GENETIC AND ENVIRONMENTAL RISK FOR THIN-IDEAL INTERNALIZATION: AN INVESTIGATION USING CLASSIC TWIN METHODOLOGY AND THE CO-TWIN CONTROL DESIGN

By

Jessica Lynn Suisman

A DISSERTATION

Submitted to
Michigan State University
in partial fulfillment of the degree requirements
for the degree of

Psychology – Doctor of Philosophy

2014

ABSTRACT

GENETIC AND ENVIRONMENTAL RISK FOR THIN-IDEAL INTERNALIZATION: AN INVESTIGATION USING CLASSIC TWIN METHODOLOGY AND THE CO-TWIN CONTROL DESIGN

By

Jessica Lynn Suisman

Thin-ideal internalization has received increasing support as an important risk factor in the development of disordered eating attitudes/behaviors and eating disorders. However, relatively little is known about the etiology of thin-ideal internalization itself. The current research used a series of studies to better understand risk factors for thin-ideal internalization during adolescence.

Study 1 investigated whether known phenotypic changes in thin-ideal internalization across adolescence correspond to developmental changes in etiological (i.e., genetic and environmental) risk. Participants included 846 female twins (ages 8-25 years) from the Michigan State University Twin Registry (MSUTR). 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. Phenotypic analyses generally indicated significant increases in thin-ideal internalization across age and pubertal development. With few exceptions, twin models suggested no significant differences in etiologic effects across development. At all developmental phases, environmental influences were most important in the etiology of thin-ideal internalization, with genetic, shared environmental, and nonshared environmental influences accounting for roughly 10%, 30%, and 60%, respectively, of the total variance. Findings suggest that despite mean-level increases in thin-ideal internalization across

development, the relative influence of genetic versus environmental risk does not shift across this period, with the majority of variance accounted for by environmental factors. Results suggest that early risk factors for thin-ideal internalization are likely to be important across development, and mean-level increases in thin-ideal internalization may reflect increases in the magnitude/strength of environmental risk across this period.

Study 2 examined if affiliation with body-conscious peer groups may influence thin-ideal internalization through socialization processes (e.g., conversations focused on thinness) versus selection processes (e.g., selection into body-conscious peer groups) using co-twin control methodology. Participants included 392 female twins (ages 8-15) from the MSUTR. Thin-ideal internalization and peer group characteristics were assessed via self-report questionnaires. Co-twin control analyses examined whether twin discordance in exposure to weight-focused peers predicted within-twin pair discordance in thin-ideal internalization. Within co-twin control analyses, predictive effects in monozygotic (MZ) and dizygotic (DZ) twins suggest socialization effects, as increased exposure to weight-focused peers would be associated with increased risk for thin-ideal internalization in one co-twin relative to the other, regardless of the degree of genetic and/or environmental sharing. Analyses suggested a role for socialization, as increased exposure to weight-focused peers predicted increased thin-ideal internalization in MZ twin pairs. Results in DZ twins were less consistent, but overall were similar to results in MZ twins. Findings supported etiological theories that suggest socialization processes in the association between weight-focused peers and thin-ideal internalization. Longitudinal and observational research is needed to confirm causal effects and identify peer socialization processes that increase thin-ideal internalization risk.

This dissertation is dedicated to my parents,
for their immeasurable support and encouragement.

ACKNOWLEDGMENTS

This dissertation would not have been possible without the ongoing encouragement of mentors, colleagues, family, and friends who supported me throughout the completion of graduate school. I am especially grateful to my advisor, Dr. Kelly Klump, for her unconditional support, guidance, and mentoring since I began working with her as an undergraduate student nine years ago. Kelly's contagious passion for research is the reason that I decided to pursue graduate school in clinical psychology, and she will continue to be a role model to me as a scientist, teacher, and practitioner. The impact Kelly has had on me both professionally and personally is immense, and I feel grateful, and proud, to have been mentored by someone that I so look up to.

I also feel fortunate to have had the opportunity to work with my committee members, Drs. Alex Burt, Jason Moser, and NiCole Buchanan, and I thank them each for their training, feedback, and perspectives. They have all been excellent resources for both my research and clinical training through various interactions in courses, clinical science forums, committee work, and clinical supervision. A special thank you to Alex, who played an important role in my research training through my experiences working in her lab and taking her behavioral genetics courses. She taught the twin moderation models used in this dissertation when I was a student in her course, which was key to me being able to understand and implement the models used herein.

I could not have completed this program without the support of my family and friends. I thank my parents, Gary and Kathy, for their support and confidence in me, even when my own confidence faded. I was able to complete graduate school, and this research project, because of

them. My siblings, Marc and Sara, provided a wonderful space for laughter. Thank you to my friends, both in the department and in the “real world”, who have been there to support me through every celebration as well as periods of stress or tight deadlines. Finally, I want to thank my partner, Mike, for his love, support, understanding, and willingness to stand by me through all of graduate school, including not only the successes, but also the difficult moments of doubt. His support and love never wavered through all of the demands that graduate school placed upon me, even when it required me to move away for a year to complete my internship training. Thank you to all of you, and I love you all very much.

I would also like to acknowledge the sources of funding for this project, the National Institute of Mental Health award numbers R01MH092377-02 and R01MH0820-54, both awarded to Dr. Kelly Klump.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

KEY TO ABBREVIATIONS

AIC = Akaike's Information Criteria

BIC = Bayesian Information Criterion

BMI = Body Mass Index

DE = Disordered Eating

DZ = Dizygotic

MEBS = Minnesota Eating Behavior Survey

MLM = Multilevel Model

MSUTR = Michigan State University Twin Registry

MZ = Monozygotic

PDS = Pubertal Development Scale

SATAQ-3 = Sociocultural Attitudes toward Appearance Questionnaire, version 3

CHAPTER 1: Risk Factors for Thin-Ideal Internalization

Eating disorders (i.e., anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified) are serious psychiatric disorders that are associated with significant medical and psychiatric consequences and greatly limit quality of life (Klump, Bulik, Kaye, Treasure, & Tyson, 2009). Full-threshold eating disorders are relatively rare, with prevalence estimates of approximately 0.5-1% for anorexia nervosa, 1-2% for bulimia nervosa, and 4-5% for eating disorder not otherwise specified (Hay, Mond, Buttner, & Darby, 2008; Hudson, Hiripi, Pope Jr, & Kessler, 2007; Keski-Rahkonen et al., 2010; Machado, Machado, Gonçalves, & Hoek, 2007). However, more mild, subthreshold symptoms (e.g., chronic dieting, occasional binge eating) are very common, with some studies estimating the prevalence of these behaviors in college women to be as high as 68% (Mintz & Betz, 1988). Although relatively common, these subthreshold eating disordered behaviors are also associated with decreased self esteem, higher levels of body dissatisfaction, and interference with life activities (Mintz & Betz, 1988). Given the seriousness of eating disorders and subthreshold symptoms, it is critical to understand risk factors for the development of these disorders to aid in treatment efforts, and, better yet, prevent the development of these disorders.

Thin-ideal internalization (i.e., the acceptance and adherence to sociocultural beauty standards of thinness) has received increasing support as an important risk factor in the development of disordered eating attitudes and behaviors and eating disorders (Stice, 2002; Thompson & Stice, 2001). Generally, thin-ideal internalization has been conceptualized as a mediator in the association between exposure to sociocultural risk factors (i.e., factors associated with the thin-ideal that permeates Western society) and the development of body dissatisfaction, disordered eating, and eating disorders. For example, the tripartite model of body dissatisfaction

and eating disturbance (Keery, van den Berg, & Thompson, 2004; Thompson, Heinberg, Altabe, & Tantleff-Dunn, 1999) postulates that sociocultural influences are reinforced and perpetuated by three primary factors: the media (i.e., television, advertisements, magazines, etc.), parents (e.g., parental focus on weight/dieting) and peers (e.g., peer discussions about dieting, weight-based teasing). In the tripartite model, thin-ideal internalization and social comparison (i.e., evaluating and comparing oneself to others) are thought to emerge as a result of exposure to these three primary factors. It is through the development of thin-ideal internalization and social comparison that body dissatisfaction and other disordered eating attitudes and behaviors are thought to develop. Indeed, thin-ideal internalization has repeatedly been shown to operate as hypothesized in the tripartite model across several investigations (Keery et al., 2004; Shroff & Thompson, 2006b; Yamamiya, Shroff, & Thompson, 2008).

The importance of thin-ideal internalization is further highlighted when examining the literature on prevention programs for eating disorders. Initial efforts toward the prevention of eating pathology focused on psychoeducation on eating disorders (e.g., teaching about the dangers associated with eating disordered behaviors). Unfortunately, these prevention programs were ineffective, and, in some cases, predicted increased levels of eating pathology, likely as a result of participants learning new weight loss techniques (Stice & Shaw, 2004). However, more recent prevention programs that focus on decreasing thin-ideal internalization have been shown to be quite effective (Coughlin & Kalodner, 2006; Stice, Chase, Stormer, & Appel, 2001; Stice, Mazotti, Weibel, & Agras, 2000; Yamamiya, Cash, Melnyk, Posavac, & Posavac, 2005). These interventions use techniques such as teaching participants about the ways in which media images are altered (“media literacy”) and dissonance techniques that help individuals make arguments against the internalization of the thin-ideal. These techniques have been shown to successfully

decrease not only thin-ideal internalization, but disordered eating symptoms (e.g., body dissatisfaction, dieting, bulimic symptoms), particularly in individuals at high risk for the development of eating pathology. This evidence suggests that thin-ideal internalization may be a critical variable for ongoing efforts to decrease eating pathology, particularly since girls as young as three years old have been shown to internalize the thin-ideal (Harriger, Calogero, Witherington, & Smith, 2010).

Interestingly, these prevention programs have focused almost entirely on just *one* potential risk factor for thin-ideal internalization; media influences. It may be possible to create even more effective prevention techniques that could reach broad audiences if risk factors for thin-ideal internalization are more clearly elucidated and, as a result, incorporated into prevention efforts. Unfortunately, perhaps since thin-ideal internalization has conceptually been categorized as a risk factor rather than an outcome of interest, little research has examined what risk factors lead to the development of thin-ideal internalization. Studies that have been conducted have shown significant associations with risk factors for thin-ideal internalization as proposed in the tripartite model (i.e., family, peers, and media), but these studies are limited by their correlational designs (Keery, Boutelle, Van Den Berg, & Thompson, 2005; Keery et al., 2004; Shroff & Thompson, 2006b; Yamamiya et al., 2008).

In addition to the overall lack of research on potential environmental risk factors for thin-ideal internalization, prior work has almost entirely ignored the possibility that genetic factors contribute to individual differences in thin-ideal internalization. However, there is reason to believe that genetic factors may be involved. Most psychological/behavioral constructs, including the disordered eating symptoms that thin-ideal internalization predicts (e.g., body dissatisfaction, eating disorder symptoms) are known to be significantly heritable, with estimates

often exceeding 40% (Malouff, Rooke, & Schutte, 2008; Suisman et al., 2012). Moreover, although all (or most) women in Western cultures are exposed to environmental factors that reinforce the thin-ideal (e.g., thin-ideal media), only some women ultimately develop high levels of thin-ideal internalization. Genetic factors may help to explain who does, and who does not, internalize the thin-ideal within these cultures.

Given the lack of research on risk factors for thin-ideal internalization, the aims of the proposed set of studies are to use twin methodology to comprehensively examine genetic and environmental risk factors for thin-ideal internalization. The first study will use twin study methods to determine the extent to which genetic and/or environmental influences explain the variance in thin-ideal internalization across development. Importantly, this study will extend previous research in this and other areas by examining whether the proportion of genetic versus environmental influences vary or remain relatively similar across adolescent age and pubertal development. These findings will be particularly important because, despite the developmental nature of thin-ideal internalization (i.e., increases in thin-ideal internalization across adolescent ages and pubertal development), prior studies of risk factors for thin-ideal internalization have generally not taken a developmental approach (Hermes & Keel, 2003). Thus, findings from study 1 will be the first to provide information on developmental differences and similarities in genetic and environmental influences across adolescence and puberty.

In the second study, co-twin control methodology will be used to further elucidate the origins of the associations between thin-ideal internalization and one environmental risk factor that has been highlighted in the tripartite model: influences of weight-focused peer groups. Specifically, this study will examine extent to which the association between body-conscious peer groups and thin-ideal internalization is environmentally mediated (i.e., due to socialization

effects), or, instead, whether the association is due to selection effects. Selection effects occur when exposure to environmental risk factors is nonrandom; that is, a third variable (e.g., genetic predispositions, life events) causes the exposure to environmental risk factors (body-conscious peer groups) which then increases thin-ideal internalization. The co-twin control method can parse apart socialization versus selection effects by comparing outcomes in reared-together twins who have experienced differing degrees of exposure to body-conscious peer groups. If the socialization effects account for associations between body-conscious peer groups and thin-ideal internalization, it is expected that the twin who was exposed to greater levels of body-conscious peer groups will have higher levels of thin-ideal internalization. If twin pairs have relatively equal levels of thin-ideal internalization despite differing degrees of exposure to body-conscious peer groups, it is inferred that other circumstances that the twins share (e.g., genes, life events) were "selection" factors that led to similar levels of thin-ideal internalization in both twins, rather than the environmental exposure factor.

Results of these studies will greatly extend the existing literature on the etiology of thin-ideal internalization by examining the validity of prior assumptions that body-conscious peer groups are an environmentally mediated risk factor for thin-ideal internalization. Further, findings are expected to allow for the development of specific hypotheses regarding environmental and/or biological mechanisms that contribute to thin-ideal internalization and during which developmental periods these mechanisms may be the most important. The more comprehensive etiological models for thin-ideal internalization that will result from this and future research will ultimately inform and aid in preventing the development of clinically significant symptoms of eating pathology (e.g., strict dieting, eating disorders).

CHAPTER 2: Genetic and Environmental Influences on Thin-Ideal Internalization Across Development (Study 1)[1]

ABSTRACT

Objective: Mean-levels of thin-ideal internalization increase across 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, developmental 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. Methods: Participants were 846 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 generally indicated significant increases in thin-ideal internalization across age and pubertal development. With few exceptions, twin models suggested no differences in etiologic effects across development. At all developmental phases, environmental influences were most important in the etiology of thin-

[1] An amended version of Chapter 2 is also being published in the *International Journal of Eating Disorders*, a publication by John Wiley & Sons. The full citation for the manuscript is: Suisman, J.L., Thompson, J.K., Keel, P.K., Burt, S.A., Neale, M., Boker, S., Sisk, C., & Klump, K.L. (in press). Genetic and environmental influences on thin-ideal internalization across puberty and preadolescent, adolescent, and young adult development. *International Journal of Eating Disorders.*

ideal internalization, with genetic, shared environmental, and nonshared environmental influences accounting for roughly 10%, 30%, and 60%, respectively, of the total variance. Discussion: Despite mean-level increases in thin-ideal internalization across development, the relative influence of genetic versus environmental risk does not shift across this period, with the majority of variance accounted for by environmental factors. Findings are significant in suggesting that early risk factors for thin-ideal internalization are likely to be important across development, and mean-level increases in thin-ideal internalization may reflect increases in the magnitude/strength of environmental risk across this period.

