Published in final edited form as:

Int J Eat Disord. 2012 December ; 45(8): 942–948. doi:10.1002/eat.22056.

Genetic and Environmental Influences on Thin-Ideal Internalization

Jessica L. Suisman, MA¹, Shannon M. O'Connor, BS¹, Steffanie Sperry, MA², J. Kevin Thompson, PhD², Pamela K. Keel, PhD³, S. Alexandra Burt, PhD¹, Michael Neale, PhD⁴, Steven Boker, PhD⁵, Cheryl Sisk, PhD^{1,6}, and Kelly L. Klump, PhD¹

¹Department of Psychology, Michigan State University, East Lansing, Michigan

²Department of Psychology, University of South Florida, Tampa, Florida

³Department of Psychology, Florida State University, Tallahassee, Florida

⁴Department of Psychiatry and Human Genetics, Virginia Commonwealth University, Richmond, Virginia

⁵Department of Psychology, University of Virginia, Charlottesville, Virginia

⁶Neuroscience Program, Michigan State University, East Lansing, Michigan

Abstract

Objective—Current research on the etiology of thin-ideal internalization focuses on psychosocial influences (e.g., media exposure). The possibility that genetic influences also account for variance in thin-ideal internalization has never been directly examined. This study used a twin design to estimate genetic effects on thin-ideal internalization and examine if environmental influences are primarily shared or nonshared in origin.

Method—Participants were 343 post-pubertal female twins (ages 12–22; $M=17.61$) from the Michigan State University Twin Registry. Thin-ideal internalization was assessed using the Sociocultural Attitudes toward Appearance Questionnaire-3.

Results—Twin modeling suggested significant additive genetic and nonshared environmental influences on thin-ideal internalization. Shared environmental influences were small and non-significant.

Discussion—Although prior research focused on psychosocial factors, genetic influences on thin-ideal internalization were significant and moderate in magnitude. Research is needed to investigate possible interplay between genetic and nonshared environmental factors in the development of thin-ideal internalization.

Keywords

Thin-ideal; internalization; body image; disordered eating; twin study; heritability; Tripartite Model

Psychosocial perspectives on the development of eating disorders and disordered eating symptoms (e.g., body dissatisfaction, weight preoccupation) often center on sociocultural

Corresponding Author: Kelly L. Klump, PhD, Associate Professor, Department of Psychology, Michigan State University, 107B Psychology Building, East Lansing, MI 48824-1116, PH: 517-432-9861, Fax: 517-432-2476, klump@msu.edu.

Parts of this manuscript were presented at the International Conference on Eating Disorders, Miami, Florida, April 27-30, 2011.

None of the authors have financial conflicts of interest.

factors such as thin-ideal images that are perpetuated by the mass media within Western cultures¹⁻³. Typically, these theories place an emphasis on thin-ideal internalization (i.e., the acceptance and adherence to sociocultural beauty standards of thinness), which is hypothesized to be a primary mechanism that links exposure to mass media and the development of eating pathology³⁻⁴. For example, the Tripartite Model^{2, 5} of body dissatisfaction and eating disturbance postulates that the sociocultural emphasis on thinness is reinforced and perpetuated by three primary factors: the media (i.e., television, advertisements, magazines), parents (e.g., parental focus on weight/dieting) and peers (e.g., peer discussions about dieting, weight-based teasing). According to the Tripartite Model, the reinforcement of the thin-ideal by these primary factors leads to thin-ideal internalization, as well as social comparison (i.e., evaluating and comparing oneself to others, including individuals displayed in the media). It is through the development of thin-ideal internalization and social comparison that body dissatisfaction, disordered eating, and eating disorders are thought to emerge.

Empirical support for the Tripartite Model, including the significance of thin-ideal internalization as a central factor in this model, has begun to emerge, as structural equation modeling results have generally demonstrated good fits to Tripartite Model data⁶⁻⁹. Moreover, research continues to demonstrate robust associations between thin-ideal internalization and disordered eating¹⁰⁻¹³, and interventions that decrease thin-ideal internalization have also been shown to decrease body dissatisfaction¹⁴⁻¹⁵. Taken together, it is clear that thin-ideal internalization is an important component in the pathway from sociocultural risk factors to the development of eating pathology.

