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Genetic and Environmental Influences on Thin-Ideal Internalization across Puberty and Pre-Adolescent, Adolescent, and Young Adult Development

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Abstract

Objective—Mean-levels of thin-ideal internalization increase during adolescence and pubertal development, but it is unknown whether these phenotypic changes correspond to developmental changes in etiological (i.e., genetic and environmental) risk. Given the limited knowledge on risk for thin-ideal internalization, research is needed to guide the identification of specific types of risk factors during critical developmental periods. The present twin study examined genetic and environmental influences on thin-ideal internalization across adolescent and pubertal development.

Method—Participants were 1,064 female twins (ages 8–25 years) from the Michigan State University Twin Registry. Thin-ideal internalization and pubertal development were assessed using self-report questionnaires. Twin moderation models were used to examine if age and/or pubertal development moderate genetic and environmental influences on thin-ideal internalization.

Results—Phenotypic analyses indicated significant increases in thin-ideal internalization across age and pubertal development. Twin models suggested no significant differences in etiologic effects across development. Nonshared environmental influences were most important in the etiology of thin-ideal internalization, with genetic, shared environmental, and nonshared environmental accounting for approximately 8%, 15%, and 72%, respectively, of the total variance.

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DISCLOSURE OF CONFLICTS

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Discussion—Despite mean-level increases in thin-ideal internalization across development, the relative influence of genetic versus environmental risk did not differ significantly across age or pubertal groups. The majority of variance in thin-ideal internalization was accounted for by environmental factors, suggesting that mean-level increases in thin-ideal internalization may reflect increases in the magnitude/strength of environmental risk across this period. Replication is needed, particularly with longitudinal designs that assess thin-ideal internalization across key developmental phases.

Keywords

Thin-ideal; thin-ideal internalization; body image; twin study; behavior genetics; risk factors; developmental risk

Thin-ideal internalization (i.e., the acceptance of and adherence to sociocultural beauty ideals for women that focus on thinness) is an important risk factor in the development of body dissatisfaction, disordered eating, and eating disorders (see 1; 2 for reviews of this literature.). Cross-sectional and prospective studies have supported the role of thin-ideal internalization in the development of eating problems (e.g., body dissatisfaction, dieting; 1.), and eating disorder prevention programs that target thin-ideal internalization have been effective in decreasing disordered eating (3.). However, as noted by others (4.), prevention programs could, and should, be improved further, particularly given their potential to decrease the development of eating disorders. Increasing knowledge on the etiology of thin-ideal internalization may be key to developing additional intervention techniques.

Unfortunately, risk factors for thin-ideal internalization have been studied less frequently than risk factors for eating pathology (5–8.). Existing research has focused on the role of environmental risk factors that are thought to teach and reinforce beauty ideals of thinness, such as media images, parental and peer influences (5; 6.) Although research has demonstrated that hypothesized media, peer, and parental risk factors are indeed associated with thin-ideal internalization (5; 6.), further research is needed to confirm the direction of these effects, as current studies are limited by cross-sectional designs.

In addition to environmental risk factors, twin research has demonstrated that genetic influences explain approximately 40% of the variance in thin-ideal internalization in a sample of post-pubertal adolescent and young adult twins (9.). Thus, genetic influences may explain why, despite almost ubiquitous exposure to the thin-ideal in Western countries, only *some* women ultimately internalize this ideal and go on to develop disordered eating behaviors (9.). More specifically, in the context of environmental risk factors (e.g., thin-ideal focused media) that nearly all women within Western culture experience, it may be level of genetic risk for thin-ideal internalization that differentiates those women who go on to internalize these ideals, and those who do not.

Given that only one twin study of thin-ideal internalization has been conducted, further research is needed to extend knowledge of genetic and environmental effects. In particular, it is necessary to examine etiologic effects across adolescence, a key period for the development of thin-ideal internalization. Indeed, mean levels of thin-ideal internalization have been shown to increase across adolescence (9; 10.). The pubertal period appears to be

particularly important in this regard, as girls in pre-to-early puberty report significantly lower levels of thin-ideal internalization than girls in mid-puberty and beyond (11.). These increases in mean levels of thin-ideal internalization may indicate key etiological shifts that should be examined as well. Indeed, related phenotypes, such as disordered eating, show significant etiological changes across this period, such that the heritability of disordered eating is negligible in pre-adolescence and pre-pubertal twins, but is significant (i.e., approximately 50% of variance) in pubertal twins and in twins who are in middle adolescence (i.e., about age 14) or older (i.e., ages 16–40 years; 12; 13; 14.). The effects of the shared environment are opposite of those observed for genetic influences: shared environmental influences account for 40% of the variance in disordered eating in pre-adolescence/pre-puberty, and 10% or less from middle adolescence into middle adulthood and in pubertal twins. These findings have been useful because they have led researchers to develop specific hypotheses regarding *mechanisms* that may account for differences in heritability across puberty, such as changes in ovarian hormones during puberty (15.). Given that adolescence also seems to be a key developmental period for thin-ideal internalization (11.), developmental twin studies of thin-ideal internalization may help elucidate etiological mechanisms that contribute to risk for thin-ideal internalization across development.