INTRODUCTION

Thin-ideal internalization (i.e., the acceptance of and adherence to sociocultural beauty ideals for women that focus on thinness) has emerged as an important risk factor in the development of body dissatisfaction, disordered eating, and eating disorders (Thompson & Stice, 2001). Specifically, multiple cross-sectional and prospective studies have supported the role of thin-ideal internalization in the development of eating problems (e.g., body dissatisfaction, dieting; Thompson & Stice, 2001), and eating disorder prevention programs that aim to decrease thin-ideal internalization have been effective in decreasing disordered eating (Stice, Shaw, & Marti, 2007). Given the prominent role of thin-ideal internalization in the development of disordered eating, research on the etiology of thin-ideal internalization is also needed. Indeed, the identification of risk factors for thin-ideal internalization will likely lead to improved understanding and prevention of disordered eating and eating disorders.

Research on risk for thin-ideal internalization has focused on the role of environmental risk factors that are thought to teach and reinforce beauty ideals of thinness. For example, one model of the etiology of thin-ideal internalization, the tripartite model of body dissatisfaction and

eating disturbance (Keery et al., 2004; Shroff & Thompson, 2006b; Yamamiya et al., 2008), posits that three primary risk factors lead to thin-ideal internalization: images of thin women in the media (i.e., in television, advertisements, magazines, etc.), parental/family influences (e.g., parental focus on weight/dieting), and peer influences (e.g., peer focus on thinness, weight related teasing). Although research on the tripartite model has demonstrated that hypothesized media, peer, and parental risk factors are indeed associated with thin-ideal internalization (Keery et al., 2004; Shroff & Thompson, 2006b; Yamamiya et al., 2008), further research is needed to confirm the direction of these effects, as current studies are limited by cross-sectional designs.

In addition to the environmentally mediated risk factors suggested by the tripartite model, it was recently 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 (Suisman et al., 2012). 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 (Suisman et al., 2012). 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.

In addition to significant genetic effects, the prior twin study of thin-ideal internalization also suggested significant non-shared environmental influences on thin-ideal internalization (Suisman et al., 2012). Non-shared environmental risk factors are those that make siblings different from one another (e.g., differential life experiences such as different peer groups, different life events). Shared environmental influences, which are environmental effects that make siblings more similar to one another (e.g., broad sociocultural influences, shared life

events) were found to be small and non-significant. These findings were somewhat surprising, as it was expected that the significant cultural influences on thin-ideal internalization (e.g., images in TV, magazines, and other forms of media) would be captured by estimates of the shared environment, since these influences are shared among most women in Western cultures and would conceivably act to make them more similar to one another. Instead, our findings suggested that the influence of the *nonshared* micro-environment, rather than *shared* environmental influences, appeared to be most important in explaining individual differences in thin-ideal internalization. The lack of shared environmental effects, however, does not imply that broad sociocultural influences that most women in Western cultures share (e.g., thin-ideal media) are unimportant in the development of thin-ideal internalization. Instead, results suggest that these influences operate at the level of the nonshared micro-environment that acts to make siblings different from one another. For example, media influences may differentially increase thin-ideal internalization in one twin relative to another, perhaps as a result of differential exposure to media images within a twin pair (e.g., one twin spends more time reading fashion magazines that reinforce the thin-ideal while the other twin spends more time engaging in non-media hobbies).

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 the possibility that there are developmental differences in etiologic effects, particularly across adolescence. Mean levels of thin-ideal internalization have been shown to increase across adolescence (Durkin & Paxton, 2002; Suisman et al., 2012) and predict the development of eating pathology (Thompson & Stice, 2001). 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 (Hermes & Keel, 2003).

Developmental increases in mean levels of a phenotype can be an indicator of key etiological shifts that should be examined as well. For example, observed mean level increases in disordered eating across adolescence (Klump, McGue, & Iacono, 2000) were previously hypothesized to signal potential changes in *etiology* (i.e., genetic and environmental influences; (Klump et al., 2000). Cross-sectional and longitudinal twin studies subsequently confirmed that this was the case by showing significant age differences in genetic and environmental influences on disordered eating such that the heritability of disordered eating was negligible in pre-adolescence (age 11), but became significant (i.e., approximately 50% of variance) in middle adolescence (age 14) and beyond (i.e., ages 16-40 years; Klump, Burt, McGue, & Iacono, 2007; Klump, Burt, et al., 2010; Klump et al., 2000). Although estimates of the nonshared environment remained significant across all age groups, the effects of the shared environment showed substantial changes that were the opposite of those observed for genetic influences: shared environmental influences decreased from accounting for 40% of the variance in disordered eating in pre-adolescence to 10% or less from middle adolescence into middle adulthood.

Given that the increase in genetic influences occurred during the transition from pre-adolescence (age 11) to mid-adolescence (age 14), the time when most girls experience puberty (mean age of menarche = approximately 12.5 years; Chumlea et al., 2003; Herman-Giddens et al., 1997), it was hypothesized that pubertal development drives changes in genetic effects. Research that examined genetic and environmental influences on disordered eating by pubertal status confirmed this hypothesis. Specifically, in twins in pre-to early puberty, genetic factors accounted for a negligible proportion of the variance (0%) in disordered eating, while in late puberty, genetic factors accounted for approximately 50% of the variance. Shared environmental influences again showed the opposite effect; they accounted for a significant proportion of the

variance in pre-to early puberty (50%), and decreased to a negligible proportion of the variance in late puberty (0%; Culbert, Burt, McGue, Iacono, & Klump, 2009; Klump, McGue, & Iacono, 2003; Klump, Perkins, Burt, McGue, & Iacono, 2007). Importantly, these effects accounted entirely for the age differences observed in prior studies, as genetic and environmental effects in young pubertal twins (i.e., twins who were pubertal at age 11) were identical to genetic and environmental influences in older, pubertal twins (i.e., they showed significant genetic influences and negligible shared environmental influences; Klump, Burt, et al., 2007; Klump et al., 2003). These findings have been especially 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 (Klump, Keel, Sisk, & Burt, 2010).

As noted by others (Hermes & Keel, 2003), it is possible that changes in genetic and shared environmental effects on thin-ideal internalization follow the same pattern as those for disordered eating. Although thin-ideal internalization and disordered eating are independent constructs, the phenotypes do correlate moderately (Calogero, Davis, & Thompson, 2004; Thompson, van den Berg, Roehrig, Guarda, & Heinberg, 2004) and they follow similar developmental trajectories (i.e., mean level increases across puberty; Hermes & Keel, 2003). Further, in post-pubertal samples, the magnitude of genetic and environmental influences on disordered eating and thin-ideal internalization are similar (i.e., no shared environmental influences, significant genetic influences), which provides some evidence for similar patterns of etiology across phenotypes (Klump et al., 2003; Klump, Perkins, et al., 2007; Suisman et al., 2012). Finally, there appear to be some common etiological mechanisms for thin-ideal internalization and disordered eating in late adolescence. Specifically, previous analyses have shown a genetic correlation between thin –ideal internalization and disordered eating of .72,

suggesting at least some common genetic etiology (Suisman et al., in preparation). Given these associations, thin-ideal internalization may indeed show similar developmental differences in etiologic effects as those observed for disordered eating, i.e., increasing genetic and decreasing shared environmental effects across age/puberty.

Given the above, 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=846) sample of same-sex female twins (ages 8-25 years). To ensure that effects are specific to thin-ideal internalization, developmental differences in genetic and environmental effects were also examined while controlling for disordered eating. Specificity of effects are important to establish given phenotypic and genetic overlap in thin-ideal internalization and disordered eating (see above) and the need to identify etiological risk factors that contribute uniquely to thin-ideal internalization.

METHODS

Participants

Participants were drawn from a sample of 848 same-sex female twins between the ages of 8 and 25 (mean = 15.06, SD = 3.93) from the Michigan State University Twin Registry (MSUTR; Burt & Klump, 2012; Klump & Burt, 2006). Missing data was minimal for thin-ideal internalization (N = 42, 4.9%); disordered eating (N = 6, 0.7%), pubertal group status (N = 14; 1.6%); and body mass index (N = 3; 0.3%), and twin pairs were excluded from analyses only when data was unavailable for both twins. Thus, final age moderation models included 846 twins (Monozygotic [MZ] = 454, dizygotic [DZ] = 392), and final pubertal moderation models included 830 twins (MZ = 444, DZ = 386)

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; Burt & Klump, 2012; Klump & Burt, 2006). Twins included in this study were participants in one of two ongoing studies within the MSUTR, 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+). Comparisons with prior research have indicated that the use of these inclusion/exclusion criteria do not inadvertently affect the range or variability in thin-ideal internalization scores (Suisman et al., 2012). 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; Burt & Klump, 2012; Culbert et al., 2009; Klump et al., 2013), a pattern that was consistent with the current sample (80% Caucasian, 14% African American, 6% Multiracial, 0.7% Asian, 0.2%American Indian or Alaka Native). Participant parental income also represented a range of socioeconmic backgrounds, with 8% of participants reporting family income less than $20,000 anually; 17% reporting $20,000-$40,000; 21% reporting $40,000-$60,000; 26% reporting $60,000-$100,000; and 23% reporting family incomes greater than $100,000 anually.

Notably, 42% of participants in the current study were also included in the prior twin study on thin-ideal internalization (Suisman et al., 2012). The aim of the Suisman et al. (2012)

study was to examine genetic/environmental effects in post-pubertal twins only, so all pre-pubertal twins included in the present sample (N = 260) are unique to the current study. Additionally, a portion of post-pubertal twins (N = 216) for whom data collection had not yet been completed at the time of the Suisman et al. (2012) study are unique to the current investigation.

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 (Lykken, Bouchard, McGue, & Tellegen, 1990; Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). 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. In cases where results from any of these raters (i.e., twins, parent, and research assistants) were not in agreement (29% of sample), 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 the Sociocultural Attitudes toward Appearance Questionnaire-3 (SATAQ-3; Thompson et al., 2004). The 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). In the past, the SATAQ-3 has been

administered either with all 30 items positively keyed or with eight of the items reverse keyed (Markland & Oliver, 2008). As suggested by Thompson et al., (2004), the version that included eight reverse-scored items was utilized in the present study.

The present study focused on the 9-item, general internalization subscale that assesses the extent to which participants want to look like individuals from various media sources (e.g., television, magazines, movies). The general internalization subscale was used in the prior twin study on thin-ideal internalization (Suisman et al., 2012) and is commonly used to assess thin-ideal internalization in risk factor and intervention studies (Cafri, Yamamiya, Brannick, & Thompson, 2005; Coughlin & Kalodner, 2006). This subscale differentiates individuals with eating disorders from controls and demonstrates excellent internal consistency (*a*'s >.90) in prior samples (Calogero et al., 2004; Thompson et al., 2004), as well as in our current sample (see Table 1).

Disordered Eating. As described above, possible moderating effects of age and/or puberty on thin-ideal internalization while controlling for disordered eating were also examined. Overall levels of disordered eating were assessed using the total score of the Minnesota Eating Behavior Survey (MEBS; von Ranson, Klump, Iacono, & McGue, 2005)[2] The MEBS is a 30-item true/false questionnaire that includes items regarding weight preoccupation (i.e., tendency to think about/be concerned with ones weight), body dissatisfaction (i.e., dissatisfaction with body weight and/or shape), binge eating (i.e., actual binge eating or thoughts of binge eating),

[2] The 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.

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). The psychometric properties of the MEBS total score are excellent. Internal consistency is high in prior studies, in twins as young as age 11 (a =0.86) through late adolescence (a =0.89; von Ranson et al., 2005). Internal consistency was also excellent in the current study, even in our youngest (ages 8-12) age group (a =0.87), as well as in older age groups (a's = .87-.88). Additionally, 3-year test-retest reliability for the MEBS total score is also good (Klump et al., 2000). Finally, women with eating disorders score significantly higher on the MEBS scales than control women (Klump et al., 2000; von Ranson et al., 2005). 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 (Culbert et al., 2009; Klump et al., 2000, 2003; Klump, Perkins, et al., 2007). Thus, this scale is particularly useful since the aim of the present study is 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 asses each participant's degree of pubertal development, the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988), 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., Klump, Burt, et al., 2007). 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 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 (Culbert et al., 2009; Petersen et al., 1988), and it was also excellent in the current sample (See Table 1). 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 ([weight]) / [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. Given the modest, but statistically significant association between BMI and thin-ideal internalization (Pearson's $r = .21$, $p<.01$), BMI was partialled out of thin-ideal internalization scores prior to analyses in order to ensure results are not unduly influenced by BMI. For analyses that controlled for disordered eating (see below), we also partialled out MEBS total scores from each twins' thin-ideal internalization score (in addition to partialling out BMI). Thin-ideal internalization scores did not violate normality assumptions (skewness = .41, kurtosis = -.62).

Twin analyses were used to examine if age and/or puberty moderate genetic and environmental effects on thin-ideal internalization, both with and without controlling for disordered eating. Etiological (i.e., genetic and environmental) influences across age and pubertal categories were examined using intraclass twin correlations and twin moderation biometric models. 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 (Culbert et al., 2009; Klump et al., 2003). 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 (Klump, Burt, et al., 2007; Klump, Burt, et al., 2010). In addition to corresponding closely to previously used age categories, these age categories also map on to the timing of changes in etiological risk factors, especially changes in environmental risk across development. Specifically, our age categories roughly capture the relative decreases in time spent with parents, and corresponding increases in time spent with peers, from mid/late childhood (i.e., approximately the 8-12 group) to adolescence (i.e., approximately 13-16 years; Parker, Rubin, Erath, Wojslawowicz, & Buskirk, 2006). The oldest age group encompasses the transitional developmental stage of graduating high school and starting college or full-time work, and living independently from parents (i.e., approximately 17-25 years).[3]

[3] In addition to these categorical analyses, differences in genetic and environmental effects were examined using continuous measures of age (i.e., each participant's age in years) and pubertal development (i.e., the PDS total score for each participant). Results of these models were essentially identical to the categorical models described above. Thus, we only present results from categorical models throughout this paper, particularly because sample sizes at each level of the moderator were much larger for categorical models, while in continuous models, sample

Twin Correlations. Intraclass twin correlations were first computed to offer preliminary indications of moderating effects of age and puberty. Specifically, separate twin correlations were calculated for each age and puberty group in order to examine whether the pattern of co-twin similarity (or dissimilarity) in thin-ideal internalization scores in MZ versus DZ twins changes with age and/or pubertal status. These correlations were computed for thin-ideal internalization controlling only for BMI, followed by analyses controlling for disordered eating and BMI. Within each set of intraclass twin correlations, additive genetic influences are implied if MZ twin correlations are approximately double the DZ twin correlations. Shared environmental influences are suggested if 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. Comparisons of twin correlations across age and puberty groups were used to provide initial indications of moderating effects (or the lack thereof) of age and/or puberty.

Twin Moderation Models. Differences in the etiology of thin-ideal internalization across age and pubertal development were then more precisely evaluated using twin moderation models, both with and without controlling for the effects of disordered eating (Purcell, 2002). Three versions of twin moderation models have been developed to date. The first model, the standard univariate moderation model, is most useful when the moderator is a family-level variable that is shared within twin pairs, as is true for our age moderation models (Purcell, 2002; van der Sluis, Posthuma, & Dolan, 2012). This is because when twin pairs are 100% concordant for the moderator (i.e., age) there is no need to control for co-twin covariance on the moderator,

---

sizes at each level of the moderator were quite limited (e.g., as small as 8 twins, and frequently limited to only 25-50 twins).

which is part of the goal of the other moderation models described below. Thus, standard univariate moderation models were used to examine possible moderating effects of age on the etiology of thin-ideal internalization (Purcell, 2002).