Despite these promising findings, additional research is needed to more fully understand risk factors for the development of thin-ideal internalization. Theories such as the Tripartite Model inherently suggest that risk factors (e.g., exposure to media, behaviors of peers and family) are environmental in nature, and that these environmental factors account for the variance in thin-ideal internalization. One additional component that is strikingly absent from current theoretical models and research is the possibility that genetic influences contribute to the variance in thin-ideal internalization. This possibility is supported by the fact that despite almost ubiquitous exposure to thin-ideal images in Western countries, only *some* women ultimately internalize this ideal and go on to develop disordered eating behaviors. Individual difference factors, such as genetic risk, may predict which of these women are more likely to internalize the thin-ideal and which are not. The presence of significant genetic influences on eating disorders, and disordered eating symptoms (e.g., dietary restraint, drive for thinness, weight preoccupation, body dissatisfaction, shape/weight concerns) lend additional credence to the possibility that genetic factors may contribute to thin-ideal internalization¹⁶⁻²¹. The fact that thin-ideal internalization is a pre-cursor to many of these symptoms (rather than a correlate of, or proxy for, the symptoms;^{10, 22} further highlights the need to clarify genetic and environmental influences on thin-ideal internalization specifically.

Twin studies are a powerful approach for determining the degree to which variance in a trait is accounted for by genetic versus environmental factors. The classical twin design allows for the investigation of additive genetic effects, or genetic influences on a trait are due to the additive effects of multiple genetic loci. Twin designs also allow the variance in a trait to be parsed into two forms of environmental effects; the shared and nonshared environment. Shared environmental factors (C) are environmental influences that are common to siblings raised in the same family and make them more similar to each another. These influences are generally conceptualized as family-wide or culture-wide factors, such as parenting style, socioeconomic status, and exposure to certain cultural beliefs/practices. Nonshared environmental factors (E), on the other hand, are environmental influences that are unique to

siblings reared in the same family that make them more dissimilar from one another. Examples of nonshared environmental factors include differential experiences between siblings such as peer groups, extracurricular activities, or traumatic life events. To date, twin and adoption studies of post-pubertal adolescents and adults have demonstrated little-to-no effect of the shared environment on eating disorders and disordered eating symptoms, but a rather large and significant effect of nonshared environmental factors^{23–25}.

Importantly, the current conceptualization of the etiology of thin-ideal internalization would suggest a significant effect of the shared environment, as the exposure to media (i.e., almost all women are exposed to high levels of thin-ideal media) and parental attitudes/beliefs (e.g., emphasis on thinness) are likely common to siblings reared in the same family. However, as noted above, it is also possible that genetic factors contribute to this variance and predict which girls/women internalize the shared beauty ideals and which are relatively immune to the environmental effects. Moreover, it may be that the sociocultural influences act in a nonshared rather than shared environmental fashion. For example, siblings may experience differential exposure to cultural and family pressures for thinness that lead to differential internalization of thin-ideal messages. A prototypical example in this regard is the situation in which parental reinforcement of the thin-ideal in only one child (e.g., encouraging a heavier child to lose weight) might lead to thin-ideal internalization in only one sibling. These types of environmental effects would load onto the nonshared rather than shared environment, since they would make siblings' thin-ideal internalization different rather than similar to each other.

Clarification of the extent to which genetic, shared environmental, and/or nonshared environmental factors contribute to thin-ideal internalization is needed to identify more specific risk factors and *the ways in which* they are most likely to operate. Consequently, the aim of the present study was to investigate the extent to which genetic and environmental influences contribute to the variance in thin-ideal internalization in a large sample of female adolescent and young adult twins.

METHOD

Participants

Participants included 343 (205 MZ; 138 DZ) female twins between the ages of 12 and 22 ($M=17.59$, $SD=2.25$) from the Michigan State University Twin Registry (MSUTR)²⁶. The MSUTR is population-based and recruits twins through birth records in collaboration with the Michigan Department of Community Health (Further details are available elsewhere²⁶). Most participants in the current study were from a complete twin pair, where data was available for both twins ($N=336$), although 7 twins were from incomplete pairs in which only one twin provided data. Importantly, given prior research suggesting variations in the etiology of disordered eating in pre-versus post-pubertal twins^{25, 27}, all twins included in the present study were pubertal, as indicated by the onset of menarche. Although presence of a minimum of only one menstrual cycle was required to be included in the present study, the majority of twins (97%) had been regularly cycling for at least 6 months, and prior research has demonstrated that twins who have begun menstruating are identical to adult twins in terms of genetic and environmental influences on disordered eating^{25; 27–28}.