The aim of the present study was to investigate the extent to which genetic and environmental influences on thin-ideal internalization differ across age and pubertal development in a large (N=1,064) sample of same-sex female twins (ages 8–25 years). To ensure that effects are specific to thin-ideal internalization, we examined developmental differences in genetic and environmental effects while controlling for disordered eating. Specificity of effects are important to establish given phenotypic and genetic overlap in thin-ideal internalization and disordered eating (16–18.) and the need to identify etiological risk factors that contribute uniquely to thin-ideal internalization.

METHOD

Participants

Participants were 1,064 same-sex female twins between the ages of 8 and 25 ($M = 15.06$, $SD = 3.93$) from the Michigan State University Twin Registry (MSUTR; 19; 20.). The MSUTR is a population-based registry that recruits twins through the use of birth records in collaboration with the Michigan Department of Community Health (Further details are available elsewhere; 19; 20.). Twins included in this study were participants in one of two ongoing studies within the MSUTR (i.e., the *Twin Study of Hormones and Behavior across the Menstrual Cycle* and the *Twin Study of Hormones and Disordered Eating Across Puberty*). These projects have both been reviewed and approved by an institutional review board, and participation in the studies involved informed consent/assent. Both studies have primary aims involving the investigation of ovarian hormone influences on disordered eating. As a result, several inclusion/exclusion criteria were applied to ensure accurate sampling of hormones (e.g., no psychotropic or steroid medication use; no pregnancy or lactation, regular menstrual cycles in participants ages 16+). Prior investigations have indicated that the use of these inclusion/exclusion criteria do not inadvertently affect the range or variability in thin-ideal internalization scores (9.). Participants from the MSUTR

have been shown to be representative of the population from which they were drawn in terms of racial and ethnic background (i.e., 83% Caucasian; 20; 21; 22.). Notably, 29% of participants in the current study were also included in our prior twin study on thin-ideal internalization (6).

Measures

Zygosity Determination—Twin zygosity was determined using a physical similarity questionnaire that has been shown to be over 95% accurate when compared to genotyping (23; 24.). To assess zygosity, research assistants independently completed the physical similarity questionnaire for the twin pair. The questionnaire was also completed by the twins' parent (usually the mother) for all twins under age 16, and in approximately 41% of twin pairs age 16 or older. Additionally, twins age 16 or older each completed a self-report version of the zygosity questionnaire. Discrepancies in zygosity status among raters were resolved using questionnaire responses, photographs of the twins, and DNA (i.e., twin concordance across several single-nucleotide polymorphisms) were examined by study principal investigators to determine final zygosity status.

Thin-Ideal Internalization—Internalization of the thin-ideal was assessed with a modified version of the general internalization scale of the Sociocultural Attitudes toward Appearance Questionnaire-3 (SATAQ-3; 16.). The original SATAQ-3 includes 30 items assessed on a 5-point Likert scale (ranging from definitely disagree to definitely agree) that load onto one of four subscales (i.e., general internalization, athlete internalization, pressures, and information). The general internalization subscale was used in the prior twin study on thin-ideal internalization (9.) and is commonly used to assess thin-ideal internalization in risk factor and intervention studies (25; 26.). This subscale has demonstrated excellent reliability and validity in older adolescent and young adult samples, as it differentiates individuals with eating disorders from controls and demonstrates excellent internal consistency in prior samples (α 's >.90; (16; 17.).

Importantly, the SATAQ-3 has eight items (items 3, 6, 9, 12, 13, 19, 27, and 28) that are worded negatively (e.g., “I do not care if my body looks like the body of people who are on TV”) (27.). Although the original recommendations for the measure stressed including these negatively worded items (16.), later work suggested that the reverse-scored items may form a method factor on the SATAQ-3 and may exhibit smaller loadings on the SATAQ-3 factors (27.). In order to examine the functioning of the negatively worded items in the current sample, we investigated the factor structure of the general internalization scale and conducted factor invariance analyses (via exploratory structural equation models (ESEM); see (28.) for the SATAQ-3. As described by others (27.), ESEM models are particularly useful for scales with reverse-scored items, as ESEM allows for the a priori specification of correlated uniqueness among these types of items. Moreover, ESEM allows for specific tests of factor invariance, a feature that is not available in some other types of analyses (e.g., exploratory factor analysis, [EFA]). ESEM models were conducted using Mplus version 7, and models accounted for the non-independence of the twin data using the “Complex” options in Mplus (29.).