The second type of model, the bivariate moderation model, is used when there is genetic or environmental covariance between the moderator and outcome, which is only possible when the moderator can vary within twin pairs (as is true for puberty) versus a twin pair or family level variable (i.e., age). Essentially, the bivariate moderation model partitions moderation effects into those that are unique to the outcome, and those that fall onto the shared covariance between the moderator and outcome. The bivariate moderation model is critical when there is significant covariance (i.e., a significant genetic correlation, shared environmental correlation, or nonshared environmental correlation) between the moderator and outcome, as false negative moderation results are likely in the standard univariate twin moderation model under these circumstances. In the current sample, however, bivariate twin models suggested no significant covariance between the moderator (puberty) and outcome (thin-ideal internalization; genetic correlation = -.13; 95% CI [-1.0, 1.0], shared environmental correlation =.03; 95% CI [-.19, .29], nonshared environmental correlation = -.004, 95% CI [-.14, .13]). Given the lack of covariance on the etiology of puberty and thin-ideal internalization, bivariate moderation models were not necessary for the current analyses.

Since the bivariate moderation model was not needed in our puberty analyses, the third type of moderation model, the extended univariate moderation model (van der Sluis et al., 2012), was used. 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, Pearson 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 (i.e., if the standard univariate model is used instead of the extended univariate model)[4].

Within both 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., $\beta_{X,}$ $\beta_{Y,}$ and $\beta_{Z;}$ see Figures 1 and 2). 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 Figures 1 and 2), allowing for linear and quadratic genetic, shared, and nonshared environmental moderating effects of age/puberty.

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 (−2lnL) in the least restrictive model(s) (i.e., the linear/quadratic moderator model for

[4] Given the high correspondence in pubertal status among twins (i.e., *r*=.94), it was important to ensure that the extended univariate model was not overly conservative, since twins generally did share pubertal status. Thus, we also examined pubertal moderation effects using standard univariate models, the same models used for age analyses that are recommended for use when the moderator is shared among co-twins. The pattern of results for the standard univariate models were generally the same as those presented herein, and the standard univariate models did not detect any significant moderation paths. Since the standard univariate models are more conservative (i.e., less likely to detect moderation), these results helped to confirm that the extended univariate models presented herein were not preventing detection of moderation effects that would have otherwise been significant.

age, the linear model for puberty) were compared with the −2lnL 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; Akaike, 1987) and the Bayesian Information Criterion (BIC; Reftery, 1995), 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 (Markon & Krueger, 2004), 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 (Markon & Krueger, 2004). Given that the sample size for the present study is relatively moderate, both both AIC and BIC were examined, in addition to the likelihood-ratio chi-square.

Model fitting was conducted using raw data techniques with Mx statistical software (Neale, 1995), which treats missing data at random and allows for the inclusion of all twin pairs, even when one twin has missing data (Little & Rubin, 1987). To further ease interpretation, age and puberty scores were "floored" prior to analyses, such that the minimum score was 0 for both age and puberty. 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 −2lnL value (Purcell, 2002).

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 (Hermes & Keel, 2003;

Thompson & Stice, 2001), thin-ideal internalization was positively and significantly correlated with age, pubertal development, and disordered eating. The 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). This suggests that there are significant mean-level increases in thin-ideal internalization across development are *not* accounted for by developmental differences in levels of disordered eating.

Since twins participating in the ***Twin Study of Hormones and Behaviors across the Menstrual Cycle*** were required to be experiencing regular menstrual cycles to participate in the study and were assigned a maximum PDS total score (i.e., 4) for statistical analyses, we also examined phenotypic associations between pubertal development and thin-ideal internalization only in twins who did complete the PDS (i.e., twins who participated in the ***Twin Study of Hormones and Disordered Eating Across Puberty*, ages 8-15 years).** Interestingly, as shown in Table 1, the association between thin-ideal internalization and pubertal development was small and no longer statistically significant when examining only this younger subgroup of twins, although the association between pubertal development and disordered eating remained significant in this subgroup. This pattern of effects may suggest that mean level increases in thin-ideal internalization from late childhood through young adulthood may be driven by environmental changes during development (e.g., changing sociocultural and peer pressures from late childhood through high school) rather than biological changes related to pubertal status per se.

Twin Correlations

Twin correlations for thin-ideal internalization, stratified by age and pubertal development, are presented in Table 2. Overall, the pattern of twin correlations when not

controlling for disordered eating suggested the possibility of moderation of etiologic influences by age and pubertal status. Specifically, at younger ages and during pre/early pubertal development, MZ and DZ twins had approximately equal twin correlations, or in some cases, DZ twins had higher twin correlations than the MZ twins. These correlations suggested that in the younger/early puberty groups, genetic effects were negligible, with shared and nonshared environmental effects accounting for the majority of the variance in thin-ideal internalization. However, at older ages and more advanced pubertal development, the pattern of twin correlations changed, such that MZ twin correlations were greater than DZ twin correlations, particularly in the mid/late puberty group. These results suggested increases in genetic effects in mid-adolescence and mid-puberty, with subsequent decreases in shared and nonshared environmental effects. However, even in the oldest age groups/pubertal twins, the MZ twin correlations were not double the DZ correlations, which is the expected pattern when genetic effects are robust. Thus, despite some evidence of increasing genetic effects across age/puberty, it was unclear from the twin correlations how strong or significant these effects were.

Twin correlations after controlling for disordered eating suggested a different pattern of results. As shown in Table 2, there were no significant differences between MZ and DZ twins regardless of age or puberty group, and generally, the MZ and DZ twin correlations were approximately equal to one another. This pattern suggested no substantial differences in etiological effects across development, and regardless of developmental stage, shared and nonshared environmental influences appeared to be most important to the etiology of thin-ideal internalization. The only exception to this pattern was some suggestion of genetic effects in the middle age group (i.e., ages 13-16), given that the MZ twin correlation was larger than the DZ twin correlation (although these differences were not statistically significant). Overall, however,

the pattern of similar twin correlations in MZ and DZ twins suggested that once controlling for disordered eating, there are no significant differences in genetic or environmental effects across development.

Twin Moderation Models

Age: Results from the twin models are summarized in Tables 3 and 4 and Figure 3. Path and moderator coefficients (see Table 4) were used to create plots of the estimates from the full and best-fitting models (Figure 3). 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.

Prior to controlling for disordered eating, the full and linear models reflect the pattern observed from the twin correlations, as there was some indication of moderation effects (see Table 3 and Figure 3a). Further, model fit statistics supported the linear moderation model as best-fitting, given the lowest AIC value and the significantly worse fit of the no moderation model (p =.04; see Table 3). However, none of the moderation paths in the linear (or full) models were statistically significant (see Table 4), and BIC did suggest a better fit of the no moderation model (see Table 3). Thus, although there was some ambiguity regarding which model provided the best fit to the data, results were clear in suggesting that none of the moderation paths were statistically significant. In the no moderation model, standardized estimates for genetic influences were nonsignificant and accounted for approximately 10% of the variance. Shared environmental influences were statistically significant and were estimated to account for about 35% of the variance. Nonshared environmental effects were also statistically significant, accounting for about 55% of the variance in thin-ideal internalization.

Similarly, after controlling for disordered eating, the full model suggested possible non-linear moderating effects for genetic and shared environmental influences (see Figure 3b), but there were no statistically significant moderating paths in the full or linear moderation models. Additionally, the best fitting model was the no moderation model, as indicated by the non-significant change in chi-square *(p* = .06*)*, and smallest BIC values as compared to the linear and full models (see Table 3). As shown in Figure 3, in the best-fitting no moderation model, there were no differences in the magnitude of genetic or environmental effects across age groups.

Taken together, age moderation results generally suggested that there were no significant changes in etiological effects across development, although differences may be present that we were unable to detect in the current study. Additionally, environmental influences consistently accounted for more variance in thin-ideal internalization scores than genetic influences.

Puberty: Results from the puberty moderation models are summarized in Tables 3 and 4 and in Figure 4. 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). Again, path and moderator coefficients (see Table 4) were used to create plots of the unstandardized estimates from the linear and best-fitting models (See Figures 4a-4f). Prior to controlling for disordered eating, the linear models provided the best fit to the data, as suggested by the smallest AIC and BIC values and the significant decrement in fit of the no moderation model ($p < .001$). The linear model suggested significant increases in genetic influences across puberty (See Table 4 and Figure 4a), with standardized estimates suggested increases from 5% of the variance in pre/early puberty, to 29% of the variance in middle/late puberty. There were no significant changes in shared or nonshared environmental effects across this period, with shared

environmental effects accounting for roughly 25% of the variance, and nonshared environmental effects accounting for roughly 50% of the variance..

After controlling for disordered eating, the linear model again provided the best fit to the data, as indicated by the lowest AIC value and the significant decrement of fit of the no moderation model *(p* = .001). However,, none of the moderating effects were statistically significant, and BIC was smaller in the no moderation model. Thus, after controlling for disordered eating, there appears to be no etiological moderation between pre-puberty and post-puberty. Figures 4d demonstrates the equal estimates of genetic and environmental effects across pubertal development derived from the no moderation model. In this model, standardized estimates for genetic influences were nonsignificant and accounted for approximately 5% of the variance. Shared environmental influences were also not statistically significant but were estimated to account for 26% of the variance. Nonshared environmental effects were statistically significant and accounted for 64% of the variance.

DISCUSSION

This was the first study to examine genetic and environmental influences on thin-ideal internalization across age and pubertal development. Results generally demonstrated similarities rather than differences in estimates of genetic and environmental effects across development, with only one statistically significant moderating effect detected across all models. Findings were largely consistent with and without controlling for disordered eating and highlighted the much more prominent role for environmental influences on thin-ideal internalization than genetic factors across all developmental stages examined.

Findings in the current study differ from the pattern of developmental effects previously observed for disordered eating (Klump, Burt, et al., 2007; Klump, Perkins, et al., 2007). Indeed,

the current results suggest a significant increase in genetic effects across puberty only in models that do not control for disordered eating. However, no changes in other etiological effects were detected (e.g., decreases in shared environmental effects), and no significant changes in the relative influences of genetic and environmental effects on thin-ideal internalization were detected across age groups (regardless of whether disordered eating was accounted for) or across puberty groups (after controlling for disordered eating). Together, these results suggest that although genetic influences on thin-ideal internalization may increase across puberty, these effects are no longer present after accounting for disordered eating. Instead, moderating effects of disordered eating, rather than thin-ideal internalization per se, may be driving the significant genetic moderation effects detected in the puberty model that did not account for disordered eating. Indeed, studies of disordered eating have strongly demonstrated increases in genetic, and decreases in shared environmental, influences across adolescent age and puberty (Klump, Perkins, et al., 2007).

Other differences in findings in the current study versus studies of disordered eating emerged. For example, the present study suggests small (and non-significant) genetic influences on thin-ideal internalization, which differs from the strong and significant genetic effects for disordered eating and eating disorders in mid-puberty and beyond (i.e., 50% or greater; Thornton, Mazzeo, & Bulik, 2011). The present findings suggest the possibility of shared environmental influences on thin-ideal internalization even in late adolescence, a period in which studies of disordered eating have generally suggested no significant influence of the shared environment. Although the effects of the shared environment were not consistently statistically significant, estimates of the shared environment were much more substantial than has been observed for disordered eating (Klump, Perkins, et al., 2007) and would likely be significant

across all models with a larger sample size. These patterns suggest that environmental influences are more important in the etiology of thin-ideal internalization than disordered eating, and these environmental risk factors remain stable across development.

Notably, the larger environmental as compared to genetic effects was not identified in the only previous twin study of thin-ideal internalization (Suisman et al., 2012). However, that 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 differences in sample sizes across studies, as shared environmental effects are difficult to detect when underpowered, with these effects instead loading onto estimates of additive genetic effects (Martin, Eaves, Kearsey, & Davies, 1978). Indeed, even in the current study (N=836), estimates of the shared environment were moderate (i.e., 21-38% of the variance), but were still nonsignificant, supporting the notion that significant shared environmental influences on thin-ideal internalization may be difficult to detect without an even larger sample or different design (e.g., adoption study). Together, findings suggest that there are likely effects of the shared environment on thin-ideal internalization across adolescence, but larger twin samples will be needed to detect them and provide stable estimates of their effects.

Despite the lack of significant differences in etiologic influences on thin-ideal internalization across development, analyses in our full sample did suggest significant mean-level increases in thin-ideal internalization across age and pubertal development. These effects remained significant even when controlling for disordered eating (see Table 1). Interestingly, however, the association between thin-ideal internalization and pubertal development was small and no longer statistically significant when examining only a younger subgroup of twins (i.e., ages 8-15 years) although the association between pubertal development and disordered eating

remained significant in this subgroup (See Table 1). Thus, pubertal development was clearly more predictive of disordered eating than thin-ideal internalization in the younger age groups. As suggested above, this pattern suggests that environmental changes (e.g., increasing sociocultural pressures) that occur with increased age, rather than biological changes related to pubertal status, may drive the mean-level increases observed across our full sample.

The significant mean-level increase in thin-ideal internalization that was observed from childhood through young adulthood may seem to contradict the limited differences in etiological effects 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. In the present case, the proportion of genetic, shared environmental, and nonshared environmental effects do not change across adolescence, but the overall prevalence of these types of risk factors may increase across adolescence/puberty and drive mean-level increases in effects. This possibility is especially interesting as it suggests that many of the early risk factors for thin-ideal internalization remain important in the etiology of thin-ideal internalization across development, and may even become stronger. Given the particularly strong and significant influences of the nonshared environment identified in the current study, it is likely that many of the early risk factors that remain significant across development would fall in this domain. For example, differential thin-ideal media exposure may be a nonshared environmental risk factor for thin-ideal internalization that begins at an early age (Rideout, Foehr, & Roberts, 2010), but increases across age and development. Indeed, although the total number of hours of media usage per day does not vary

largely between 8-18 year olds (Rideout et al., 2010), there are likely increases in the amount of thin-ideal content in the types of media children versus adolescents typically engage with, which may contribute to increases in thin-ideal internalization across this period.

Although this possibility is intriguing, it is important to consider that the current findings only suggest that the relative *magnitude* of genetic and environmental influences are relatively stable across development; results do not necessarily suggest that *identical* etiological influences act on thin-ideal internalization across all age/pubertal groups examined. Thus, in addition to the possibility that the same risk factors for thin-ideal internalization that are most important in childhood remain important in adolescence and early adulthood, 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. In other words, although the magnitude of nonshared environmental influences do not change markedly across development, it is possible that the specific risk factors that contribute to these nonshared environmental effects differ over time. For example, differential exposure to the thin-ideal within a twin pair may emerge in childhood via involvement in different extracurricular activities (e.g., one twin is involved in ballet, while the other twin is focused on piano lessons). These sorts of differences may represent a primary source of nonshared environmental effects in childhood. In adolescence, although differences in extracurricular activities may continue to contribute to differential thin-ideal exposure within the twin pair, other influences may become more prominent, such as differential exposure to weight-focused peer groups. Thus, although the current study suggested no changes in the proportion of variance in thin-ideal internalization that is accounted for by genetic and environmental risk factors across development, future work is still needed to identify which specific environmental risk factors are contributing at different development stages.

