignored since the late 1980s when nonshared environmental factors were found to be most important for behavioral phenotypes in adulthood [54]. However, our results add to a growing literature suggesting that shared environment is critically important for the development of psychological symptoms in childhood [55], where a recent meta-analysis found shared environment to significantly contribute to the variance in externalizing and internalizing disorders in childhood [55]. These findings point to a need for studies identifying the factors that underlie these main effects of shared environment. Important shared environmental factors to examine in future research include (but are not limited to) cultural messages about thinness [56,57] and modeling of disordered eating behavior by family members [58]. The examination of these factors in genetically informative, pre-adolescent samples will significantly advance understanding of the development of eating disorders during this understudied developmental period.

We found moderate heritability for weight/shape concerns during adolescence and young adulthood. This period is one in which females experience the highest level of eating disorder risk [30-32]. Our data suggest that at least part of this increased risk is due to genetic factors that may “tip the scales” toward eating disorders, particularly in the presence of other risk factors (e.g., stressful life events, involvement in weight-focused peer groups). Importantly, although shared environmental influences were non-significant during this time, they may contribute to disordered eating through gene-environment interactions [55]. For example, exposure to cultural messages regarding beauty/thinness ideals may interact with genetic risk to increase susceptibility to eating disorders in some females relative to others. These types of gene  $\times$  shared environment interactions are partitioned into the additive genetic estimates within twin models, leading to increased heritability and decreased shared environmental variance, a pattern that is similar to that observed in our study (see Figure 1). Adolescence and young adulthood are therefore prime developmental periods for investigating genetic risk and gene  $\times$  shared environment interactions in the development of eating disorders.

Our study was the first to examine age differences in middle adulthood. Findings from the fully unconstrained model suggested that genetic factors decrease somewhat during this time period, while shared and nonshared environmental factors again become important. However, model fit comparisons suggested that these changes were not statistically significant, as the best fitting model was one which genetic and environmental factors could be constrained across late adolescent, young adult, and mid-adult groups (see Table 4). Given parameter estimates in Table 4, it is possible that a larger sample would have detected significant differences in etiologic effects. Future research should directly examine this possibility.

In the meantime, our data indicate few differences in the broad categories of etiologic factors (i.e., genetic and nonshared environment) contributing to weight/shape concerns in middle adulthood, adolescence, and young adulthood. Nonetheless, it is important to note that while the broad categories of risk may be the same across these developmental periods, the types of genetic and nonshared environmental factors that are important probably vary. For example, it is likely that nonshared environmental risk factors experienced by middle aged women are very different from those experienced by women during adolescence and young adulthood. Factors such as marital separations/divorces [59], stressors emanating from work (e.g., job loss or change of job) and/or family life (e.g., parent-child conflict) [59], the number of pregnancies [60] and physiological changes (e.g., weight changes) [59] are likely to be critical. Although some of these factors (e.g., physical changes) are partially under genetic control [61], their differential experience and/or timing could serve as nonshared environmental risk factors that contribute to weight concerns in one twin relative to another.

The results of this study should be interpreted within the context of its limitations. First, although our overall sample size was quite large, the size of some age groups was relatively small (e.g., the 10-12 age group, and twins over the age of 35). This resulted in broad confidence intervals that, at times, included 0 (e.g., see Table 4). Moreover, and as noted above, sample sizes likely impacted our ability to detect significant differences between some models. Additional studies with larger samples are needed to investigate this possibility. These studies should include a higher proportion of twins over the age of 35 in order to directly examine genetic and environmental factors that may be specific to the peri-menopausal and menopausal period.

Second, age was confounded with twin registry in the youngest and oldest age groups. However, initial analyses confirmed that there were no significant differences in mean levels of weight/shape concerns or genetic and environmental influences across registry after controlling for age. Moreover, data from all three registries were collected during a narrow and similar time period (i.e., 1996-2007); cohort effects would be unlikely to significantly impact the etiology of weight/shape concerns during such a relatively small window of time. Taken together then, these data suggest that our findings likely reflect true age differences in etiologic influences rather than simply registry or cohort effects.