We examined invariance across groups by first fitting a baseline model, where only the number of factors is constrained to be equal across groups. We then fit a series of models that impose additional constraints (e.g., factor loadings, item uniqueness, variance-covariance), and invariance is inferred when additional constraints do not result in a significantly worse fit as compared to the baseline model. In order to examine model fit, we examined the root mean squared error of approximation (RMSEA), the Tucker-Lewis Index (TLI), and the comparative fit index (CFI). Generally, RMSEA values $< .05$ reflect a close fit to the data, and TLI and CFI values $> .95$ suggest an excellent fit. Invariance is generally supported if TLI and CFI do not decrease by more than $.01$ in the invariance compared to baseline model (27.).

We initially tested the original factor structure of the SATAQ-3 (i.e., the 4-factor solution for the 30-item questionnaire) and found some evidence for factor loading invariance across age and pubertal groups. Indeed, fit was fair in each age and pubertal group when examined separately (i.e., RMSEA = $.03-.05$, TLI = $.926-.971$, CFI = $.950-.985$), and then were minimal decreases in fit (i.e., TLI and CFI decreased by $< .01$) when constraining factor loadings to be equal across groups. Despite this relatively good fit, the factor loadings for the negatively worded items were small ($< .20$) and non-significant, particularly in the pre-pubertal and 8–12 year-old age groups. We then examined fit using the 4-factor solution identified in a sample of adolescents that focused on a 19-item version of the SATAQ-3, (30.). A similar pattern of results emerged, whereby fit was fair (i.e., RMSEA = $.025-.050$, TLI = $.925-.981$, CFI = $.950-.990$) and some evidence for factor loading invariance emerged (i.e., decreases in TLI and CFI $< .01$). However, again, factor loadings of the negatively worded items were small and non-significant, particularly in younger twins.

Given the marginal fit of the original and 19-item scale in our sample, we conducted a new factor analysis of the 30 items, using ESEM, to identify a core group of items that performed best across age and puberty status. We estimated a 4-factor solution and again found that the eight negatively worded items did not load onto any factor (all factor loadings $< .20$), even when allowing for correlated uniqueness among negatively worded items (27.). Thus, we removed the reverse-scored items from the questionnaire, as well as an additional five items (items 1, 4, 8, 16, and 22) that had substantial cross-loadings ($> .30$) on two or more factors. This resulted in a 17-item questionnaire with the same four factors as the original SATAQ-3 (i.e., general internalization, pressures, information, and athlete internalization). The 17-item questionnaire provided a good fit to each age and pubertal group, and investigations of invariance across age and pubertal groups provided strong support for factor loading invariance. The baseline model that required only the same number of factors in each group provided a good fit to the data for both age (RMSEA = $.044$, TLI = 0.960 , CFI = $.978$) and puberty (RMSEA = $.041$, TLI = 0.964 , CFI = $.981$). Constraining factor loadings to be equal across age and between pubertal groups did not worsen fit for age (RMSEA = $.039$, TLI = 0.969 , CFI = $.975$) or puberty (RMSEA = $.038$, TLI = 0.969 , CFI = $.977$), suggesting that the 17-item version of the SATAQ-3 demonstrates factor loading invariance. There also was some support for item-intercept and item uniqueness invariance across age and pubertal groups, as evidenced by minimal decreases in TLI ($< .01$), although CFI did worsen by between $.01$ to $.02$ when adding these additional constraints.

Given the superior factor structure and invariance of this 17-item questionnaire, we focused our analyses on the general internalization scale that emerged from this shortened measure. This 3-item scale included items 7 ["I would like my body to look like the models who appear in magazines"], 11 ["I would like my body to look like the people who are in movies"], and 15 ["I wish I looked like the models in music videos"]. Internal consistency was excellent for this scale overall ($\alpha = 0.87$) as well as in each individual age and puberty group (α 's = .84–.87). Moreover, the new 3-item scale correlated significantly with the original 9-item scale ($r = .87, p < .01$) and the 4-item scale suggested by Wilksch & Wade (2012) ($r = .95, p < .01$). The new 3-item measure also correlated significantly with expected measures (i.e., disordered eating, age, BMI, puberty, see Table 1).