Twins included in this study were drawn from two studies within the MSUTR (i.e., the *Twin Study of Hormones and Behaviors across the Menstrual Cycle* and the *Twin Study of Hormones and Disordered Eating Across Puberty*) that have primary aims involving the investigation of ovarian hormone influences on disordered eating. Thus, 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+). The use of these inclusion/exclusion criteria did not appear to inadvertently affect the range or variability in thin-ideal internalization scores, as scores in our study ($M = 2.37$, $SD = 0.72$) were on par with those from previous research of similar age groups (e.g., $M = 2.22$ – 3.68 , $SD = 0.87$ – 1.13)^{29–32}. Moreover, 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)^{26; 33}, which was also true for the subset of MSUTR participants included in the present sample (i.e., 84% Caucasian).

Twin zygosity was determined using physical similarity questionnaires that have been shown to be over 95% accurate³⁴. Each research assistant that had contact with the twins during study participation completed the physical similarity questionnaire for the twin pair. Additionally, twins age 16 or older each completed a self-report version of the zygosity questionnaire, and, when possible, the questionnaire was also completed by the twins' parent (usually the mother). In twins under age 16, the zygosity questionnaire was completed by the twins' mother only (i.e., the twins did not provide a self-report). If results across any of these raters (i.e., twins, parent, and research assistants) were not in agreement, questionnaire responses and pictures of the twins were examined by study investigators (KLK and JLS) to determine final zygosity status.

Measures

Thin-Ideal Internalization—Internalization of the thin-ideal was assessed with the 9-item, thin-ideal internalization subscale from the Sociocultural Attitudes toward Appearance Questionnaire-3 (SATAQ-3)^{5; 30}. Using a 5-point Likert scale (ranging from definitely disagree to definitely agree), this subscale asks participants to rate their agreement with statements about the degree to which participants want to look like individuals from various media sources, such as magazines, television, and movies. Higher scores indicate greater levels of thin-ideal internalization. This scale has demonstrated excellent reliability and validity in prior research^{30; 35}. In general it correlates moderately with measures of disordered eating^{30; 35}, differentiates individuals with eating disorders from controls^{30; 35}. Internal consistency is excellent in our sample ($\alpha = 0.91$) and in prior research (0.92–0.96)^{30; 35}.

Body Mass Index (BMI)—Body mass index was calculated ($[\text{weight}]/[\text{height}]^2$) from laboratory assessments of height and weight made using a wall-mounted ruler and digital scale, respectively.

Statistical Analyses

In order to control for positive associations between both BMI and age with thin-ideal internalization, BMI and age were partialled out of thin-ideal internalization scores prior to analyses. Intraclass twin correlations were then calculated to provide an initial estimate of genetic and environmental influences on thin-ideal internalization. Additive genetic influences are implied when MZ twin correlations are approximately double the DZ twin correlations. Shared environmental influences are suggested when the MZ and DZ twin correlations are approximately equal. Finally, nonshared environmental influences are inferred when the MZ correlation is less than 1.00, and/or both the MZ and DZ twin correlations are small and non-significant. Importantly, nonshared environmental estimates also include measurement error.

The structural-equation modeling program Mx³⁶ was then used to perform biometric model fitting analyses. Models were fit to raw data, which treats data as missing at random. This allows for the inclusion of all twin pairs, even if one twin is missing data, and is an

advantage over the use of covariance matrices (where pairwise deletion occurs for missing data)³⁷. An initial “full” ACE model was first fit to the data. Three nested models were then fit to the data and compared to the corresponding initial “full” model: an AE model (constraining C to 0), a CE model (constraining A and E to 0), and an E model (constraining A and C to 0). Comparisons between models were made by calculating a chi-square test of model fit, computed by calculating the difference in minus twice the log likelihood ($-2\ln L$) between the “full” models and the submodels. Submodels were rejected if the chi-square test was statistically significant, as a significant chi-square test indicates that dropping the parameters from the full model provided a significantly worse fit to the data. (AIC; χ^2-2df)³⁸, a measure of model fit versus model parsimony, was also used to determine model fit. Smaller (i.e., more negative) AIC values indicate improved fit to the data among nested models.

RESULTS

Twin correlations for thin-ideal internalization were significantly greater in MZ versus DZ twins (MZ $r=.44$; DZ $r=.24$; $Z=2.60$, $p=.02$), suggesting the presence of significant genetic effects. Large differences between MZ and DZ twin correlations, and an MZ correlation less than 1.00, suggested that shared environmental influences were small, but nonshared environmental influences likely contributed to the variance in thin-ideal internalization.