Third, we were limited to the EDE-Q weight/shape concerns scale, which may have posed problems for our phenotypic measurement. For example, it would have been preferable to access interview-based measures as well as questionnaires to ensure that we captured the full range of weight/shape concerns. In addition, it would have been ideal to use latent scores in addition to EDE-Q sum scores in the model-fitting analyses, as sum scores can sometimes produce biased estimates of genetic and environmental effects [see Neale, Lubke, Aggen and Dolan, 2005 62]. Nonetheless, questionnaire measures [4,23-25] of eating disorder symptoms provide similar estimates of genetic influences as interviews [1,6,63,64], and sum scores tend to produce more biased estimates when binary items are used, scores are based on parental reports, and/or the phenotypes are directly observable by others (e.g., twin activity level) [62]. Given that our sum scores do not fit these criteria, it is unlikely that our findings were unduly influenced by assessment or scoring method.

A final limitation is that our data were cross-sectional rather than longitudinal in nature. Additional studies examining the same cohort of twins across time is needed to ensure that cross-sectional findings are robust and extend to within-twin changes in genetic and environmental influences.

In summary, our results underscore the importance of age differences in etiologic influences on weight and shape concerns. Future studies should improve upon our work by examining more extensive measures of shape/weight concerns and using cross-national, longitudinal samples that span childhood through middle and older adulthood. Such analyses will allow us to develop more comprehensive models of eating disorder risk that take into account the dynamic interplay of genes and environment across development.

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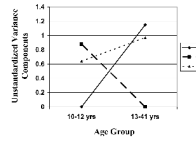
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**Figure 1. Unstandardized Estimates of Additive Genetic, Shared Environmental, and Nonshared Environmental Factors from the Best-Fitting Model for the Weight and Shape Concerns Scale**  
Abbreviations include: a = additive genetic effects; c = shared environment; e = nonshared environment.



**Table 1**  
**Sample Sizes by Twin Registry and Age Group (Total N = 2,618 twins from 1,100 complete and 209 incomplete pairs)**

Age Group	MSUTR			MTFS			ATR			Total		
	Total	MZ	DZ	Total	MZ	DZ	Total	MZ	DZ	Total	MZ	DZ
10-12 yrs	172	84	88	0	0	0	0	0	0	172	84	88
13-15 yrs	146	78	68	532	342	190	0	0	0	678	420	258
16-19 yrs	152	94	58	24	26	22	0	0	0	200	120	80
20-25 yrs	164	92	72	516	346	170	0	0	0	680	438	242
26-30 yrs	16	8	8	0	0	0	334	206	128	350	214	136
31-41 yrs	0	0	0	0	0	0	538	348	190	538	348	190
<b>Total</b>	<b>650</b>	<b>356</b>	<b>294</b>	<b>1,072</b>	<b>714</b>	<b>382</b>	<b>872</b>	<b>554</b>	<b>318</b>	<b>2,618</b>	<b>1,624</b>	<b>994</b>

Note. MSUTR = Michigan State University Twin Registry; MTFS = Minnesota Twin Family Study; ATR = Australian Twin Registry; MZ = monozygotic; DZ = dizygotic. Values are the number of twins per age category and twin registry.

**Table 2**  
**Internal Consistency and Descriptive Statistics for Weight and Shape Concerns by Twin Registry and Age Group**

Sample	Alphas	Means (SD)	Score Range
<u>Registry:</u>			
MSUTR	.94	1.70 (1.50)	.00-5.80
MTFS	.95	1.42 (1.43)	.00-6.00
ATR	.94	1.89 (1.52)	.00-6.00
<u>Age Group:</u>			
10-12 years	.92	1.19 (1.31) <sup>a</sup>	.00-4.60
13-15 years	.95	1.22 (1.39) <sup>a</sup>	.00-6.00
16-19 years	.95	1.78 (1.63) <sup>b</sup>	.00-5.75
20-25 years	.95	1.89 (1.51) <sup>b</sup>	.00-6.00
26-30 years	.94	1.95 (1.45) <sup>b</sup>	.00-5.80
31-41 years	.94	1.99 (1.51) <sup>b</sup>	.00-6.00

Note. MSUTR = Michigan State University Twin Registry; MTFS = Minnesota Twin Family Study; ATR = Australian Twin Registry. Means with different superscripts are significantly different from each other at  $p < .05$ .