Several limitations of this study must be noted. First, 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 (i.e., 500 MZ and 500 DZ pairs; Purcell, 2002), and we had fewer twins in our sample (i.e., 448 MZ twins and 388 DZ twins). Our somewhat small 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 in some of our models, as described above. Secondly, it is possible that a larger sample size would detect significant moderation effects that were not detected in the current study, particularly given that fit statistics frequently supported the selection of the linear model as best-fitting, in the absence of statistically significant moderation paths. However, studies of age and pubertal moderation effects in 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 (Culbert et al., 2009; Klump, Burt, et al., 2007; Klump, Burt, et al., 2010; Klump et al., 2000, 2003; Klump, Perkins, et al., 2007). Therefore, moderating effects on thin-ideal internalization may be less robust or more nuanced, and thus more difficult to detect, than those for disordered eating. Future studies in larger samples, or other behavioral genetic designs that are more effective in identifying shared environmental effects, such as adoption studies, (see Klump, Suisman, Burt, McGue, & Iacono, 2009) are needed to confirm the current findings.

Secondly, it is important to consider that nonshared environmental estimates were the most robust, and in many cases, the only significant, estimates across the moderation models presented herein. Since nonshared environmental estimates include both true nonshared environmental effects as well as random error that would make twins different from one another, it is difficult to know which proportion of the nonshared environmental effects are indeed due to

measurement error (Burt, 2009). The nonshared environmental estimates were particularly high when controlling for disordered eating, suggesting the possibility that partialling out disordered eating score introduced additional error to the measurement of thin-ideal internalization (Lynam, Hoyle, & Newman, 2006). The high internal consistency for the thin-ideal internalization measure (see Table 1) provides some reassurance that measurement error alone may not account for the strong nonshared environmental effects. However, the magnitude of these estimates should be interpreted with caution.

Thirdly, the current study used cross-sectional data. As a result, developmental stability or change within twin pairs as they advanced through puberty could not be examined. Future longitudinal work that follows the same sample of twins across time is needed to replicate the cross-sectional effects identified in the current study. As noted above, genetically informed, longitudinal studies that carefully measure specific environmental risk factors (e.g., differential media exposure) would be most informative for elucidating the specific etiological risk factors contributing to thin-ideal internalization across time.

Finally, this study was confined to twins between the ages of 8-25. Although this is a wide age range, and allowed for examination of twins at all phases of adolescence and pubertal development, the possibility remains that the magnitude of genetic and environmental effects may vary in younger or older age groups than those examined herein. For example, phenotypic data on thin-ideal internalization and related sociocultural constructs is extremely limited in adults older than about age 30 (Cafri et al., 2005; Slevec & Tiggemann, 2011), and thus it is unknown the extent to which women in middle to late adulthood are influenced by thin-ideal internalization and whether etiologic effects identified in the current study would extend to these age groups.

CHAPTER 3: Socialization or Selection Effects? A Co-Twin Control Study of the Association between Body Conscious Peer Groups and Thin-Ideal Internalization (Study 2)

ABSTRACT

Objective: Affiliation with body-conscious peer groups is theorized to cause increases in thin-ideal internalization through socialization processes (e.g., conversations focused on thinness). However, genetic and/or environmental selection could account for these associations if predispositions toward thin-ideal internalization (via genetic or environmental propensities) lead girls to select into body-conscious peer groups. In that case, increased thin-ideal internalization within the group could be due to pre-existing selection factors rather than group socialization processes. The current study used co-twin control methodology to disentangle socialization from selection effects in these associations. Method: Participants included 392 female twins (ages 8-15) from the Michigan State University Twin Registry. Thin-ideal internalization and peer group characteristics were assessed via self-report questionnaires. Co-twin control analyses examined whether twin discordance in exposure to weight-focused peers predicted within-twin pair discordance in thin-ideal internalization. Predictive effects in monozygotic (MZ) and dizygotic (DZ) twins suggested socialization effects, as increased exposure to weight-focused peers would be associated with increased risk for thin-ideal internalization in one co-twin relative to the other, regardless of the degree of genetic and/or environmental sharing. Results: Analyses suggested a role for socialization, as increased exposure to weight-focused peers predicted increased thin-ideal internalization in MZ twin pairs. Results in DZ twins were less consistent, but overall were similar to results in MZ twins. Discussion: Findings supported etiological theories that suggest socialization processes in the

association between weight-focused peers and thin-ideal internalization. Longitudinal and observational research is needed to confirm causal effects and identify peer socialization processes that increase thin-ideal internalization risk.

INTRODUCTION

Thin-ideal internalization (i.e., the acceptance and adherence to sociocultural beauty standards of thinness) has been identified as a risk factor for the development of disordered eating and eating disorders (Stice, 2002; Thompson & Stice, 2001). Indeed, multiple cross-sectional and prospective studies have demonstrated that elevations in thin-ideal internalization predict increased levels of body dissatisfaction, disordered eating behaviors (e.g., dieting), and eating disorders (Thompson & Stice, 2001). Given the role of thin-ideal internalization in the development of disordered eating, improvements in understanding the etiology of thin-ideal internalization itself may be a critical step in preventing the development of thin-ideal internalization and subsequent disordered eating.

The current study aimed to closely examine one risk factor that is often implicated in the development of thin-ideal internalization; body conscious peer groups that are highly focused on body weight, body shape, and dieting. Body-conscious peer groups are a core risk factor in the tripartite model of body dissatisfaction and eating disturbance, which is a leading conceptual theory about how sociocultural factors and thin-ideal internalization lead to the development of disordered eating (van den Berg, Thompson, Obremski-Brandon, & Coovert, 2002). The tripartite model posits that thin-ideal internalization develops from sociocultural influences, such as when peers, family/parents, and media teach and reinforce the cultural ideals of thinness through behaviors such as exposing girls to ideals of thinness, encouraging weight/loss and dieting, pressuring girls to be thin, and/or engaging in weight-related teasing. Thin-ideal

internalization is then hypothesized to lead to the development of disordered eating. In other words, thin-ideal internalization is thought to mediate the association between sociocultural influences and disordered eating.

To date, only cross-sectional investigations have examined the association between body-conscious peer groups and thin-ideal internalization. Findings from these studies have demonstrated that affiliation with weight-focused peer groups (i.e., peers who frequently talk about the thin-ideal and issues such as dieting and weight-loss) is significantly associated with increased thin-ideal internalization, with effect sizes generally in the moderate-to-large range (Clark & Tiggemann, 2006; Jones, Vigfusdottir, & Lee, 2004; Keery et al., 2004; Shroff & Thompson, 2006a; Shroff & Thompson, 2006b; Yamamiya et al., 2008). Researchers have concluded that affiliation with weight-focused peer groups may cause increases in thin-ideal internalization (See Figure 5a; Shroff & Thompson, 2006b). However, there have been no longitudinal or experimental studies on the association between weight-focused peer groups and thin-ideal internalization, limiting the causal inferences that can be drawn.

Rather than affiliation with these peer groups directly causing increases in thin-ideal internalization (i.e., socialization effects), it may be that girls who are already more inclined toward thin-ideal internalization are more likely to select into weight-focused peer groups (i.e., selection effects). Such selection effects would occur if pre-existing genetic and/or environmental factors lead an individual to select into potentially "risky" environments. These types of selection effects can operate in two different ways; in the first scenario, predisposing selection factors cause exposure to the "risk" environment, and the risk environment then causes higher levels of thin-ideal internalization (see Figure 5b). For example, genetic predispositions for thin-ideal internalization may cause an individual to select into environments consistent with

their genotype (e.g., selecting weight-focused peer groups). Exposure to weight-focused peer groups would then further reinforce genetic predispositions for thin-ideal internalization, and lead to increased levels of thin-ideal internalization. This possibility is consistent with the theory of gene-environment correlations, in which an individual's exposure to risk environments is influenced by (i.e., correlated with) their genotype (Scarr & McCartney, 1983).

In the second scenario, main effects of the selection factor (e.g., genetic risk for thin-ideal internalization) directly cause increases in thin-ideal internalization. Additionally, the selection factor causes exposure to "risk" environment (e.g., weight-focused peer groups). In this case, there may be no causal association between the "risk" environment and thin-ideal internalization. Instead, main effects of the predisposing selection factors cause exposure to weight focused peer groups and thin-ideal internalization (See Figure 5c).

Each type of selection effect described above can cause problems when drawing conclusions based on studies that use correlational designs to examine associations between body-conscious peer groups and thin-ideal internalization. Specifically, in each of the above scenarios, an association would emerge between body-conscious peer groups and thin-ideal internalization, which may led researchers to believe that exposure to these peer groups directly caused thin-ideal internalization. However, these correlational designs are not able to test for the presence of the selection effects described above, including both the gene-environment correlation processes (Figure 5b) as well as main effects of the selection factors (Figure 5c). In the case of gene-environment correlations, correlational designs "miss" the fact that predisposing factors cause exposure to the risk factor. In the case of main effects of the selection factor, correlational designs may lead to conclusions that the risk factor directly leads to increases in thin-ideal internalization, when there is actually no causal relationship between these variables.

Fortunately, research methods such as the co-twin control design have been developed that can identify whether selection effects are present (McGue, Osler, & Christensen, 2010). Co-twin control methods are drawn from the counterfactual model, which eliminates environmental selection effects by matching individuals from exposure and control groups on several key demographic and other characteristics that could theoretically drive selection into the exposure versus control groups (Rubin, 2007, 2008). The reasoning behind the counterfactual model is that the best way to determine whether an environmental exposure factor is truly a risk factor would be to examine the same person's outcome both when exposed and when not exposed to the risk factor (Burt et al., 2010; McGue et al., 2010; Rubin, 2007, 2008). Of course, it is impossible to simultaneously examine these outcomes in one person, but the counterfactual method comes close by matching control and exposed participants on as many key variables as possible. Essentially, the counterfactual model uses matched individuals to estimate the missing, non-exposure outcomes for those who were exposed, and, conversely, the missing exposure outcomes for those who were not exposed (Burt et al., 2010).

In a co-twin control study, outcomes are compared in reared-together co-twins discordant for level of exposure to an environmental factor, in this case, degree of peer group focus on weight/dieting. This design eliminates the need to establish matched samples to control for selection effects, as the members of reared-together twin pairs are already matched on key shared environmental experiences (i.e., environmental influences that are common to co-twins such as age, socioeconomic status, and key sociocultural influences such as thin-focused media, parental focus on weight, etc.) that the traditional counterfactual model goes to great lengths to control for. Further, the co-twin control design improves upon the counterfactual model because, due to their genetic relatedness, twin pairs are entirely (in the case of identical twins) or partially (in the

case of fraternal twins) matched on genetic predispositions. Thus, in the co-twin control design, shared environmental and genetic selection effects are controlled for, since twin-pair discordance in an exposure variable such as body-conscious peer groups cannot be explained by differences in genetic or shared environmental predispositions.

In order to determine whether selection effects are present, three sets of regression results are compared within the co-twin control design; 1) individual level effects (i.e., each individual twin's level of exposure to the risk factor predicting her own level of thin-ideal internalization, without consideration of the co-twin), 2) effects in fraternal (i.e., DZ) twins, which is estimated by examining if twin pair discordance on level of exposure to the risk factor predicts each individual twin's level of thin-ideal internalization and 3) effects in identical (i.e., MZ) twins (calculated in the same way as for DZ twins). The individual level effect most closely approximates correlational research results, as it does not control for any selection effects (genetic or environmental) and thus, does not explain whether an association is due to selection or socialization.

The inferences drawn from the second set of results, the comparison of outcomes in discordant DZ twin pairs, are driven by the fact that reared-together DZ twins share 50% of their genes and 100% of their shared environment. As a result, in discordant DZ twins, associations between twin pair discordance in body-conscious peer groups and thin-ideal internalization may be due to genetic selection effects (since DZ twins have genetic differences), but not shared environmental selection effects. Thus, significant associations between twin pair discordance in body-conscious peer groups and thin-ideal internalization can emerge in DZ twins when no selection is present (see Figure 6, Scenario A), or when genetic selection is present (see Figure 6, Scenario B). However, since shared environmental selection is entirely controlled for in DZ

twins, an association between discordance in body-conscious peer groups and thin-ideal internalization would not emerge when shared environmental selection is present (see Figure 6, Scenario C).

The third, and most influential, set of results are those from discordant MZ twins. MZ twins that are discordant on exposure to a risk factor come extremely close to meeting the goal of the counterfactual model – to examine the same person's outcome both when exposed and when not exposed to the risk factor. This is because MZ twins share all of their genes and shared environmental experiences, so the exposure effect in discordant MZ twins controls entirely for genetic and shared environmental selection effects. Since all possible selection effects are controlled, in MZ twins discordant for levels of exposure to a "risk" factor, a significant association between peer groups and thin-ideal internalization cannot be due to selection effects, and instead are explained by socialization effects (see Figure 6, Scenario A). Alternatively, when the association between level of exposure to risky peer groups and thin ideal-internalization is not significant in discordant MZ twins, either genetic and/or shared environmental selection effects are suggested (see Figure 6, Scenarios A and B), since levels of thin-ideal internalization are similar despite differential exposure to body-conscious peer groups. More specifically, scenario B suggests genetic selection effects, since the association is not significant *only* in MZ twins, where genetic selection effects are controlled for entirely, but remains significant in DZ twins, where genetic selection effects are only partially controlled for. Scenario C suggests genetic *and* shared environmental selection effects, since an association is present only at the individual level, and is not present when genetic and environmental selection is partially or entirely controlled for (i.e., in MZ and DZ twins).

Taken together, by comparing individual level effects (which do not control for selection effects), effects in DZ twins (which control for shared environmental selection and partially control for genetic selection), and effects in MZ twins (which control for shared environmental and genetic selection), the co-twin control design allows for a powerful test of socialization versus selection effects for thin-ideal internalization and its key risk factors.

Given the advantages of the co-twin control design, the present study aimed to investigate whether socialization or selection effects better explain the association between self-reported exposure to body-conscious peer groups and thin-ideal internalization in a sample of pre-adolescent and adolescent female twins. It is hoped that results will more clearly delineate whether exposure to body-conscious peer groups operates as a purely environmentally mediated risk variable (i.e., socialization) or instead, whether genetic and/or environmental selection explains the association between body-conscious peer groups and thin-ideal internalization. The identification of selection versus socialization processes will extend knowledge on specific mechanisms by which peer groups are linked to thin-ideal internalization.

METHODS

Participants

Participants for the present study included 392 same-sex female twins (208 monozygotic; 184 dizygotic) between the ages of 8 and 15 (M=11.16, SD = 1.89) from the ongoing ***Twin Study of Hormones and Disordered Eating Across Puberty*** within the Michigan State University Twin Registry (MSUTR). The MSUTR is population-based and recruits twins through birth records in collaboration with the Michigan Department of Community Health (further details are available elsewhere; Burt & Klump, 2012; Klump & Burt, 2006). The primary aims of the study from which these data are drawn involves the investigation of ovarian

hormone influences on disordered eating. Thus, several exclusion criteria were applied to ensure accurate hormone sampling (i.e., no psychotropic, steroid, or other medication use that is known to influence hormone functioning). Prior research has shown that the use these inclusion/exclusion criteria do not inadvertently affect the range or variability in thin-ideal internalization or disordered eating measures (Klump et al., 2013; Suisman et al., 2012). 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; Burt & Klump, 2012; Culbert et al., 2009; Klump et al., 2013), a pattern that was consistent with the current sample (83% Caucasian, 10% African American, 8% Multiracial, 0.5% Asian). Participant parental income also represented a range of socioeconmic backgrounds, although there were a higher proportion of participants in upper income brackets. Specifically, 6% of participants reporting family income less than $20,000 anually; 12% reported $20,000-$40,000; 18% reported $40,000-$60,000; 27% reported $60,000-$100,000; and 37% reported family incomes greater than $100,000 anually.