Disordered Eating—As described above, we examined possible moderating effects of age and/or puberty on thin-ideal internalization while controlling for disordered eating. Overall levels of disordered eating were assessed using the total score of the Minnesota Eating Behavior Survey (MEBS) (31.)¹ The MEBS is a 30-item true/false questionnaire that includes items regarding weight preoccupation (i.e., tendency to think about/be concerned with one's weight), body dissatisfaction (i.e., dissatisfaction with body weight and/or shape), binge eating (i.e., actual binge eating or thoughts of binge eating), and compensatory behaviors (i.e., the use of, or thoughts of the use of, excessive exercise, vomiting, laxatives, or other medicines in order to change weight or shape). Previous investigations of psychometric properties of the MEBS suggest high internal consistency in twins as young as 11 ($\alpha = .86$) through late adolescence ($\alpha = .89$) (31.). Internal consistency was also excellent in the current study, even in our youngest (ages 8–12) age group ($\alpha = .87$), as well as in older age groups ($\alpha = .87$ –.88). Additionally, 3-year test-retest reliability for the MEBS total score is also good (12.). Finally, women with eating disorders score significantly higher on the MEBS scales than control women (12; 31.). Of note, the total score of the MEBS is the same scale used in the majority of the previously discussed developmental studies of disordered eating, which found significant moderation of disordered eating by age and pubertal development (12; 21; 32; 33.). Thus, this scale is particularly useful since we aim to examine whether findings for thin-ideal internalization are independent of previously identified effects for disordered eating.

Pubertal Development—Twins from the *Twin Study of Hormones and Disordered Eating Across Puberty* were between the ages of 8–15 at the time of study participation and thus, range from pre-pubertal to post-pubertal development. To assess each participant's degree of pubertal development, the Pubertal Development Scale (PDS; 34.), was completed by each twin, which is the same scale used to assess puberty in prior developmental studies of changes in the heritability of disordered eating (e.g., 13.). The PDS is a self-report measure that assesses the extent to which participants have experienced physical markers of puberty (i.e., body hair growth, growth spurt, breast changes, skin changes, and onset of

¹The MEBS (previously known as the Minnesota Eating Disorder Inventory [M-EDI]) was adapted and reproduced from "Development and Validation of a Multidimensional Eating Disorder Inventory for Anorexia and Bulimia Nervosa" by D. M. Garner, M. P. Olmstead, and J. Polivy, 1983, *International Journal of Eating Disorders*, 2, by special permission of Psychological Assessment Resources, 16204 North Florida Avenue, Lutz, FL 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2). Copyright 1983 by Psychological Assessment Resources. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources.

menarche). Participants indicate whether development for each physical marker (1) *has not yet begun* (2) *has barely started* (3) *is definitely underway* or (4) *seems completed*. Menarche is rated as present (4) or absent (1). Prior research with this scale has indicated excellent reliability and validity (21; 34.), and it was also excellent in the current sample (.83). As in prior research, a PDS total score, indicating overall pubertal development, was computed by summing and computing an average score across all items, including menarche.

All twins from the *Twin Study of Hormones and Behaviors across the Menstrual Cycle* were age 16 or older and were required to be experiencing regular menstrual cycles to participate in the study. Since all participants were post-pubertal, they did not complete a measure of current pubertal development during study participation. Thus, all women from this study were assigned a maximum PDS total score (i.e., 4) for statistical analyses.

Body Mass Index (BMI)—BMI was used as a covariate in the present study (see Statistical Analyses), and was calculated ($[\text{weight}] / [\text{height}]^2$) from height and weight assessed by research assistants. Height was measured using a wall-mounted ruler or a tape measure. Weight was measured using a digital scale in all participants.

Statistical Analyses

Data Preparation—To ensure that results were not unduly influenced by disordered eating or BMI, we partialled out MEBS total scores and BMI from each twins' thin-ideal internalization score prior to analyses. To ease interpretation of results, age and puberty scores were “floored”, prior to analyses, such that the minimum score was 0 for both age and puberty.

Missing data were minimal for general internalization ($N = 100$, 8.7%), disordered eating ($N = 11$, 1%), pubertal group status ($N = 14$; 1.2%), and BMI ($N = 4$; <0.1%). The twin analyses used in the present study were conducted using the raw data option of the statistical program Mx (35.), which treats missing data as missing at random (36.), and thus the parameter estimates and fit indices are appropriately adjusted, and twin pairs are retained in analyses even if one member of the pair is missing data. Final age moderation models included 1,064 twins (92% of the original sample) (Monozygotic [MZ] = 568 (53%), dizygotic [DZ] = 496 (47%), and final pubertal moderation models included 1,050 twins (91% of the original sample) (MZ = 560 (53%), DZ = 490 (47%).

For analyses examining pubertal moderation effects, pubertal development was dichotomized into a pre/early puberty and pubertal group using a PDS cut-off of 2.5, as has been used in prior research on differences in heritability across puberty (21; 32.). For age analyses, categories were developed to closely match age groups examined in prior papers of disordered eating while still maintaining reasonable sample sizes in each group. This resulted in three age categories: 8–12 years, 13–16 years, and 17–25 years (13; 14.)²

²We also examined alternate scoring algorithms for the general internalization scale (i.e., original 9-item version, and 4-item version [Wilksch & Wade, 2012]), as well as the 3-item version used herein with no covariates. In all cases, model-fitting results were consistent with those reported herein, and also suggested minimal significant moderation effects.