Twin model-fitting analyses generally supported these initial impressions. The full ACE model suggested large influences of A and E, but smaller effects of C (see Table 1). Most submodels did not provide a significantly worse fit to the data than the full model (i.e., chi-square differences were generally non-significant). However, the AE model was determined to be the best fitting and most parsimonious model, as this model had the lowest AIC value and the chi-square value was identical to that of the full ACE model. These results suggest significant genetic and nonshared environmental influences on thin-ideal internalization scores, but minimal effects of shared environment.

DISCUSSION

This was the first study to examine genetic and environmental influences on thin-ideal internalization. Findings suggested significant genetic and nonshared environmental influences on thin-ideal internalization, but effects of the shared environment were small. Indeed, the best-fitting model allowed us to constrain the effects of the shared environment to zero. Importantly, these effects were independent of participant age and BMI, as these variables were controlled for in all analyses. Our findings are broadly consistent with prior research on disordered eating and eating disorders in post-pubertal females suggesting no significant effects of the shared environment on these phenotypes after puberty^{25; 27}, but significant genetic and nonshared environmental influences.

The significant influence of additive genetic effects on thin-ideal internalization supports our hypothesis that genetic predispositions may explain why some individuals internalize the thin-ideal while others do not. Interestingly, the heritability estimate of over 40% is remarkably similar to the heritability of disordered eating and eating disorders^{23; 39} and suggests that thin-ideal internalization is just as heritable as the outcomes it predicts (e.g., body dissatisfaction, disordered eating). These results suggest that models of the development of thin-ideal internalization and disordered eating, such as the Tripartite Model², should be updated to include genetic influences. This will encourage the investigation of genetic predispositions, rather than only environmental influences, when examining the origins of thin-ideal internalization.

One set of “genetic” predispositions that might explain the heritability of thin-ideal internalization are personality traits. Although research has not extensively investigated personality traits that are associated with thin-ideal internalization, initial results suggest some associations with variables such as perfectionism⁴⁰, and researchers have hypothesized that personality may influence individual differences in internalization of the thin ideal⁴¹. Interestingly, such personality characteristics are known to be heritable⁴² and may contribute to the additive genetic variance on thin-ideal internalization. In other words, a large proportion of the heritability of thin-ideal internalization may overlap with genetic factors that influence personality. Future twin studies should directly investigate this possibility using bivariate twin analysis techniques.

The non-significance of the shared environment in explaining the variance in thin-ideal internalization may seem surprising, particularly given the “shared” nature of exposure to media among women (i.e., almost all women are exposed to high levels of thin-ideal media) and the expectation that siblings growing up in the same home would be exposed to similar levels of such media, and thus, would likely internalize the thin-ideal to a similar degree. The present findings do not support these predictions, as siblings being raised in the same home did differ largely in the extent to which they internalize the thin-ideal. All of the twin correlations were significantly less than 1, and varied by zygosity, such that MZ twins were more similar to one another than DZ twins. Thus, even despite shared cultural exposure and shared exposure within the home, siblings differ in their degree of thin-ideal internalization. As discussed above, genetic influences explain a large proportion of variance in the degree to which the thin-ideal is internalized. Additionally, differences in thin ideal internalization might be predicted by gene-environment interactions (GxE). Specifically, the effects of the shared environment may interact with genetic influences on the thin-ideal, such that shared environmental effects (e.g., shared media in the home) are “risky” only in individuals who also have genetic predispositions for thin-ideal internalization. This possibility is especially intriguing because interactions between genes and the shared environment inflate estimates of additive genetic effects in the classical twin model. Thus, the robust estimates of additive genetic effects present in this study may include untested interaction effects between additive genetic and shared environmental influences. This possibility should be directly investigated in future studies that have adequate sample sizes to test gene-environment interaction twin models⁴³.

An additional possibility is that influences traditionally conceptualized as “shared” (e.g., media and cultural factors) are operating at the level of the nonshared environment. For example, *differences* in exposure to environmental risk factors for thin-ideal internalization among siblings (e.g., parental reinforcement of dieting in only a slightly heavier child) might result in siblings becoming more discrepant from each other in their levels of thin-ideal internalization. Such effects would load on estimates of the nonshared environment and could contribute to the significant nonshared environmental effects observed in our study. This possibility is supported by evidence suggesting some differences in exposure to media among siblings⁴⁴, and extensive research demonstrating that increased mean levels of media exposure or exposure to certain types of media (i.e., that is heavily focused on the thin-ideal) is associated with increased levels of thin-ideal internalization^{45–52}. Thus, nonshared environmental effects may operate, whereby the sibling who is exposed to more media is more likely to internalize the thin-ideal.