Measures

Zygosity Determination

Twin zygosity was determined using physical similarity questionnaires that have been shown to be over 95% accurate compared to genotyping (Lykken et al., 1990; Peeters et al., 1998). During study assessment, two research assistants independently completed the physical similarity questionnaire for the twin pair. Additionally, the twins' parent (usually the mother) completed the questionnaire on his/her twins. When results across raters (i.e., parent and research assistants) were discrepant (21% of sample), questionnaire responses and pictures of the twins

were examined by the study principal investigator (KLK) and graduate students to determine final zygosity status.

Body-Conscious Peer Groups

In order to assess multiple peer group characteristics related to body-consciousness and weight focus, four different self-report measures were used, which assessed different aspects of weight-focused peer groups. Specifically, these measures assessed degree of peer group preoccupation with weight, dieting, and appearance, as well as how much the participant perceived her friends to influence her perspectives on weight, dieting, and appearance. Although significantly correlated, the peer group questionnaires appear to identify distinct characteristics of friend groups. Indeed, correlations among the questionnaires were moderate, ranging from 0.31 to 0.56 (see Table 5), and the squared correlation coefficients (r^2) indicate that only 10-31% of the variance is shared amongst each of the questionnaires. Importantly, however, the peer questionnaires do generally appear to be more correlated with one another than they are with thin-ideal internalization (see Table 5), supporting the idea that each of these questionnaires may be part of an overarching "peer group" construct. Additionally, with the exception of the peer preoccupation with weight and dieting scale, each of the peer questionnaires is a significant predictor of thin-ideal internalization, even when the other questionnaires are accounted for (data not shown), suggesting that examining the questionnaires separately may be useful in identifying differential etiological associations with thin-ideal internalization.

Peer Preoccupation with Weight/Dieting: The Perceived Friend Preoccupation with Weight and Dieting Scale (PFP; Schutz, Paxton, & Wertheim, 2002) is a 9-item self-report scale that was used to assess the degree to which a participant's friends think and talk about weight and dieting (e.g., "My friends encourage each other to lose weight", "My friends worry about

what they eat", "Weight and shape are important to my friends"). On this questionnaire, participants rate the extent to which each item is true for their friend groups on a 5-point likert scale ranging from 1 (*never/definitely not*) to 5 (*always/a lot*). Prior research has demonstrated that all items load on a single factor and have demonstrated excellent internal consistency (alpha = .87) in samples of adolescent girls (Schutz et al., 2002).

Appearance Conversations with Friends: The Appearance Conversations with Friends Scale (Jones et al., 2004) was used to assess the frequency in which participants engage in conversations with peers regarding physical appearance (e.g., "My friends and I talk about what we can do to look our best"; "My friends and I talk about how our bodies look in clothes"). Although similar in some regards to the Peer Preoccupation with Weight/Dieting questionnaire described above, that scale focuses specifically on conversations related to weight/shape/dieting, while the Appearance Conversations with Friends scale focuses on appearance more generally. The Appearance Conversations with Friends scale is a 5-item questionnaire and is rated on a 5-point likert scale ranging from 1 (*never*) to 5 (*very often*). Prior investigations using this scale (or slightly modified versions of the scale) have demonstrated acceptable internal consistency with alphas between .78-.88 (Clark & Tiggemann, 2006; Thompson et al., 2007).

Friends as a Source of Influence: The Friends as a Source of Influence Scale (Paxton, Schutz, Wertheim, & Muir, 1999) was used to assess how important participants think their friends' opinions are in influencing her ideas regarding diets, having a "perfect" body, and weight loss techniques (e.g., exercise, diet products). This scale consists of 5 items that are rated on a 5-point likert scale from 1 (*not at all important*) to 5 (*very important*). Prior studies have reported excellent internal consistency for this scale, with alphas ranging from .86-.87 (Paxton et al., 1999; Thompson et al., 2007)

Appearance Peer Attribution Scale: The Appearance Peer Attribution Scale (Thompson et al., 2007) was created using four appearance related items included in the full Peer Attribution Scale (Lieberman, Gauvin, Bukowski, & White, 2001). The Appearance Peer Attribution Scale assess the degree to which participants believe that her friends/peers would like her better and would result in increased popularity if she lost weight and/or was better looking (e.g., "My friends would like me more if I lost weight"). Each item is rated on a 6-point likert scale ranging from 1 (*false*) to 6 (*true*). Internal consistency for this scale has been excellent in prior studies with alphas of 0.85 (Thompson et al., 2007).

Thin-Ideal Internalization

Internalization of the thin-ideal was assessed using the Sociocultural Attitudes toward Appearance Questionnaire-3 (SATAQ-3; Thompson et al., 2004) This 30-item questionnaire includes four subscales (i.e., general internalization, athlete internalization, pressures, and information). Importantly, in prior studies, the SATAQ-3 has been administered with all 30 items worded positively, or with eight of the thirty items worded negatively. As suggested by Thompson et al. (2004), the version that included the eight reverse-scored items was utilized in the current study.

The 9-item general internalization subscale was examined for the current study, which is commonly used to assess thin-ideal internalization in risk factor and intervention studies (Cafri et al., 2005; Coughlin & Kalodner, 2006; Yamamiya et al., 2005). This subscale assesses the extent to which participants want to look like individuals from various media sources (e.g., television, magazines, movies), which is rated on a 5-point Likert scale from 1 (*definitely disagree*) to 5 (*definitely agree*). It has demonstrated excellent reliability and validity as it differentiates individuals with eating disorders from controls and demonstrates excellent internal consistency in

prior samples (*a*'s >.90) (Calogero et al., 2004; Thompson et al., 2004), as well as in the current sample (see Table 6).

Covariates

Age and Pubertal Development: Recent work has suggested that genetic and environmental influences on thin-ideal internalization do not vary across age or adolescent/pubertal development (Suisman et al., submitted). However, mean levels of thin-ideal internalization do increase across adolescence and pubertal development (Hermes & Keel, 2003; Suisman et al., submitted). In order to ensure that phenotypic associations between age, pubertal development, and thin-ideal internalization do not unduly influence results of the current study, these variables were controlled for in all analyses. Participant age on the date of study participation was computed based on parent-reported date of birth. To asses each participant's degree of pubertal development, the Pubertal Development Scale (PDS; Petersen et al., 1988), 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 and thin-ideal internalization (e.g., Klump, Burt, et al., 2007; Suisman et al., submitted). 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 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 (Culbert et al., 2009; Petersen et al., 1988), and it was also excellent in the current sample (See Table 5). 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.

Body Mass Index (BMI): Given significant associations between BMI and thin-ideal internalization, BMI was used as a covariate in all analyses to account for possible differences in weight-focused peer group/thin-ideal internalization associations that may be explained by weight status. BMI was calculated ([weight]) / [height]^2) from laboratory assessments of height and weight made using a wall-mounted ruler and digital scale, respectively.

Statistical Analyses

Data Transformation

Prior to analyses, scores on each of the peer group measures were log transformed to account for positive skew and kurtosis (i.e., skew and/or kurtosis >1). Thin-ideal internalization scores did not violate normality assumptions (skewness = .60, kurtosis = -.14), and thus log transformations were not needed.

Phenotypic correlations between body-conscious peer groups and thin-ideal internalization

Pearson correlations were first used to replicate prior research showing significant within-person associations between weight-focused peer groups and thin-ideal internalization (Hermes & Keel, 2003; Suisman et al., submitted). Next, within-twin pair difference scores on measures of peer groups and thin-ideal internalization were calculated by subtracting twin 2's score from twin 1's score on each of the questionnaires. In order to retain information on which twin scored higher on each measure, the sign of the difference score was preserved. Thus, positive difference scores indicate that twin 1 scored higher on the measure, and negative difference scores indicate that twin 2 scored higher on the measure.

Pearson correlations were then used to examine if differences within twin pairs in peer group affiliation is associated with within-pair differences in thin-ideal internalization. These within-twin pair difference score correlations were used to begin to examine if differential exposure to body conscious peer groups is associated with differences in thin-ideal internalization (i.e., socialization effects), or whether thin-ideal internalization is similar in co-twins despite differential exposure to peer groups (i.e., selection effects). As a reminder, since MZ twins share 100% of their genes and shared environment, significant positive correlations between these difference scores was indicative of socialization effects. This is because a significant positive correlation between difference scores indicated that the twin with higher levels of "risky" peer group exposure also had higher levels of thin-ideal internalization, even after controlling for genetic or shared environmental selection. Selection effects, on the other hand, were inferred if correlations in MZ twins were non-significant and close to zero, since this indicated that co-twin differences in peer groups are not associated with differences in thin-ideal internalization. DZ twin correlations were particularly useful if selection effects were implied based on associations in MZ twins (i.e., if there is no significant correlation between difference scores). Specifically, genetic selection effects were implied if DZ twin but not MZ twin correlations were significant, because, genetic selection effects are controlled for entirely in MZ twins, but only partially in DZ twins. The possibility of both genetic and shared environmental selection effects were inferred if MZ and DZ twin difference correlations were both non-significant.

In order to quantify differences in MZ versus DZ correlations, the Z-test of independence was used to examine whether MZ and DZ twin correlations were significantly different from one another.

Co-Twin Control Analyses

Co-twin control analyses were then used to definitively confirm or disconfirm selection versus socialization effects. The co-twin control analyses used regression-based models within a multilevel model (MLM) framework (Burt et al., 2010; McGue et al., 2010). MLM accounts for the non-independence of the twin data within a pair by nesting each individual within the twin pair. For each MLM, the difference in each twin's level of exposure from the mean level of exposure within her family was used to predict the level of thin-ideal internalization in each twin. Specifically, let y_{ij} be the observed outcome for the jth twin (j=1,2) in the ith twin pair (i=1,2,…,N) and let x_{ij} be the corresponding exposure index (i.e., peer group exposure). The overall regression of thin-ideal internalization on level of peer group exposure is given by the model

$$y_{ij} = \beta_0 + \beta_1 x_{ij} + \varepsilon_{ij,}$$

where β_1 is the individual-level effect of peer group exposure on thin-ideal internalization, β_0 is the intercept term, and ε_{ij} is the residual (which is correlated across the two members of a twin pair). The overall regression effect can be further broken down into a within-pair (β_w) effect and a between pair (β_b) effect. The β_w effect is the core of the co-twin control design and represents how well discordance on the peer groups within a twin pair predicts thin-ideal internalization in each twin. This effect was calculated separately in MZ and DZ twins. The between-pair (β_b) effect approximates the individual-level effect (i.e., the association between the exposure factor and thin-ideal internalization without taking into account twin pair discordance (i.e., approximates the correlation between peer group exposure and thin-ideal internalization). The within-pair and between-pair effects can be represented using the regression model

$$y_{ij} = \beta_0 + \beta_w(x_{ij}-\bar{x}_i) + \beta_b\bar{x}_i + \varepsilon_{ij,}$$

where $\bar{x}_i$ is the mean exposure index (peer group expousure) for the ith pair. Notably, by subtracting the *mean* exposure index within the twin pair from the level of exposure in the twin with higher exposure, the within-pair effect estimates how much more exposure the more highly exposed twin experienced as compared to what the exposure would be expected to be simply from being a member of that family. In order to determine whether exposure and/or selection explain the association between body conscious peer groups and thin-ideal internalization, the magnitude and significance of the individual level effect (i.e., β_b) and within-pair effects (β_w; in MZ and DZ twins separately) was compared. As outlined above and in Figure 6, exposure effects were suggested if all three effects are similar in magnitude and significance, while selection effects were suggested if β_w in MZ twins is small and non-significant. If selection effects were suggested (i.e., β_w in MZ twins is small and non-significant), effects in DZ twins would help to differentiate genetic selection effects (Figure 6, scenario B) versus genetic and/or shared environmental effects (Figure 6, Scenario C).

All regression analyses were conducted in SPSS software and were repeated for each of the peer questionnaires. In all analyses, age, pubertal development, and BMI were included as covariates. As previously recommended (Burt et al., 2010), the outcome variable (thin-ideal internalization) was standardized to have a mean of 0 and a standard deviation of 1 for all multilevel modeling analyses to aid in interpretation of the unstandardized fixed-effect coefficients that result from multilevel models.

RESULTS

Descriptive Statistics. Table 6 includes descriptive statistics for all exposure (i.e., peer group measures) and outcome (i.e., thin-ideal internalization) variables, as well as covariates (BMI, age). Means, standard deviations, and ranges of scores are presented for both raw scores

and difference scores. Additionally, information is presented for the full sample, as well as MZ and DZ twins separately.

Mean difference scores and standard deviations do suggest that twin pairs tended to score somewhat similarly on each of the measures, but there does seem to be adequate variability within twin pairs (see Table 6). For each of the questionnaires, the majority of twins did score differently from her co-twin (i.e., difference score >0). Indeed, across questionnaires, 54-92% of twins had a difference score greater than zero (M=77%, SD=15%), and 24-65% percent had a difference score greater than 1 (M=49%, SD=17%).

Given that the core of the co-twin control design requires comparison of associations between variables in MZ versus DZ twins, mean or variance differences across zygosity on key measures were investigated. As shown in Table 6, there were minimal differences between MZ and DZ twins on exposure or outcome measures, and sample sizes were similar for MZ and DZ twins. Of particular importance, there are no significant differences between groups on any of the difference score variables, which are the core of the co-twin control design. Given the overall pattern of similarity across zygosity, any differences in associations between body-conscious peer groups and thin-ideal internalization in MZ versus DZ twins are unlikely to be explained by mean or variance differences between groups.

Phenotypic Correlations

Pearson correlations were next used to examine *within individual associations* between peer groups and thin-ideal internalization (see Table 7). As expected based on prior work (e.g., Keery et al., 2004), within individual correlations indicated that individual scores on peer group measures were significantly associated with thin-ideal internalization. Significant associations were present in the full sample, as well as in MZ and DZ twins separately. These data support

prior findings suggesting within-individual associations between peer groups and thin-ideal internalization.

Next, within-pair difference scores were used to examine if twin differences in exposure variables (i.e., peer groups) are associated with twin differences in outcomes (i.e., thin-ideal internalization). As shown in Table 7, these results strongly suggested socialization, rather than selection mechanisms. In MZ twins, co-twin differences in peer group exposure were positively correlated with twin differences in thin-ideal internalization, suggesting that the twin with higher levels of peer group exposure was also more likely to have higher levels of thin-ideal internalization. Interestingly, given the results in MZ twins, significant associations were also expected in DZ twins (i.e., evidence for socialization), since the only difference between these groups is that correlations in DZ twins do not control entirely for selection effects. Contrary to expectations, most of the DZ twin associations were not statistically significant. However, as indicated by the Z-test (see Table 7), in all cases, the DZ twin correlations were not significantly smaller than the MZ twin correlations, suggesting that although they did not reach statistical significance, they were still similar to the findings in MZ twins, which were uniformly statistically significant. Thus, despite the somewhat inconsistent findings for DZ twins, the twin difference correlation results, particularly in MZ twins, suggest socialization, rather than selection, mechanisms.