Twin Moderation Models—Differences in the etiology of thin-ideal internalization across age and pubertal development were evaluated using twin moderation models. We used standard univariate moderation models to examine possible moderating effects of age on the etiology of thin-ideal internalization, as these are the recommended models when the moderator is a family-level variable that is shared within twin pairs, as is true for age (37; 38.). For puberty moderation models, we used the extended univariate moderation model (37.). This model is identical to the standard univariate moderation model that was used for age analyses, except that the model also accounts for co-twin covariance on the moderator (i.e., in this case, twin pair covariance on pubertal status, $r = .94$, $p < .01$). It is necessary to use the extended univariate model for examination of pubertal moderation because simulation studies have demonstrated substantial increases in false positive moderation results if co-twin covariance on the moderator is *not* accounted for (37.). As previously recommended (37.), before proceeding with the extended univariate models, we first confirmed that there was no significant genetic (genetic correlation = -1.0 ; 95% CI [-0.52 , 1.0]), shared environmental (shared environmental correlation = $.23$; 95% CI [$-.19$, $.66$]), or nonshared environmental (nonshared environmental correlation = $-.04$, 95% CI [$-.19$, $.12$]) covariance between the moderator (puberty) and outcome (thin-ideal internalization).

Within the standard and extended univariate moderation models, three nested moderation models are fit. The most restrictive baseline “no moderation” model estimates standard additive genetic (A), shared environmental (C), and nonshared environmental (E) path estimates *without* consideration of the moderator (i.e., a, c, and e; see Figures 1 and 2). The second model allows for linear genetic, shared, and nonshared moderating effects of age/puberty by adding linear moderation terms to the model (i.e., β_X , β_Y , and β_Z ; see Figure 1). The third and least restrictive model (i.e., “full” model) also adds quadratic moderation terms to the model (i.e., β_X^2 , β_Y^2 , and β_Z^2 ; see Figure 1), allowing for linear and quadratic genetic, shared, and nonshared environmental moderating effects of age/puberty. Importantly, in analyses of pubertal groups, we present only “no moderation” and “linear moderation” model results, as quadratic moderation would not be possible in a two-group model.

Model fit for age analyses was determined by comparing the no moderation and linear moderation models to the least restrictive full moderation model (which allows for linear *and* quadratic moderation). For pubertal analyses, the least restrictive linear moderation model was compared to the no moderation model. Specifically, the minimized value of minus twice the log likelihood ($-2\ln L$) in the least restrictive model(s) (i.e., the linear/quadratic moderator model for age, the linear model for puberty) were compared with the $-2\ln L$ value obtained in the most restrictive model. This comparison yields a likelihood-ratio chi-square test for the significance of the moderator effects. Additionally, Akaike’s Information Criteria (AIC; (39.)) and the Bayesian Information Criterion (BIC; (40.)), which evaluate model fit relative to model parsimony, were also used to indicate model fit. The best-fitting model is selected by identifying the model in which AIC and BIC are the smallest. Both AIC and BIC are commonly used in behavioral genetic analyses (41.), and each have their strengths and weaknesses. Simulation studies have demonstrated that BIC is particularly likely to outperform AIC when sample sizes are large, there are a large number

of variables in the model (e.g., 8+ variables), and/or the models are particularly complex (41.). Given that our sample size is relatively moderate, we examined both AIC and BIC, in addition to the likelihood-ratio chi-square. Consistent with previous recommendations, the moderator models were run a minimum of 5 times using multiple start values to ensure that the obtained estimates minimize the $-2\ln L$ value (38.).

RESULTS

Descriptive Statistics

Means and variances of thin-ideal internalization, disordered eating, age, and pubertal development are presented in Table 1. Consistent with prior work (1; 11.), thin-ideal internalization was positively and significantly correlated with age, pubertal development, disordered eating, and BMI. Importantly, associations between thin-ideal internalization and age/pubertal development remained statistically significant, and relatively unchanged, even after partialling out levels of disordered eating (See Table 1), suggesting mean-level increases in thin-ideal internalization that are unique from disordered eating.

Twin Moderation Models

Age—Results from the twin models are summarized in Tables 2 and 3 and Figure 2. Path and moderator coefficients (see Table 3) were used to create plots of estimates for the full model (Figure 2a) and the best-fitting model (Figure 2b). As a reminder, although the results from the twin models presented in the plots are unstandardized estimates, thin-ideal internalization scores were standardized prior to analyses to ease interpretation of these unstandardized scores.

As shown in the plot of the full model (Figure 2a), there was some suggestion of moderating effects of age for genetic and shared environmental influences, where genetic effects were higher, and shared and nonshared environmental effects were lower, in the 13–16 and 17–25 year old age groups. However, in the full model, none of the moderation paths were statistically significant, suggesting that any moderation that may be present is likely relatively small and difficult to detect.