Since the present study is the only twin study of thin-ideal internalization conducted to date, it is unclear whether and how these findings might replicate in other populations, particularly those in which the thin-ideal is not prominent. Genetic effects would be expected to decrease, and environmental estimates would be expected to increase, in cultures in which there is more variance in exposure to the thin-ideal across individuals⁵³.

This is because, in cultures where thin-ideal exposure is rare, even individuals who have genetic predispositions for thin-ideal internalization may never internalize the thin-ideal because they are never exposed to it. Thus, genetic predispositions cannot operate, driving down estimates of genetic effects. The importance of environmental exposure, on the other hand, would increase, since it is likely that only individuals who have been exposed to the thin-ideal would internalize such ideals. Importantly, such cross-cultural differences in heritability have been noted in even highly genetic characteristics, such as height. Height is extremely heritable in populations where most individuals are guaranteed adequate nutrition, however, in populations where some individuals do not receive enough nutrition, genetic influences become less important and environmental influences (i.e., exposure to food/nutrition) increase⁵⁴. Moving forward, it will be important to accrue twin study data in cultures where mean levels of exposure to the thin-ideal are more variable and/or lower than in the United States and other Western cultures. These data can be used to understand possible differences in etiology across cultures and enhance understanding of thin-ideal internalization as a risk factor for eating pathology.

Despite the strengths of this study, namely, that it was the first investigation of genetic and environmental influences on the variance in thin-ideal internalization, there were limitations that should be noted as well. One limitation is the relatively small sample size, which may have limited the ability to detect shared environmental influences⁵⁵ and resulted in broad confidence intervals for several parameter estimates. However, examination of the twin correlations and submodels clearly demonstrate the presence of genetic effects and minimal influence of the shared environment, as confidence intervals for genetic effects in the AE model were significant, and constraining genetic effects to 0 significantly reduced the fit of the models. Collectively, these findings suggest that additive genetic effects are significant, and future studies should replicate these findings using larger samples in order to identify more precise point estimates of genetic effects and confirm the negligible impact of the shared environment.

A core assumption of all twin studies is the equal environments assumption (EEA), which states that the environment should not differentially influence the phenotype in question for MZ as compared to DZ twins. For constructs such as thin-ideal internalization, it seems most likely that the EEA would be violated if greater physical similarity in MZ, as compared to DZ twins, resulted in MZ twins being more similar in thin-ideal internalization than DZ twins, thereby inflating heritability estimates. Although measures of co-twin physical similarity were not available to specifically test the EEA in the present sample, prior studies have clearly demonstrated that degree of co-twin physical similarity does not predict twin similarity for disordered eating, including measures of phenotypes that are correlated with thin-ideal internalization such as body dissatisfaction and disordered eating⁵⁶. Thus, the EEA does not appear to be violated for disordered eating attitudes and behaviors, and is unlikely to be violated for thin-ideal internalization. However, future studies of the EEA that include measures of thin-ideal internalization are needed to confirm these impressions.

Finally, we were unable to directly investigate specific environmental mechanisms or specific genes in the present study. Future studies should include environmental measures (e.g., exposure to media in the home) that might allow for direct investigation of possible gene-environment interplay. For instance, it might be expected that the heritability of thin-ideal internalization is higher in the context of a “high-media” home environment versus a “low-media” environment. Similarly, future twin and molecular studies should more closely examine genetic risk for the construct by investigating overlap in the genetic architecture of thin-ideal internalization and the disordered eating symptoms it predicts as well as exploring specific genes that may confer risk for the development of thin-ideal internalization. Such investigations of specific genes, specific environments, and the interaction between genes

and the environment will allow for an increased understanding of the etiology of thin-ideal internalization.

Acknowledgments

Funding/Support This research was supported by grants from the National Institute of Mental Health (1 R01 MH0820-54 and 5-R01MH092377-02) awarded to Dr. Klump. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute of Mental Health.