Co-Twin Control Analyses

Results of the co-twin control analyses were similar to findings from the Pearson correlations (see Table 8). As shown in the table, all of the between pair effects estimates were statistically significant, as expected, since the between pair effects indicate associations between each individual twins' exposure to peer groups and her own thin-ideal internalization. Within-

pair effects also tended to be statistically significant, particularly in MZ twins, suggesting that twins with higher levels of weight focused peer groups also had higher levels of thin-ideal internalization. As was true for the correlations between co-twin difference scores, there was less consistency in results for DZ twins. Specifically, within-pair effects for DZ twins were only significant for one peer measure (the Peer Appearance Attribution Scale). However, there were generally not statistically significant differences in results for MZ versus DZ twins, suggesting that even in cases where statistical significance was not reached for DZ twins, the effect in DZ twins was not significantly smaller than the effect in MZ twins. Taken together, overall, results most closely represent scenario C from Figure 6, since significant associations were consistently found between and within pairs for MZ twins, and results in DZ twins, although not significant, did not generally differ significantly from results in MZ twins.

DISCUSSION

Using a powerful co-twin control design, this study demonstrated that the association between weight-focused peer groups and thin-ideal internalization appears to be driven by effects of socialization, rather than selection. These results suggest that peer groups are an environmentally mediated contributor to thin-ideal internalization during late childhood and early adolescence. Importantly, analyses controlled for BMI, age, and pubertal development, suggesting that effects of these variables do not unduly influence findings. Taken together, findings strongly support prior theories that proposed that associations between body-conscious peer groups and thin-ideal internalization are due to effects of socialization (Shroff & Thompson, 2006b)

Interestingly, a recent co-twin control study demonstrated that selection, rather than socialization effects, contributed to the association between body-conscious peer groups and

disordered eating symptoms) (i.e., scenario C in Figure 6 was present; O'Connor, Burt, & Klump, in preparation). The O'Connor et al (in preparation) study was conducted with nearly the same sample as the current study (92% overlap in participants), and the peer measures used were identical to those in the current study. Indeed, the only substantial difference between the O'Connor et al (in preparation) study and the current investigation is the outcome measure; the O'Connor et al (in preparation) paper studied *disordered eating symptoms*, whereas the current paper examined thin-ideal internalization.

The fact that socialization effects were identified for body-conscious peer group/thin-ideal internalization associations while selection effects were identified for body-conscious peer group/disordered eating associations may initially seem incompatible. However, similar patterns have been observed for other phenotypes, whereby different mechanisms (i.e., selection versus socialization) explain associations between the same predictor but different outcome variables. For example, research has shown that the association between gang membership and selling drugs appears to be due primarily to socialization effects, as the tendency to sell drugs increases only after joining a gang (i.e., the tendency is not present before joining a gang; Gordon et al., 2004). By contrast, the association between gang membership and engaging in violent behavior appears to be due to selection effects, as boys with elevated levels of aggression and violence are more likely to join a gang than are those who are less aggressive/violent (Gordon et al., 2004). Overall, these findings regarding gang membership highlight how selection and socialization effects can be complimentary processes that may both contribute to the development of complex, but related, phenotypes.

This type of a complimentary process may be present for thin-ideal internalization as well. As shown in Figure 7, it may be that genetic and/or environmental predispositions for

disordered eating initially cause girls to *select* into body-conscious peer groups (as suggested by O'Connor et al., in preparation). Once involved in these body-conscious peer groups, socialization effects may then then drive increases in thin-ideal internalization (as suggested by the present study) that would then lead to further increases in disordered eating symptoms (Thompson & Stice, 2001). The increases in disordered eating would likely lead to even further selection into similarly minded, weight-focused peer groups (as suggested by O'Connor et al. [in preparation]) which may then further increase the development of thin-ideal internalization (via socialization effects). These processes may continue in a cyclical fashion, leading to increasingly disordered behaviors, particularly in girls who are initially predisposed to eating disordered behavior.

Overall, this re-conceptualization could enrich the existing hypotheses made by the tripartite model by proposing the specific processes through which weight-focused peer groups, thin-ideal internalization, and disordered eating might develop in relation to each other. However, the model is speculative, as no longitudinal data exist to test its directional (and causal) hypotheses. A longitudinal, co-twin control study could examine whether differences in disordered eating at baseline predict later affiliation with weight focused peer groups, providing additional evidence in favor of the selection processes suggested by O'Connor et al. (in preparation). Such a study could also investigate socialization processes by examining longitudinal effects of affiliation with weight-focused peer groups, above and beyond the initial effects of selection. Finally, a longitudinal co-twin control study could examine whether the increased thin-ideal internalization within peer groups predicts further selection into weight-focused friendships. In essence, by following participants over a long period of time, a longitudinal co-twin control study could replicate our cross-sectional results and those of

O'Connor et al. (in preparation) and also make stronger inferences regarding the direction of effects and the main etiologic pathways in Figure 7.

Nonetheless, even without confirmation via longitudinal designs, the significant socialization effects observed for thin-ideal internalization in this study have implications for prevention and intervention work. The most effective and well-studied eating disorder prevention programs, cognitive dissonance programs (Stice, Becker, & Yokum, 2013) have been shown to reduce thin-ideal internalization and disordered eating in non-clinical samples of adolescent girls (Becker, Bull, Schaumberg, Cauble, & Franco, 2008; Becker, Smith, & Ciao, 2006; Coughlin & Kalodner, 2006; Stice et al., 2001; Stice et al., 2000; Yamamiya et al., 2005), and initial evidence also suggests that enhanced cognitive dissonance interventions are effective in treatment with girls with diagnosed eating disorders (Stice, Butryn, Rohde, & Shaw, 2013). However, these prevention programs are focused almost entirely on changing *interpretations* of beauty ideals by providing psychoeducation on the realities of images presented in the media (e.g., digital editing of body sizes) and asking participants to engage in writing and other activities that actively argue against the thin-ideal. Results of the current study suggest that focusing on reducing body-conscious conversations within peer groups might enhance the effectiveness of cognitive dissonance prevention programs. For example, a prevention program that provided psychoeducation about the effects of focus on weight and dieting among peer groups, and then trained the members of the prevention program to be "peer representatives" that are able attempt to change the focus of conversation among her friends, could be developed to supplement existing cognitive dissonance programs. Indeed, peer-facilitated cognitive-dissonance prevention programs have been developed and used successfully in college sororities (Becker et al., 2006), so it is possible that similar efforts within high-school or middle-school aged peer groups may be

possible. It is intriguing to think that the effects of prevention efforts, with the proper training of program participants, could spread within the peer groups of program participants, even if those peers did not participate in the prevention program.

Despite the strengths of the current study, there are several limitations that need to be considered when interpreting its results. The current data are cross-sectional, limiting the ability to confirm causal associations between peer groups and thin-ideal internalization. Although the co-twin control design is powerful for ruling out the presence of selection effects, it does not allow for examination of the direction of the association between the risk factor (body-conscious peer groups) and outcome (thin-ideal internalization) without the use of longitudinal designs. Given the robust theory suggesting that exposure to risky peer groups precedes the development of thin-ideal internalization(Shroff & Thompson, 2006b) the association was conceptualized in this way throughout the paper. However, the possibility of a reverse effect cannot be ruled out. It is possible that a high level of thin-ideal internalization in an individual causes her peer group, or her perceptions of her peer group, to become more weight/diet focused. Regardless of the direction of effects, the current study does allow for the conclusion that pre-existing selection factors do not account for *associations* between peer groups and thin-ideal internalization. However, as described above, longitudinal data within a co-twin control framework would be particularly powerful to examine causal associations, while controlling for selection effects. However, given the absence of such data at this time, this study is a critical first step in understanding selection versus socialization effects for peer groups and thin-ideal internalization.

Additionally, analyses in the current study were based entirely on self-report data. As a result, objective data on the degree to which the peer groups were actually focused on weight and shape were not available. Thus, it is unclear whether twins pairs who are discordant on peer

group measures were truly discordant in terms of the amount their peer groups were body-conscious, or if they were instead discordant only in the *perceptions* of their friend group. Adding to this potential problem is that the co-twin with higher thin-ideal internalization may be more likely to report higher focus on weight and dieting in her peer group simply because she is more sensitive to or perceptive of these topics. If this is the case, there may be a bias toward finding socialization rather than selection effects in the current study, as the association between thin-ideal internalization and friend groups would be higher in the co-twin with elevated thin-ideal internalization as a result of her increased sensitivity to these topics, rather than actual differences in peer groups. However, as noted above, a study using the same sample and the same peer group measures as the current study did identify selection, rather than socialization effects, for disordered eating (O'Connor et al, in preparation). These results provide some reassurance that the use of self-report data did not seem to prevent the identification of selection effects. However, future studies with more objective data on similarities and differences in friend groups (e.g., by collecting data from the peers directly), or observational data on topics of conversation within friend groups, would be useful for confirming the current findings.

In the current study, MZ and DZ twins tended to report equally similar characteristics of their peer groups. Specifically, correlations within twin pairs were about the same for MZ and DZ twins for the peer preoccupation with weight and dieting scale (MZ r = .48, DZ r=.47); appearance conversations with friends (MZ r = .50, DZ r = .49); and the peer attribution scale (MZ r = .48, DZ r =.40). The only exception was the friends as a source of influence scale, where MZ twins did report more similar peer groups than DZ twin (MZ r = .50, DZ r = .19). The overall pattern of similar peer characteristics in both MZ and DZ twins contrasts with prior literature, which demonstrates that MZ twins tend to share more friends than DZ twins (Cronk et

al., 2002). If twins in our sample also followed this pattern, than it seems that MZ twins should report more similar peer characteristics than DZ twins, which was not generally the case. Unfortunately, we did not explicitly assess the percentage of friends that twins objectively share. It is possible that our sample still fits this general pattern of MZ twins sharing more friends than DZ twins, but that twins still reported relatively similar levels of weight focused peer groups. Future work should further validate the peer questionnaires used in the current study by also examining how many friends the twins objectively share, and examining whether this is related to the degree to which twins report similar peer groups, as it would be expected that co-twins who share more friends would report more similar peer characteristics.

Third, the co-twin control design does not account for nonshared environmental influences, or those unique environmental experiences between co-twins (e.g., experiencing differential levels of media exposure) that make them different from one another (McGue et al., 2010). Thus, all types of selection effects were not controlled for, since nonshared environmental variables that could cause twins to differentially select into body-conscious peer groups (e.g., one twin being involved in a weight-focused sport) were not accounted for. As a result, although the significant associations between differential peer group exposure and outcomes observed in the present study strongly suggest socialization versus selection effects, selection into weight-focused peer groups based on other nonshared experiences between twins cannot be ruled out. Future co-twin control studies that explicitly measure and account for several relevant, nonshared experiences between twin pairs (e.g., differential exposure to thin-ideal media) would strengthen conclusions that can be drawn from the current study.

Finally, although the co-twin control design was a powerful way to begin to understand selection versus socialization effects in the association between body-conscious peer groups and

thin-ideal internalization, other twin designs may also provide useful contributions to understanding these effects. Indeed, although the co-twin control design provides information on the extent to which co-twin differences in exposure to a risk factor (body-conscious peer groups) contribute to differences in outcome (thin-ideal internalization), leading to the inference of selection versus socialization effects, it does not provide specific estimates of the percent of variance in body-conscious peer groups and thin-ideal internalization that is accounted for by genetic, shared environmental, and/or nonshared environmental effects. Indeed, a bivariate twin study would be especially useful, as such a design would indicate the extent to which genetic or environmental effects that contribute to having body-conscious peer groups overlap with genetic or environmental effects on thin-ideal internalization. In a bivariate twin design, large genetic correlations would support the possibility of selection effects, as it would suggest that the genetic influences on thin-ideal internalization also contribute to selecting weight-focused peers. Small and nonsignificant genetic correlations, on the other hand, would be more consistent with socialization processes. Unfortunately, our sample size (N = 392) did not allow us to conduct bivariate models at this time, as recommended sample sizes for these designs generally fall in the range of 500-1,000 individuals (Posthuma & Boomsma, 2000). Future studies that employ both co-twin control and bivariate twin designs would provide particularly powerful and complimentary evidence for selection versus socialization processes.

Despite these limitations, the current study provides strong evidence that the previously observed association between peer groups and thin-ideal internalization are not accounted for by unmeasured genetic or shared environmental selection effects. The socialization effects suggested in the current study strongly support existing etiological models of thin-ideal internalization and disordered eating, which hypothesize that exposure to weight-focused peer

groups leads to thin-ideal internalization via environmental effects. If longitudinal replications of confirm these findings, it may be useful for prevention programs to add modules that focus not only on direct media influences, but also on the language and conversation topics that occur within peer groups.

CHAPTER 4: Summary and Conclusions

The current set of studies expanded knowledge on the developmental course of etiological influences on thin-ideal internalization, as well as mechanisms through which body-conscious peer groups may contribute to increases in thin-ideal internalization. Study 1 demonstrated that the relative proportion of genetic and environmental influences on thin-ideal internalization do not appear to differ across pre-adolescent and adolescent development, particularly after controlling for disordered eating. Specifically, nonshared environmental, shared environmental, and genetic influences were generally estimated to be equal across development. These findings also highlighted the particularly important role of environmental influences in the development of thin-ideal internalization, and suggested that risk factors present in late-childhood may persist throughout adolescent development. Study 2 examined the mechanisms through which one risk factor, body-conscious peer groups, may lead to the development of thin-ideal internalization. This study was the first to provide empirical support that socialization effects account for the association between body-conscious peer groups and thin-ideal internalization, ruling out the role of selection effects.

Integration of the findings across these studies could further contribute to existing knowledge on the etiology of thin-ideal internalization. Prior to these two studies, models of the development of eating disorders, such as the tripartite influence model of body dissatisfaction and disordered eating, suggested that sociocultural risk factors (e.g., body-conscious peer groups, media exposure) lead to thin-ideal internalization, which then contributes to the development of disordered eating (Shroff & Thompson, 2006b). As highlighted in study 2, however, a re-conceptualization of this model may be needed (see Figure 7), particularly when also considering the results of a recent study that identified selection effects in the association between body-

conscious peer groups and *disordered eating* (O'Connor et al., in preparation). The re-conceptualized model suggests that selection factors may initially cause genetically or environmentally predisposed girls to select into body-conscious peer groups, which then leads to increases in thin-ideal internalization via socialization influences, leading to the further development of disordered eating. The results of Study 1 may enhance this proposed model further, since Study 1 suggests few changes in etiological effects across development. Thus, it is likely that the re-conceptualized etiological model would apply to girls throughout development (i.e., late childhood through young adulthood).

Additionally, Study 1 suggested relatively large environmental influences on thin-ideal internalization, and Study 2 supported the role of socialization in the association between weight-focused peer groups and thin-ideal internalization. Taken together, it is possible that body-conscious peer groups are one environmental influence that contributes to the robust environmental estimates in Study 1. The results of Study 1 also suggested small genetic influences on thin-ideal internalization. The current set of findings do not allow for the identification of what specific mechanisms contribute to this genetic variance, although one possibility is that heritable personality characteristics that contribute to thin-ideal internalization (e.g., perfectionism) play a role (see Suisman et al., 2012 for more detail on the possible role of personality in the heritability of thin-ideal internalization).