Given the lack of statistically significant moderating effects in the full model, it is not surprising that the best fitting model was the no moderation model, as indicated by the non-significant change in chi-square (all p 's > .71) and smallest AIC and BIC values as compared to the linear and full models (see Table 2). As shown in Figure 2, in the best-fitting no moderation model, there were no differences in the magnitude of genetic or environmental across age groups. Environmental influences consistently accounted for more variance in thin-ideal internalization scores than genetic influences. We again calculated standardized estimates (i.e., the proportion of variance accounted for by genetic, shared environmental, and nonshared environmental effects) by squaring the parameter estimates presented in Table 3. Across age all groups, standardized estimates for genetic influences were not statistically significant and accounted for only approximately 9% of the variance. Similarly, shared environmental estimates accounted for approximately 15% of the variance.

Nonshared environmental effects were statistically significant and estimated to account for approximately 75% of the variance in thin-ideal internalization scores.

Puberty—Results from the puberty moderation models are also summarized in Tables 2 and 3 and in Figure 2. We first examined the full model, which, given that we examined two groups for the puberty models, was the linear moderation model only (rather than quadratic moderation). The linear model suggested possible increases in genetic effects, and decreases nonshared environmental (and, to a lesser extent, shared environmental effects) in pubertal as compared to pre-pubertal twins. There was also some indication that the linear model may provide the best fit to the data, as AIC was smaller for the linear model, and the chi square statistic was just shy of suggesting a significantly worse fit for the no moderation model ($p = .06$). However, there was also substantial evidence for selecting the no moderation model, as BIC was smaller for this model, and none of the moderation paths were statistically significant in the linear model (i.e., the 95% confidence intervals all included 0; see Table 3).

Taken together, results provide the most support for no significant moderation effects. Again, standardized estimates calculated from the data presented in Table 3 for the no moderation models indicated that genetic influences were nonsignificant and accounted for approximately 7% of the variance. Shared environmental influences were also not statistically significant but were estimated to account for 18% of the variance. Nonshared environmental effects were statistically significant and accounted for 75% of the variance².

DISCUSSION

This was the first study to examine genetic and environmental influences on thin-ideal internalization across age and pubertal development. Overall, results demonstrated similarities in genetic and environmental effects across development, as no significant moderation effects were detected. Findings also highlighted the more prominent role for environmental influences on thin-ideal internalization than genetic factors across all developmental stages examined.

In addition to the lack of moderation effects detected, the current study suggested minimal genetic effects for thin-ideal internalization, with the majority of the variance accounted for by nonshared environmental effects and, to a lesser extent, shared environmental effects. Notably, the one previous twin study of thin-ideal internalization suggested primarily genetic and nonshared environmental effects (9.). However, the prior study had a smaller sample size ($N=343$) and a larger proportion of older twins than the current investigation. It is possible that differences in findings may be explained by a combination of differences in sample sizes and age groups examined across studies. Shared environmental effects, in particular, are difficult to detect with smaller sample sizes, and these effects tend to load onto estimates of additive genetic effects in these underpowered studies (42.). Even in the current study ($N=1,024$), estimates of the shared environment were moderate (i.e., 15–20% of the variance), but were still non-significant, supporting the notion that significant shared environmental influences on thin-ideal internalization may be difficult to detect without an even larger sample. Together, findings suggest that there may be effects of the shared

environment across adolescence, but larger twin samples will be needed to detect them and provide stable estimates of their effects.

Another explanation for somewhat inconsistent results may be related to instability in the SATAQ-3 general internalization measure used. Although psychometric properties for the SATAQ-3 general internalization scale are good in the adolescent and adult samples examined previously (16.), the current study used a shortened, 3-item version of the scale in order to achieve satisfactory factor structure and invariance across groups. This shortened scale has not been used in prior studies, and the SATAQ-3 has not been well validated in pre-adolescent children. However, our 3-item scale correlated significantly with the original 9-item scale ($r = .87, p < .01$) and the 4-item scale previously recommended for use with adolescents ($r = .95, p < .01$) (30.). Further, results in the current study did not differ when using these different scoring algorithms (data not shown), as all models showed non-significant moderation paths. Although future studies with other measures of thin-ideal internalization would be useful to confirm our results, our findings appear to be quite robust across measurement strategies. Continued work in defining and measuring the construct of thin-ideal internalization across age and developmental stage is still needed, however, including novel experimental methods that are being developed for preschool children (43.). Future research should aim to replicate our results using measures that are designed for and validated in samples encompassing childhood, adolescence, and adulthood.