References

1. Levine, MP.; Smolak, L. Cultural influences on body image and the eating disorders. In: Agras, WS., editor. The oxford handbook of eating disorders. Oxford: Oxford University Press; 2010.
2. Thompson, JK.; Heinberg, LJ.; Altabe, MN.; Tantleff-Dunn, S. Exacting beauty: Theory, assessment, and treatment of body image disturbance. American Psychological Association; 1999.
3. Thompson JK, Heinberg LJ. The media's influence on body image disturbance and eating disorders: we've reviled them, now can we rehabilitate them? Journal of Social Issues. 1999; 55:339–353.
4. Stice E. Review of the evidence for a sociocultural model of bulimia nervosa and an exploration of the mechanisms of action. Clinical Psychology Review. 1994; 14:633–661.
5. Thompson, JK.; Schaefer, LM.; Menzel, JE. Internalization of thin-ideal and muscular-ideal. In: Cash, TF., editor. Encyclopedia of Body Image and Human Appearance. San Diego: Academic Press; 2012.
6. Keery H, van den Berg P, Thompson JK. An evaluation of the Tripartite Influence Model of body dissatisfaction and eating disturbance with adolescent girls. Body Image. 2004; 1:237–251. [PubMed: 18089156]
7. Shroff H, Thompson JK. The tripartite influence model of body image and eating disturbance: A replication with adolescent girls. Body Image. 2006; 3:17–23. [PubMed: 18089205]
8. van den Berg P, Thompson JK, Obrowski-Brandon K, Coover M. The tripartite influence model of body image and eating disturbance: A covariance structure modeling investigation testing the mediational role of appearance comparison. Journal of Psychosomatic Research. 2002; 53:1007–1020. [PubMed: 12445590]
9. Yamamiya Y, Shroff H, Thompson JK. The tripartite influence model of body image and eating disturbance: a replication with a Japanese sample. International Journal of Eating Disorders. 2008; 41:88–91. [PubMed: 17968899]
10. Thompson JK, Stice E. Thin-ideal internalization: Mounting evidence for a new risk factor for body-image disturbance and eating pathology. Current Directions in Psychological Science. 2001; 10:181–183.
11. Stice E. Risk and maintenance factors for eating pathology: A meta-analytic review. Psychological Bulletin. 2002; 128:825. [PubMed: 12206196]
12. Clark L, Tiggemann M. Appearance Culture in Nine- to 12-Year-Old Girls: Media and Peer Influences on Body Dissatisfaction. Social Development. 2006; 15:628–643.
13. Cafri G, Yamamiya Y, Brannick M, Thompson JK. The Influence of Sociocultural Factors on Body Image: A Meta-Analysis. Clinical Psychology: Science and Practice. 2005; 12:421–433.
14. Yamamiya Y, Cash TF, Melnyk SE, Posavac HD, Posavac SS. Women's exposure to thin-and-beautiful media images: body image effects of media-ideal internalization and impact-reduction interventions. Body Image. 2005; 2:74–80. [PubMed: 18089176]
15. Coughlin JW, Kalodner C. Media literacy as a prevention intervention for college women at low-or high-risk for eating disorders. Body Image. 2006; 3:35–43. [PubMed: 18089207]
16. Wade T, Martin NG, Tiggemann M. Genetic and environmental risk factors for the weight and shape concerns characteristic of bulimia nervosa. Psychological Medicine. 1998; 28:761–771. [PubMed: 9723134]
17. Rutherford J, McGuffin P, Katz RJ, Murray RM. Genetic influences on eating attitudes in a normal female twin population. Psychological Medicine. 1993; 23:425–436. [PubMed: 8332659]