As highlighted throughout this series of studies, thin-ideal internalization is generally considered to be a risk factor for disordered eating (Thompson & Stice, 2001). When establishing a phenotype as a risk factor, it is important to provide evidence that the proposed risk factor is not simply a correlate, consequence, or symptom of the outcome (in this case, disordered eating or eating disorders; (Thompson & Stice, 2001). The current findings further

contribute to the conceptualization of thin-ideal internalization as a possible risk factor for disordered eating by highlighting the ways in which thin-ideal internalization is a separate phenotype from disordered eating with unique developmental pathways. Indeed, thin-ideal internalization appears to be unique from disordered eating in terms of the degree to which genetic and environmental effects vary across adolescent and pubertal development. Specifically, the etiology of disordered eating is known to change across development, with increasing genetic, and decreasing shared environmental effects, across puberty (Klump, Perkins, et al., 2007). On the other hand, the current study suggested relatively stable genetic and environmental influences on thin-ideal internalization across development, with larger influences of nonshared and shared environmental effects, and smaller genetic effects, than are typically reported for disordered eating. These findings suggest that the etiology of thin-ideal internalization is relatively stable, and largely environmental, while risk for disordered eating is more dynamic and is significantly influenced by genetic factors Further, as highlighted above and in Study 2, socialization effects may play a larger role in the development of thin-ideal internalization than disordered eating, at least in terms of the role of body-conscious peer groups. (O'Connor et al., in preparation). Taken together, these findings highlight that thin-ideal internalization is not an early manifestation of symptoms of disordered eating with similar etiology, but instead appears to be an early risk marker that has a unique developmental and etiological trajectory.

In order to further understand the unique etiology of both thin-ideal internalization and disordered eating, it would be particularly interesting to study the reverse effects as those examined in the current series of studies. For example, it may be interesting to examine etiological effects of disordered eating while accounting for thin-ideal internalization, or to examine thin-ideal internalization as the independent variable in a co-twin control study of thin-

ideal internalization and disordered eating. Indeed, such designs would contribute to understanding of the variance in disordered eating that is unique from thin-ideal internalization.

Relatedly, the current set of studies did not consider the possibility that eating and thin-ideal internalization have differing etiological associations across development. For example, it is feasible that in adulthood, all genetic influences on thin-ideal internalization are accounted for by disordered eating, but in adolescence, there are unique genetic contributions to thin-ideal internalization. Indeed, when examining Study 1 results, the full age models before and after accounting for disordered eating (Figures 3a and 3b, respectively) suggest that this precise pattern might be supported. Before accounting for disordered eating, genetic influences on thin-ideal internalization appear to increase for each subsequent age group (although, as described above, none of these changes are statistically significant). However, after accounting for disordered eating, the genetic effects in the oldest age group plummet, while they remain about the same as they were prior to controlling for disordered eating in the middle age group. This pattern could occur if genetic influences on thin-ideal internalization are entirely accounted for by disordered eating in adulthood but not middle adolescence. Future studies using bivariate moderation models will be important to directly examine this possibility. Indeed, it is possible that etiological associations between thin-ideal internalization and disordered eating vary markedly across development, even if the etiological influences on thin-ideal internalization remain relatively stable.

Given the importance of thin-ideal internalization in the development of disordered eating (Thompson & Stice, 2001), and the unique etiological trajectory of thin-ideal internalization identified in the current series of studies, continued efforts to intervene *specifically* in the development of thin-ideal internalization appear warranted. Indeed, current

results suggest that much of the etiology of thin-ideal internalization may be due to key environmental factors that are prominent across development (e.g., sociocultural risk factors such as body-conscious peer groups), so interventions on these environmental factors may be useful. As hypothesized in Study 2, it is possible that thin-ideal internalization is more likely to develop in vulnerable girls as a result of selection into weight-focused peer groups. This may be an especially useful area for intervention, as it is possible that thin-ideal internalization is a particularly potent risk factor when it influences girls who are already vulnerable to disordered eating. If this hypothesis is correct, then decreases in thin-ideal internalization could contribute to the prevention of the development of eating disorders even in the most vulnerable girls (i.e., those with high genetic risk for disordered eating). Although the findings of the current set of studies can not speak directly to which prevention programs might be most useful, continued use of known programs that have been identified as effective (e.g., cognitive dissonance and media literacy programs) and further dissemination of these programs, is needed (Stice, Becker, et al., 2013; Stice & Shaw, 2004). Further, as suggested in Study 2, future work that specifically examines efforts to change the thin-ideal culture within body-conscious peer groups may be a viable addition to existing programs.

Overall, these findings contribute to a growing literature on the etiology of thin-ideal internalization and disordered eating, and identify several areas in which the etiology of thin-ideal internalization is unique from that of disordered eating. Future longitudinal research is needed to specifically examine the complex interplay among genetic and environmental risk for thin-ideal internalization and disordered eating, as well as the roles of socialization and selection factors in the associations between environmental risk, thin-ideal internalization, and disordered eating.

APPENDIX

Table 1

Descriptive Statistics

Scale	α	*M* (SD)	Range	Pearson *r* with MEBS	Pearson *r* with Puberty [a]	Pearson *r* with Puberty, Only Twins that Completed PDS [a]	Pearson *r* with Age [a]
SATAQ-3 General Internalization	0.88	2.33 (0.93)	1.00-5.00	.45**	.25**(.20**)	.07 (-.02)	.28**(.24**)
MEBS Total Score	0.87	4.82 (4.87)	0.00-25.00	--	.15**	.21**	.15**
Pubertal Development Scale	0.83	3.16 (1.11)	1.00-4.00	--	--	--	.88**
Age	--	15.06 (3.93)	8.5-25.14	--	--	--	--

Note. SATAQ-3 = Sociocultural Attitudes Toward Appearance Questionnaire – 3. MEBS = Minnesota Eating Behavior Survey. PDS = Pubertal Development Scale.

[a]Pearson correlations for SATAQ-3 General Internalization are presented both with and without controlling for disordered eating. The correlations in parentheses are partial correlations that control for the MEBS total score.

Table 2

Twin Intraclass Correlations for the SATAQ General Internalization Scale by Age and Pubertal Status

	Thin-Ideal Internalization Controlling for BMI Only					Thin-Ideal Internalization Controlling for Disordered Eating and BMI				
Moderator	MZ	DZ	MZ N	DZ N	Z	MZ	DZ	MZ N	DZ N	Z
Age										
Ages 8-12	.30**	.42**	148	134	-1.15	.23**	.23**	146	132	0.00
Ages 13-16	.49**	.33**	118	104	1.42	.39**	.20*	114	104	1.52
Ages 17-25	.54**	.40**	154	118	1.46	.32**	.37**	154	116	-0.46
Puberty										
Pre/Early Puberty	.25**	.44**	122	98	-1.58	.27**	.25*	122	98	0.16
Mid/Late Puberty	.55**	.39**	270	224	2.27*	.39**	.31**	266	222	1.00

Note. BMI = Body Mass Index. MZ = Monozygotic. DZ = Dizygotic
Asterisks following twin correlations indicate that the correlation is significantly greater than zero. Asterisks following the Z-statistic indicate whether the MZ and DZ correlations are significantly different from one another (one-tailed). For all puberty twin correlations, only twin pairs who are concordant for pubertal category are included (91% of total sample).
*p<0.05, **p<0.01.

Table 3.

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

Model	-2lnL	df	Δχ2 (df)	*p*	AIC	BIC
Age Models						
Control for BMI						
Full Moderation	2181.38	803	--	--	575.38	-1340.17
Linear Moderation	**2183.89**	**806**	**2.51 (3)**	**.47**	**571.89**	**-1347.99**
No Moderation	2194.60	809	13.22 (6)	.04	576.60	-1351.72
Control for BMI & DE						
Full Moderation	2185.40	795	--	--	595.40	-1311.13
Linear Moderation	2185.92	798	0.52 (3)	--	589.92	-1319.94
No Moderation	**2197.37**	**801**	**11.97 (6)**	**.06**	**595.37**	**-1323.29**
Puberty Models						
Control for BMI						
Linear Moderation	**2148.82**	**787**	**--**	**--**	**574.82**	**-1300.55**
No Moderation	2167.30	790	18.48 (3)	<.001	587.30	-1300.37
Control for BMI & DE						
Linear Moderation	**2160.46**	**780**	**--**	**--**	**600.46**	**-1270.80**
No Moderation	2176.82	783	16.36 (3)	.001	610.82	-1271.66

Note. -2lnL = minus 2 times the log likelihood, Δχ2 = 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 quadraticaly 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 4.

Unstandardized Path and Moderator Estimates for Full and Best-Fitting Twin Models

Moderator	Model	a	c	e	A1	C1	E1	A2	C2	E2
Age Models										
	Full	.00	**-.52**	**.68**	-.63	.01	.14	.19	-.02	-.06
		(-.55, .55)	**(-.65, -.13)**	**(.60, .77)**	(-.90, .90)	(-.82, .90)	(-.13, .43)	(-.75, .75)	(-.56, .80)	(-.21, .08)
Control for	Linear	.19	**.50**	**-.69**	.13	.07	-.03	--	--	--
BMI		(-.56, .56)	**(.15, .65)**	**(-.78, -.61)**	(-.38, .38)	(-.17, .25)	(-.10, .04)			
	No Mod[a]	-.32	**-.58**	**.72**	--	--	--	--	--	--
		(-.64, .64)	**(-.72, -.26)**	**(.66, .79)**						
	Full	-.19	-.39	**.75**	-.59	.21	.03	.31	-.16	.01
		(-.61, .59)	(-.57, .56)	**(.64, .85)**	(-.90, .90)	(-.90, .90)	(-.26, .36)	(-.70, .71)	(-.73, .73)	(-.14, .15)
Control for	Linear	.27	.36	**-.74**	-.01	.12	-.06	--	--	--
BMI & DE		(-.60, .60)	(-.56, .56)	**(-.84, -.64)**	(-.39, .39)	(-.40, .40)	(-.13, .02)			
	No Mod[a]	-.26	-.48	**.80**	--	--	--	--	--	--
		(-.63, .63)	(-.62, .62)	**(.73, .87)**						
Puberty Models										
	Linear[a]	-.18	**.47**	**.67**	**.75**	.05	.05	--	--	--
Control for		(-.53, .32)	**(.14, .62)**	**(.58, .77)**	**(.05, .90)**	(-.90, .51)	(-.07, .18)			
BMI	No Mod	.31	**-.59**	**-.73**	--	--	--	--	--	--
		(-.64, .64)	**(-.73, -.29)**	**(-.79, -.66)**						
	Linear	-.43	.23	**.69**	.87	.25	.12	--	--	--
Control for		(-.64, .64)	(-.56, .54)	**(.59, .81)**	(-.90, .90)	(-.90, .90)	(-.03, .26)			
BMI & DE	No Mod[a]	.23	-.51	**-.80**	--	--	--	--	--	--
		(-.63, .63)	(-.64, .64)	**(-.87, -.73)**						

Table 4 (cont'd)

Note. BMI = Body Mass Index; DE = Disordered Eating,; 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. All age moderation models are univariate twin moderation models (Purcell, 2002). All puberty models are extended univariate moderation models (van der Sluis et al., 2012). A full moderation model was not calculated for pubertal groups since quadratic moderation is not possible when examining only two groups. [a]= best-fitting model.

Table 5.

Correlations among peer questionnaires and thin-ideal internalization

	1	2	3	4	5
1. Peer Preocc. Weight/Dieting	--				
2. Appearance Conversations	.56**	--			
3. Friends as Source of Influence	.54**	.47**	--		
4. Peer Attribution	.41**	.31**	.35*	--	
5. Thin-Ideal Internalization	.32**	.36**	.37**	.41**	--

Note. Peer Preocc. Weight/Dieting = Peer Preoccupation with Weight/Dieting Scale. Appearance Conversations = Appearance Conversations with Friends Scale. Friends as a Source of Influence = Friends as a Source of Influence Scale. Peer Attribution = Peer Attribution (Appearance) Scale. N = 383-415.
*p<0.05, **p<0.01.

Table 6.

Descriptive statistics for peer group measures, thin-ideal internalization, and covariates.

		MZ Twins Only (N = 182-206)			DZ Twins Only (N=176-200)			MZ/DZ Differences	
Variable	Alpha	*M (SD)*	Min	Max	*M (SD)*	Min	Max	t (df)	Variance F
Raw Score									
Peer Preocc. Weight/Dieting	0.98	14.08 (5.66)	9.00	36.00	14.08 (5.46)	9.00	37.00	0.002 (386)	0.16
Appearance Conversations	0.87	8.60 (4.28)	5.00	25.00	8.89 (4.15)	5.00	25.00	-0.69 (413)	0.66
Friends Influence	0.84	7.86 (4.12)	5.00	25.00	7.46 (3.06)	5.00	19.00	1.13 (385.75)	**5.41***

Note. Peer Preocc. Weight/Dieting = Peer Preoccupation with Weight/Dieting Scale. Appearance Conversations = Appearance Conversations with Friends Scale. Friends Influence = Friends as a Source of Influence Scale. Peer Attribution = Peer Attribution (Appearance) Scale. Thin-Ideal Internalization = General Internalization Subscale of the Sociocultural Attitudes Toward Appearance Questionnaire - 3; BMI = Body Mass Index. Possible range = Possible range of scores on the measure. Variance F = F-test from Levines' Test of Equality of Variances. N's for difference score analyses are approximately half the size of the N's for raw score analyses as difference score analyses were conducted for each pair, while raw score analyses were calculated for each individual. Further, raw scores include all individuals that participated in the study, but difference scores were only able to be calculated for families in which data was available for both twins. Absolute values of sibling difference scores were used for all anaylses presented in this table in order to present the range of sibling differences in peer groups and internalization. However, pearson correlations and co-twin control analyses presented in Tables 3 and 4 were conducted with the sign of the difference score retained.

Table 6 (cont'd)

		MZ Twins Only (N = 182-206)			DZ Twins Only (N=176-200)			MZ/DZ Differences	
Variable	Alpha	*M (SD)*	Min	Max	*M (SD)*	Min	Max	t (df)	Variance F
Raw Score									
Peer Attribution	0.80	5.92 (3.48)	4.00	19.00	5.76 (3.12)	4.00	22.00	0.50 (411)	1.63
Thin-Ideal Internalization	0.81	2.10 (0.85)	1.00	4.67	2.06 (0.81)	1.00	4.89	0.40 (399)	0.14
BMI	--	18.54 (3.83)	11.24	34.49	19.66 (5.39)	11.9 7	46.55	**-2.48 (376.94)****	**10.06****
Age	--	11.21 (1.91)	8.49	15.12	11.24 (2.01)	8.72	15.89	-0.17 (431)	0.72
Difference Score									
Peer Preocc. Weight/Dieting	--	3.56 (4.58)	0.00	27.00	3.82 (3.65)	0.00	21.00	-.41 (178)	0.39
Appearance Conversations	--	2.86 (3.22)	0.00	18.00	3.08 (2.84)	0.00	13.00	-.51 (201)	0.00
Friends Influence	--	2.55 (3.32)	0.00	20.00	2.62 (2.82)	0.00	11.00	-.14 (198)	0.11
Peer Attribution	--	2.01 (2.94)	0.00	12.00	1.96 (2.69)	0.00	12.00	.13 (199)	0.44
Thin-Ideal Internalization	--	0.75 (0.57)	0.00	2.89	0.63 (0.54)	0.00	2.44	1.48 (189)	0.45

Table 7.