Despite the lack of significant differences in etiologic influences on thin-ideal internalization, the current study did suggest significant mean-level increases in thin-ideal internalization in older age groups and in pubertal (as compared to pre-pubertal) twins (see Table 1). The significant mean-level increase in thin-ideal internalization across development may seem to contradict the minimal differences in etiological effects detected across adolescence. However, it is possible for mean levels of a phenotype to change even when the relative proportions of genetic versus environmental influences on the phenotype remain stable. This could occur when the type of risk factor (e.g., shared or nonshared environmental risk factor) remains constant while the prevalence or strength of the risk factor increases and drives increases in mean levels of the putative phenotype. However, it is important to consider that the current findings only suggest that the relative *magnitude* of genetic and environmental influences are stable across development; results do not necessarily suggest that *identical* etiological influences act on thin-ideal internalization across all age/pubertal groups examined. It is also feasible that the specific risk factors change across this period but that the “new” risk factors load onto the same environmental estimates as did the earlier risk factors. Future work is needed to identify which specific environmental risk factors make the most important contributions to risk for thin-ideal internalization at different development stages.

Several limitations of this study must be noted. Our sample sizes were smaller than would be ideal. Most simulation studies of twin moderation models include samples with 1,000 MZ and 1,000 DZ twins (38.), and we had fewer twins in our sample (i.e., approximately 568 MZ twins and 496 DZ twins). Our sample size may have influenced our results in two ways. First, it may have limited our ability to detect significant shared environmental influences on thin-ideal internalization, as described above. Secondly, it is possible that a larger sample

size would detect significant moderation effects. In particular, examination of the unstandardized estimates in Figures 2a and 2c suggest the possibility of some moderation effects (i.e., increasing genetic influences and decreasing shared and nonshared environmental effects) although the magnitude of change appears to be much smaller than those observed previously for disordered eating (33.). Interestingly, studies of age and pubertal moderation effects for disordered eating have employed a range of sample sizes (i.e., 510–2,618 twins), which at times were on par with those used in current study (12–14; 21; 32; 33.). Therefore, if there are moderating effects on thin-ideal internalization, they appear to be smaller and more difficult to detect than those for disordered eating. Future studies in larger samples are needed to confirm these impressions and replicate our findings of minimal etiological moderation.

Other limitations include the use of cross-sectional data, and that analyses were confined to twins between the ages of 8–25. Since our data were cross-sectional, we could not examine developmental stability or change within twin pairs as they advanced through puberty. We also could not rule out the possibility that the magnitude of genetic and environmental effects may vary in younger or older age groups than those examined herein. Future longitudinal work is needed to replicate the cross-sectional effects identified in the current study, and to extend findings to older and/or younger age groups. Genetically informed, longitudinal studies that measure specific environmental risk factors (e.g., differential media exposure) would be informative for elucidating the specific etiological risk factors contributing to thin-ideal internalization across time.

Despite these limitations, our findings are the first to demonstrate minimal differences in the etiology of thin-ideal internalization across adolescence and pubertal development. It is hoped that these results, particularly if replicated using detailed longitudinal designs, will increase understanding of etiological risk for thin-ideal internalization, and, ultimately, disordered eating.

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Figure 1. Path diagram of the univariate twin moderation model and the extended univariate moderation model

Age₁/Pub₁=age or puberty moderators for twin 1; Age₂/Pub₂=age or puberty moderators for twin 2; A= additive genetic effects; C=shared environmental effects; E=nonshared environmental effects; M₁ and M₂=Moderator in twin 1 and twin 2, respectively; β_M =phenotypic regression coefficient; a, c, and e=paths or intercepts; β_X, β_Y , and β_Z =linear moderators, β_X^2, β_Y^2 , and β_Z^2 =quadratic moderators. The model is pictured for only one twin type, but parameters are fit separately for MZ and DZ twins. Parameters in parentheses ($\beta_M M_1$ and $\beta_M M_1$) are included only in the extended univariate moderation model (i.e., puberty moderation models) in order to account for co-twin covariance on the moderator. These terms are not needed in the univariate twin moderation model (i.e., age moderation models) since the moderator (age) is shared among all twin pairs.

Figure 2. Unstandardized variance estimates for the full and best-fitting models

The no moderation models are the best-fitting for both age and puberty. Unstandardized additive genetic (—), shared environmental (•••••), and nonshared environmental (---) variance components. Results for age moderation models are presented in Figure 3a (full model) and 3b (best-fitting no moderation model); puberty moderation model results are presented in Figure 3c (linear model) and 3d (best-fitting no moderation model).