18. Keski-Rahkonen A, Bulik CM, Neale BM, Rose RJ, Rissanen A, Kaprio J. Body Dissatisfaction and Drive for Thinness in Young Adult Twins. *International Journal of Eating Disorders*. 2005; 37:188–199. [PubMed: 15822080]
19. Baker JH, Maes HH, Lissner L, Aggen SH, Lichtenstein P, Kendler KS. Genetic risk factors for disordered eating in adolescent males and females. *Journal of Abnormal Psychology*. 2009; 118:576–586. [PubMed: 19685954]
20. Mazzeo SE, Mitchell KS, Bulik CM, Aggen SH, Kendler KS, Neale MC. A twin study of specific bulimia nervosa symptoms. *Psychological Medicine*. 2010; 40:1203–1213. [PubMed: 19818201]
21. Spanos A, Burt SA, Klump KL. Do weight and shape concerns exhibit genetic effects? Investigating discrepant findings. *International Journal of eating Disorders*. 2010; 43:29–34. [PubMed: 19235850]
22. Stice E. A prospective test of the dual-pathway model of bulimic pathology: mediating effects of dieting and negative affect. *Journal of Abnormal Psychology*. 2001; 110:124. [PubMed: 11261386]
23. Bulik CM, Sullivan PF, Wade TD, Kendler KS. Twin studies of eating disorders: A review. *International Journal of Eating Disorders*. 2000; 27:2–20.
24. Klump KL, Suisman JL, Burt SA, McGue M, Iacono WG. Genetic and environmental influences on disordered eating: An adoption study. *Journal of Abnormal Psychology*. 2009; 118:797–805. [PubMed: 19899849]
25. Klump KL, Burt SA, McGue M, Iacono WG. Changes in genetic and environmental influences on disordered eating: A longitudinal twin study. *Archives of General Psychiatry*. 2007; 64:1409–1415. [PubMed: 18056549]
26. Klump KL, Burt SA. The Michigan State University Twin Registry: Genetic, environmental, and neurobiological influences on behavior across development. *Twin Research*. 2006; 9:971–977.
27. Culbert KM, Burt SA, McGue M, Iacono WG, Klump KL. Puberty and the genetic diathesis of disordered eating attitudes and behaviors. *Journal of Abnormal Psychology*. 2009; 118:788–796. [PubMed: 19899848]
28. Rowe R, Pickles A, Simonoff E, Bulik CM, Silberg JL. Bulimic symptoms in the Virginia twin study of adolescent behavioral development: Correlates, comorbidity, and genetics. *Biological Psychiatry*. 2002; 51:172–182. [PubMed: 11822996]
29. Markland D, Oliver EJ. The sociocultural attitudes towards appearance questionnaire-3: a confirmatory factor analysis. *Body Image*. 2008; 5:116–121. [PubMed: 18405870]
30. Thompson JK, van den Berg P, Roehrig M, Guarda AS, Heinberg LJ. The sociocultural attitudes towards appearance scale-3 (SATAQ-3): Development and validation. *International Journal of Eating Disorders*. 2004; 35:293–304. [PubMed: 15048945]
31. Heinicke BE, Paxton SJ, McLean SA, Wertheim EH. Internet-delivered targeted group intervention for body dissatisfaction and disordered eating in adolescent girls: a randomized controlled trial. *Journal of Abnormal Child Psychology*. 2007; 35:379–391. [PubMed: 17243014]
32. Knauss C, Paxton SJ, Alsaker FD. Body Dissatisfaction in Adolescent Boys and Girls: Objectified Body Consciousness, Internalization of the Media Body Ideal and Perceived Pressure from Media. *Sex Roles*. 2008; 59:633–643.
33. Culbert KM, Breedlove M, Burt SA, Klump KL. Prenatal hormone exposure and risk for eating disorders. *Archives of General Psychiatry*. 2008; 65:329–336. [PubMed: 18316679]
34. Peeters H, Van Gestel S, Vlietinck R, Derom C, Derom R. Validation of a telephone zygosity questionnaire in twins of known zygosity. *Behavior Genetics*. 1998; 28:159–163. [PubMed: 9670591]
35. Calogero RM, Davis WN, Thompson JK. The Sociocultural Attitudes Toward Appearance Questionnaire (SATAQ-3): reliability and normative comparisons of eating disordered patients. *Body Image*. 2004; 1:193–198. [PubMed: 18089151]
36. Neale, MC. *Mx: Statistical Modeling*. Richmond, VA: Department of Psychology; 1995.
37. Little, RJA.; Rubin, DB. *Statistical Analysis with Missing Data*. New York: Wiley; 1987.
38. Akaike H. Factor analysis and AIC. *Psychometrika*. 1987; 52:317–332.