\*\*\*  
 $p < .001$

**Table 3**  
**Intraclass Twin Correlations for the Weight and Shape Concerns Subscale**

Age Group	Twin Correlation	Test of Equality	
		<i>z</i>	One-tailed <i>p</i>
<u>10-12 years</u>			
MZ ( <i>n</i> = 31 pairs)	.54***	.21	.42
DZ ( <i>n</i> = 30 pairs)	.51**	--	--
<u>13-15 years</u>			
MZ ( <i>n</i> = 181 pairs)	.52***	3.44	.0003
DZ ( <i>n</i> = 115 pairs)	.16*	--	--
<u>16-19 years</u>			
MZ ( <i>n</i> = 56 pairs)	.59***	1.52	.06
DZ ( <i>n</i> = 28 pairs)	.30 <sup>†</sup>	--	--
<u>20-25 years</u>			
MZ ( <i>n</i> = 199 pairs)	.51***	3.34	.0004
DZ ( <i>n</i> = 110 pairs)	.16*	--	--
<u>26-30 years</u>			
MZ ( <i>n</i> = 60 pairs)	.59***	1.16	.054
DZ ( <i>n</i> = 38 pairs)	.32*	--	--
<u>31-41 years</u>			
MZ ( <i>n</i> = 117 pairs)	.59***	1.41	.079
DZ ( <i>n</i> = 45 pairs)	.40**	--	--

Note. MZ = monozygotic; DZ = dizygotic. The "Test of Equality *z*" tests for differences between the MZ and DZ twin correlations. Only pairs with complete data for both twins were included in the analysis.

<sup>†</sup> *p* < .10,

\* *p* < .05,

\*\* *p* < .01,

\*\*\* *p* < .001.

The twin correlation is greater than zero.

Table 4

Parameter Estimates

Parameter Est.	Models			
	Fully Unconstrained	Constrain ACE Ages 13-30 <sup>a</sup>	Constrain ACE Ages 13-41 <sup>b</sup>	Fully Constrained <sup>c</sup>
<u>10-12 years</u>				
A	.00 (.00-.44)	.00 (.00-.44)	.00 (.00-.44)	.54 (.38-.59)
C	.58 (.12-.73)	.58 (.12-.73)	.58 (.12-.73)	.00 (.00-.15)
E	.42 (.27-.64)	.42 (.27-.64)	.42 (.27-.64)	.46 (.41-.51)
<u>13-15 years</u>				
A	.52 (.30-.61)	.49 (.30-.48)	.54 (.40-.59)	--
C	.00 (.00-.18)	.03 (.00-.20)	.00 (.00-.13)	--
E	.48 (.39-.59)	.48 (.42-.55)	.46 (.41-.51)	--
<u>16-19 years</u>				
A	.62 (.09-.75)	--	--	--
C	.00 (.00-.44)	--	--	--
E	.38 (.25-.57)	--	--	--
<u>20-25 years</u>				
A	.51 (.28-.60)	--	--	--
C	.00 (.00-.20)	--	--	--
E	.49 (.40-.60)	--	--	--
<u>26-30 years</u>				
A	.50 (.00-.70)	--	--	--

Models					
Estimates/Model Fit Statistics	Fully Unconstrained	Constrain ACE Ages 13-30 <sup>a</sup>	Constrain ACE Ages 13-41 <sup>b</sup>	Fully Constrained <sup>c</sup>	
C	.07 (.00-.55)	--	--	--	
E	.43 (.30-.61)	--	--	--	
<b>31-41 years</b>					
A	.28 (.00-.65)	.28 (.00-.65)	--	--	
C	.27 (.00-.60)	.27 (.00-.60)	--	--	
E	.45 (.35-.57)	.45 (.35-.57)	--	--	
<b>Model Fit:</b>					
-2lnL (df)	7732.09 (2217)	7756.79 (2225)	<b>7749.98 (2229)</b>	7762.35 (2232)	
$\Delta\chi^2$ (df)	--	33.70 (8)	<b>17.89 (12)</b>	30.26 (15)	
<i>p</i>	--	<.001	<b>.12</b>	.01	
AIC	3298.09	3306.79	<b>3291.98</b>	3298.35	

Note. A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; -2lnL = -2 times the log likelihood;  $\Delta\chi^2$  = the difference in -2lnL values between the fully unconstrained and the nested constraint models; AIC = Akaike's Information Criteria. Twins from complete and incomplete pairs were included in the analysis. The best-fitting model is noted in bolded text.

<sup>a</sup>Parameter estimates are constrained to be identical from ages 13-31; thus, these estimates are only listed for the age 13-15 group.

<sup>b</sup>Parameter estimates are constrained to be identical from ages 13-41; thus, these estimates are only listed for the age 13-15 group.

<sup>c</sup>Parameter estimates are constrained to be identical across all ages; thus, these estimates are only listed for the age 10-12 group.