Table 1

Descriptive Statistics

Scale	Overall M (SD)	Age 8-12 M (SD)	Age 13-16 M (SD)	Age 16-25 M (SD)	Pre- pubertal M (SD)	Pubertal M (SD)	Pearson r with MEBS	Pearson r with Puberty ^a	Pearson r with Age ^a	Pearson r with BMI
Thin-Ideal Internalization (3-item)	2.31 (1.15)	1.88 (1.04)	2.48 (1.18)	2.64 (1.08)	1.90 (1.03)	2.52 (1.15)	.44**	.27** (.22**)	.28** (.24**)	.23**
Disordered Eating	4.89 (4.93)	3.95 (4.36)	5.21 (5.17)	5.53 (5.11)	3.74 (4.11)	5.41 (5.19)	--	.17**	.15**	.44**
Pubertal Development	3.14 (1.11)	1.76 (0.66)	3.70 (0.53)	4.00 (0.00)	1.58 (0.44)	3.84 (0.36)	--	--	.88**	.49**
Age	14.91 (3.78)	10.46 (1.37)	15.67 (1.24)	18.94 (1.82)	10.43 (1.49)	16.98 (2.52)	--	--	--	.48**
BMI	21.69 (5.76)	18.17 (4.08)	23.02 (5.04)	24.11 (6.21)	17.65 (3.55)	23.53 (5.64)	--	--	--	--

Note. BMI = Body Mass Index. Thin-Ideal Internalization was assessed with the modified, 3-item general internalization subscale of the Sociocultural Attitudes Toward Appearance Scale-3. Disordered eating was assessed using the total score of the Minnesota Eating Behavior Survey. Pubertal Development was assessed with the pubertal development scale.

^a Pearson correlations of thin-ideal internalization with puberty and age are presented both with and without controlling for disordered eating. The correlations in parentheses are partial correlations that control for disordered eating (i.e., the Minnesota Eating Behavior Survey total score.)

Table 2

Indices of Fit for Nested ACE Models Examining the Etiology of Thin-Ideal Internalization by Age and Pubertal Development.

Model	-2lnL	df	χ^2 (df)	p	AIC	BIC
Age Models						
Full Moderation	2854.44	1024	--	--	806.44	-1786.42
Linear Moderation	2855.83	1027	1.39 (3)	0.71	801.83	-1795.14
No Moderation	2857.06	1030	2.62 (6)	0.85	797.06	-1803.94
Puberty Models						
Linear Moderation	2822.34	1011	--	--	800.34	-1754.98
No Moderation	2829.67	1014	7.33 (3)	0.06	801.67	-1760.71

Note. -2lnL = minus 2 times the log likelihood. χ^2 = change in chi-square (-2lnL) from the full moderation model; AIC = Akaike information criteria; BIC = Bayesian information criterion; Mod = Moderator; DE = Disordered Eating; A=additive genetic effects; C = Shared environmental effects; E = Nonshared environmental effects. Best-fitting models, as determined from non-significant chi-square and lowest AIC and BIC values, are indicated with bold text. In the "Full" moderation model, genetic, shared environmental, and nonshared environmental estimates are allowed to vary both linearly and quadratically across levels of the moderator (i.e., age or pubertal development). In the "Linear" moderation model, genetic, shared environmental, and nonshared environmental estimates are allowed to vary linearly across levels of the moderator. In the "No Moderation" model, genetic, shared environmental, and nonshared environmental estimates are constrained to be equal across all age or pubertal groups. A full moderation model was not calculated for pubertal groups since quadratic moderation is not possible when examining only two groups.

Table 3

Unstandardized Path and Moderator Estimates for Twin Models.

Model	a	c	e	A1	C1	E1	A2	C2	E2
Age Models									
Full	.00 (-.56, .54)	-.42 (-.57, .57)	.86 (.77, .90)	-.75 (-.90, .90)	.13 (-.90, .90)	.02 (-.23, .30)	.26 (-.73, .73)	.12 (-.68, .68)	-.03 (-.17, .10)
Linear	.23 (-.57, .57)	.36 (-.56, .56)	-.87 (-.90, -.78)	.11 (-.35, .35)	-.00 (-.34, .34)	.03 (-.04, .09)	--	--	--
No Mod	-.29 (-.58, .58)	-.39 (-.55, .55)	.85 (.78, .90)	--	--	--	--	--	--
Puberty Models									
Linear	-.23 (-.53, .45)	.30 (-.53, .53)	.87 (.77, .90)	.79 (-.90, .90)	-.08 (-.90, .90)	-.07 (-.18, .06)	--	--	--
No Mod	.26 (-.59, .59)	-.42 (-.56, .56)	-.85 (-.90, -.78)	--	--	--	--	--	--

Note. Full=Full moderation model allowing for linear and quadratic moderation effects; Linear=Linear moderation model allowing for linear moderation only; No Mod=No moderation model; a=genetic path estimate; A1=linear moderator of genetic path estimate; A2=quadratic moderator of genetic path estimate; c=shared environmental path estimate; C1=linear moderator of shared environmental path estimate; C2=quadratic moderator of shared environmental path estimate; e=nonshared environmental path estimate; E1=linear moderator of nonshared environmental path estimate; E2=quadratic moderator of nonshared environmental path estimate. Estimates are followed by 95% confidence intervals in parentheses. Confidence intervals that do not overlap with zero indicate statistical significance at $p < .05$. Significant estimates are noted in bold text. The no moderation models are the best fitting models in all cases.