39. Klump KL, Bulik CM, Kaye WH, Treasure J, Tyson E. Academy for Eating Disorders position paper: Eating disorders are serious mental illnesses. *International Journal of Eating Disorders*. 2009; 42:97–103. [PubMed: 18951455]
40. Tissot AM, Crowther JH. Self-oriented and socially prescribed perfectionism: Risk factors within an integrative model for bulimic symptomatology. *Journal of Social and Clinical Psychology*. 2008; 27:734–755.
41. Stice E, Schupak-Neuberg E, Shaw HE, Stein RI. Relation of media exposure to eating disorder symptomatology: An examination of mediating mechanisms. *Journal of Abnormal Psychology*. 1994; 103:836. [PubMed: 7822589]
42. Tozzi F, Aggen SH, Neale BM, Anderson CB, Mazzeo SE, Neale MC, et al. The structure of perfectionism: A twin study. *Behavior Genetics*. 2004; 34:483–494. [PubMed: 15319571]
43. Purcell S. Variance components models for gene-environment interaction in twin analysis. *Twin Research*. 2002; 5:554–571. [PubMed: 12573187]
44. Plomin R, Corley R, DeFries JC, Fulker DW. Individual differences in television viewing in early childhood: Nature as well as nurture. *Psychological Science*. 1990; 1:371.
45. Dohnt H, Tiggemann M. The contribution of peer and media influences to the development of body satisfaction and self-esteem in young girls: A prospective study. *Developmental Psychology*. 2006; 42:929–936. [PubMed: 16953697]
46. Field AE, Camargo CA Jr, Taylor CB, Berkey CS, Roberts SB, Colditz GA. Peer, parent, and media influences on the development of weight concerns and frequent dieting among preadolescent and adolescent girls and boys. *Pediatrics*. 2001; 107:54. [PubMed: 11134434]
47. Harrison K, Hefner V. Media exposure, current and future body ideals, and disordered eating among preadolescent girls: A longitudinal panel study. *Journal of Youth and Adolescence*. 2006; 35:146–156.
48. Moriarty CM, Harrison K. Television exposure and disordered eating among children: A longitudinal panel study. *Journal of Communication*. 2008; 58:361–381.
49. Vaughan KK, Fouts GT. Changes in television and magazine exposure and eating disorder symptomatology. *Sex Roles*. 2003; 49:313–320.
50. Field AE, Camargo CA Jr, Taylor CB, Berkey CS, Colditz GA. Relation of peer and media influences to the development of purging behaviors among preadolescent and adolescent girls. *Archives of Pediatrics and Adolescent Medicine*. 1999; 153:1184. [PubMed: 10555723]
51. Martínez-González MA, Gual P, Lahortiga F, Alonso Y, Irala-Estévez J, Cervera S. Parental factors, mass media influences, and the onset of eating disorders in a prospective population-based cohort. *Pediatrics*. 2003; 111:315–320. [PubMed: 12563057]
52. Becker AE, Burwell RA, Herzog DB, Hamburg P, Gilman SE. Eating behaviours and attitudes following prolonged exposure to television among ethnic Fijian adolescent girls. *The British Journal of Psychiatry*. 2002; 180:509. [PubMed: 12042229]
53. Keel PK, Klump KL. Are eating disorders culture-bound syndromes? Implications for conceptualizing their etiology. *Psychological Bulletin*. 2003; 129:747–769. [PubMed: 12956542]
54. Silventoinen K, Sammalisto S, Perola M, Boomsma DI, Cornes BK, Davis C, et al. Heritability of adult body height: a comparative study of twin cohorts in eight countries. *Twin Research*. 2003; 6:399–408. [PubMed: 14624724]
55. Martin NG, Eaves LJ, Kearsley MJ, Davies P. The power of the classical twin study. *Heredity*. 1978; 40:97–116. [PubMed: 272366]
56. Klump KL, Holly A, Iacono WG, McGue M, Willson LE. Physical similarity and twin resemblance for eating attitudes and behaviors: a test of the equal environments assumption. *Behavior Genetics*. 2000; 30:51–58. [PubMed: 10934799]

\$watermark-text

\$watermark-text

\$watermark-text

Parameter Estimates and Model Fit Comparisons

Table 1

	Parameter Estimates			Model Fit Statistics			
	A	C	E	-2lnL (df)	-2lnLΔ (df)	p	AIC
ACE	.29 (.00-.55)	.12 (.00-.47)	.58 (.44-.74)	3.36 (6)	--	--	-8.64
AE	.43 (.28-.55)	--	.57 (.44-.72)	3.57 (7)	0.21 (1)	.65	-10.43
CE	--	.37 (.23-.49)	.63 (.51-.77)	4.59 (7)	0.99 (1)	.32	-9.41
E	--	--	1.0 (1.0-1.0)	29.55 (8)	26.19 (2)	>.01	13.55

Note. A= additive genetic effects, C= shared environmental effects, E = nonshared environmental effects; -2lnL, -2 times the log likelihood; -2lnLΔ, Change in -2 times the log likelihood. Columns for A, C, and E indicate standardized parameter estimates with confidence intervals in parenthesis. All AICs were calculated by taking the difference in -2lnL values between a baseline, unrestricted model [i.e. a model that freely estimates variances, covariances and means; -2lnL=940.84 (df=333)] and all other models.