Within-Individual and Within-Pair Correlations

	Full Sample	MZ Twins	DZ Twins	MZ/DZ Difference Two-Tailed Z
	Within-Individual Correlations			
Peer Preocc. Weight/Dieting	**.32****	**.32****	**.31****	0.11 *ns*
Appearance Conversations	**.36****	**.38****	**.35****	0.34 *ns*
Friends as Source of Influence	**.37****	**.38****	**.36****	0.23 *ns*
Peer Attribution	**.41****	**.36****	**.47****	1.32 *ns*
	Within-Twin Pair Difference Score Correlations			
Peer Preocc. Weight/Dieting	**.21****	**.29****	0.09	1.32 *ns*
Appearance Conversations	**.24****	**.27****	0.20	0.50 *ns*
Friends as Source of Influence	**.20****	**.23***	0.14	0.63 *ns*
Peer Attribution	**.28****	**.28****	**.27***	0.07 *ns*

Note. Peer Preocc. Weight/Dieting = Peer Preoccupation with Weight/Dieting Scale. Appearance Conversations = Appearance Conversations with Friends Scale. Friends as a Source of Influence = Friends as a Source of Influence Scale. Peer Attribution = Peer Attribution (Appearance) Scale .For Within-Individual Correlations, Sample sizes are as follows: Full Sample N = 374-414, MZ N = 193-212, DZ N = 181-202. For Within Twin Pair Difference Score Correlations, Sample sizes are Full Sample N = 168-203; MZ N = 87-103; DZ N =81-99

Table 8.

Co-twin control analyses examining the association between weight-focused peer groups and thin-ideal internalization

	Between-Pair b (SE)		Within-Pair b (SE)		
Peer Measures	MZ Twins	DZ Twins	MZ Twins	DZ Twins	MZ/DZ Difference
Peer Preocc. Weight/Dieting	**0.34 (0.07)****	**0.26 (0.08)****	**0.20 (0.06)****	0.01 (0.06)	**0.19 (0.08)***
Appearance Conversations	**0.39 (0.07)****	**0.35 (0.08)****	**0.19 (0.05)****	0.09 (0.06)	-0.04 (0.11)
Friends as Source of Influence	**0.33 (0.07)****	**0.29 (0.07)****	**0.18 (0.05)****	0.06 (0.05)	-0.13 (0.11)
Peer Attribution	**0.33 (0.07)****	**0.46 (0.07)****	**0.19 (0.05)****	**0.15 (0.06)****	-0.19 (0.10)

Note. Peer Preocc. Weight/Dieting = Peer Preoccupation with Weight/Dieting Scale; Appearance Conversations = Appearance Conversations with Friends Scale; Friends as a Source of Influence = Friends as a Source of Influence Scale; Peer Attribution = Peer Attribution (Appearance) Scale; b = unstandardized fixed-effect estimate; SE = standard error; MZ = Monozygotic; DZ = Dizygotic. Unstandardized fixed-effect estimates, followed by standard errors in parentheses, are presented in the table. Although fixed-effect estimates are unstandardized, thin-ideal internalization scores were standardized prior to analyses to faciliate interpretation of these effects. All analyses included BMI, age, and pubertal development as covariates. Between-pair effects approximate individual level effects. Within-pair effects demonstrate effects while controlling for shared environmental selection effects (MZ and DZ twins) and genetic selection effects (entirely in MZ twins, partially in DZ twins). Significant within-pair estimates in MZ twins suggest socialization effects, particularly when the DZ within-pair estimate is also significant, or not statistically different from effects in MZ twins. * = $p<.05$ **=$p<.01$.

Figure 1.

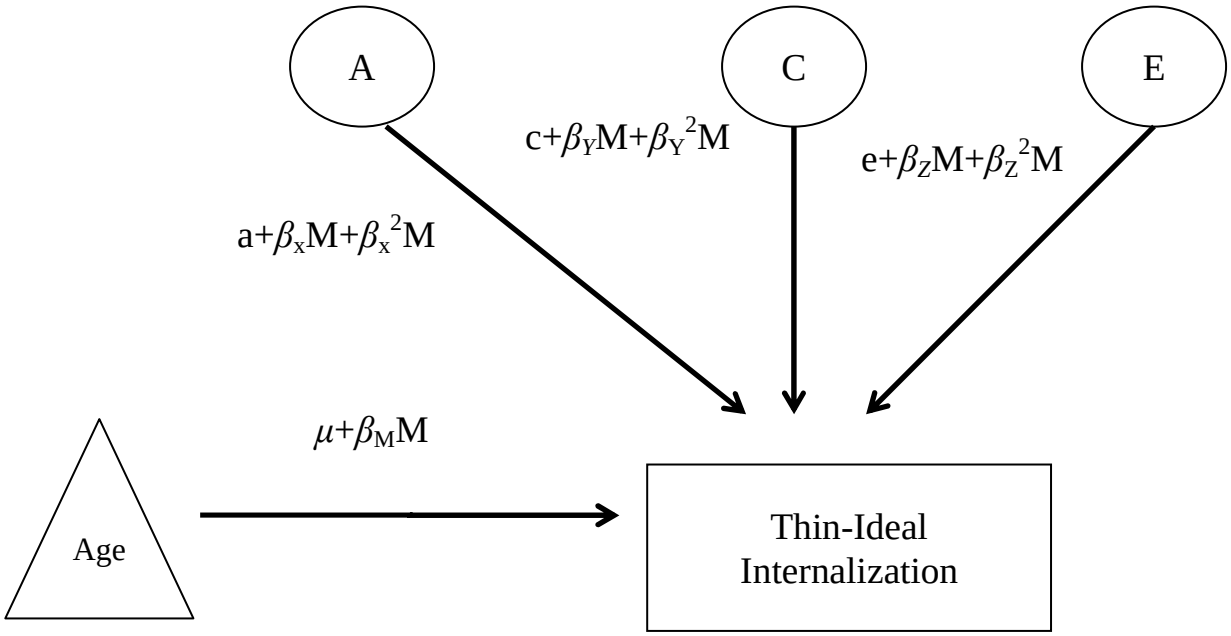


Figure 1. Path diagram for the standard univariate moderation model. Figure illustrates model for one twin only. Age=age moderator; A= additive genetic effects; C,=shared environmental effects; E=nonshared environmental effects; M=Moderator; β_M=phenotypic regression coefficient; a, c, and e=paths or intercepts; $\beta_{X,}$ $\beta_{Y,}$ and $\beta_{Z,}$=linear moderators, β_x^2, β_x^2, and β_x^2=quadratic moderators.

Figure 2.

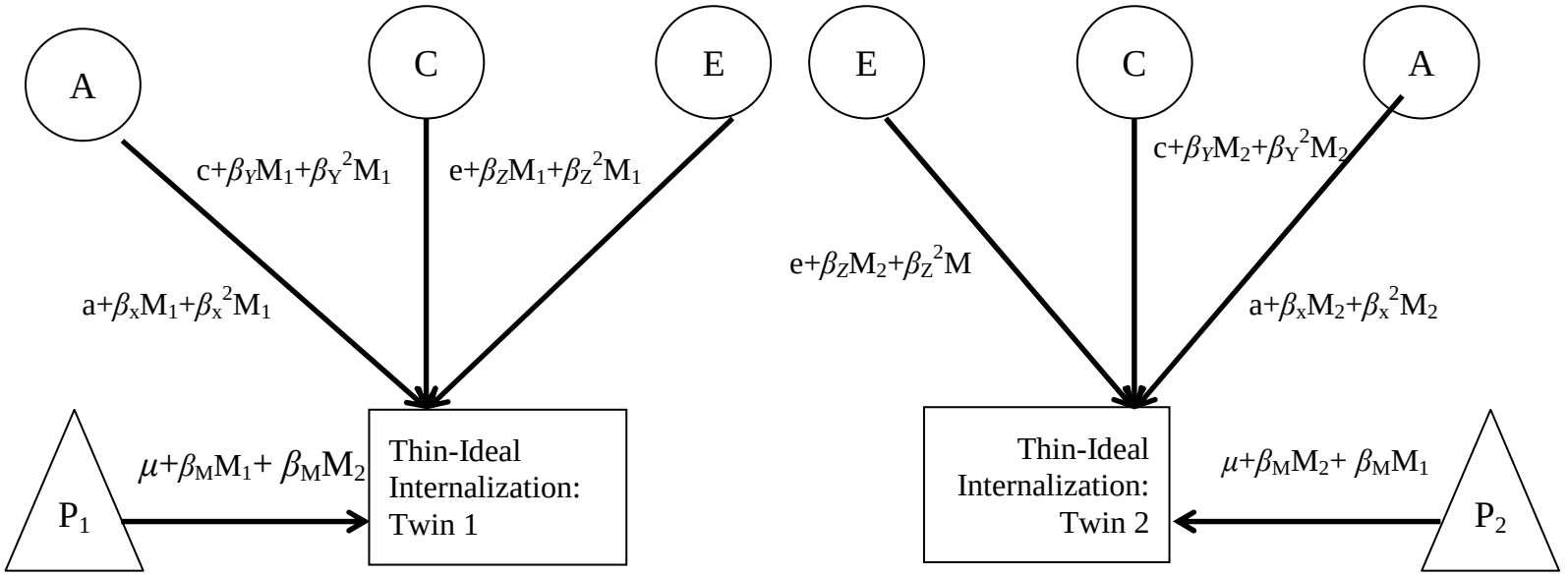
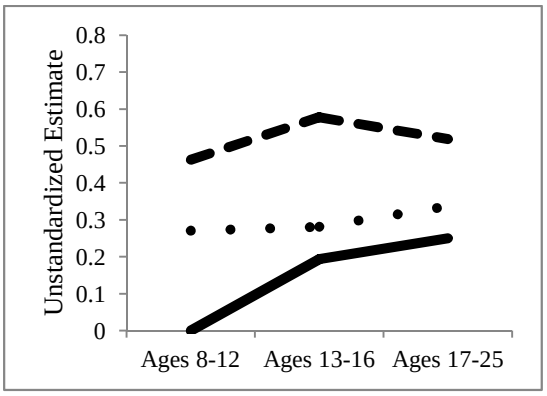
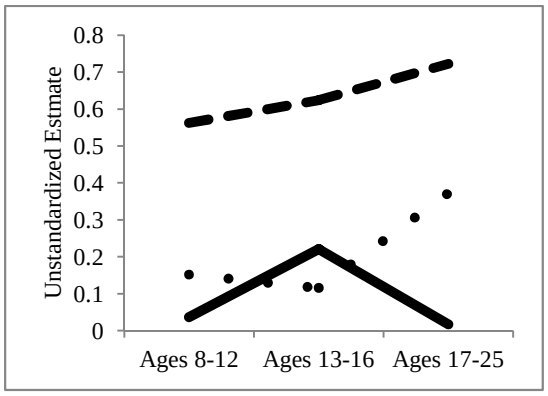


Figure 2. Path diagram of extended univariate moderation model. Although prior figures include a path diagram only for one twin, this figure includes twin 1 and twin 2 in order to illustrate the way the moderator value for twin 2 is accounted for in the model for twin 1 and vice-versa. The model is pictured for only one twin type, but parameters are fit separately for MZ and DZ twins. P_1=pubertal development in twin 1; P_2=pubertal development in twin 2; A= additive genetic effects; C,=shared environmental effects; E=nonshared environmental effects; M_1 and M_2=Moderator in twin 1 and twin 2, respectively; β_M=phenotypic regression coefficient; a, c, and e=paths or intercepts; $\beta_{X,}$ $\beta_{Y,}$ and $\beta_{Z,}$=linear moderators, $\beta_x{}^2$, $\beta_x{}^2$, and $\beta_x{}^2$=quadratic moderator

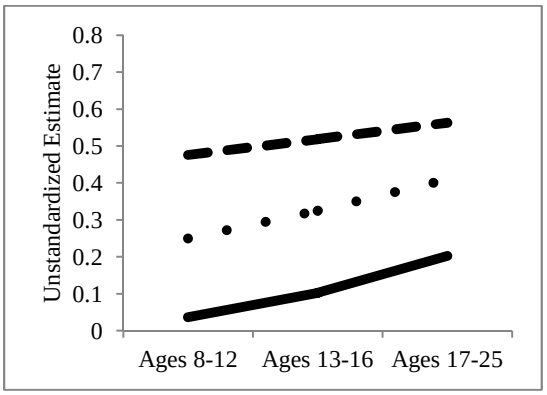
3a.



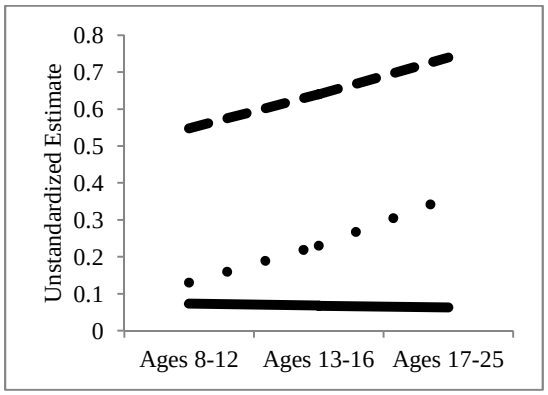
3b.



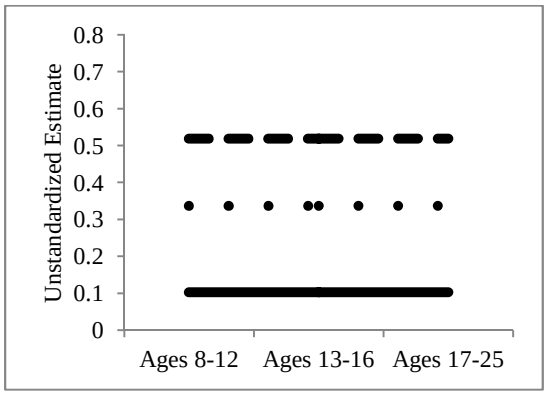
3c.



3d.



3e.



3f.

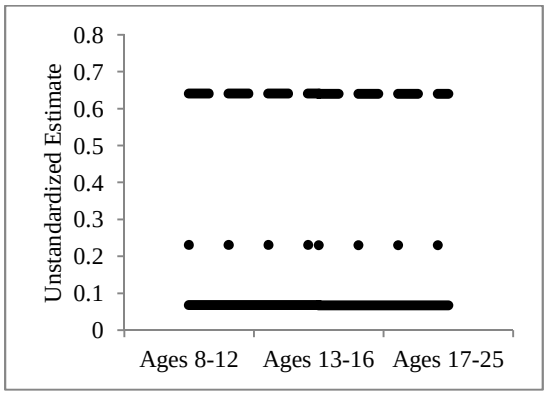
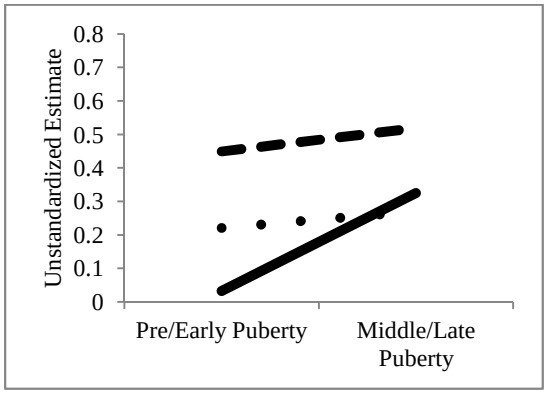
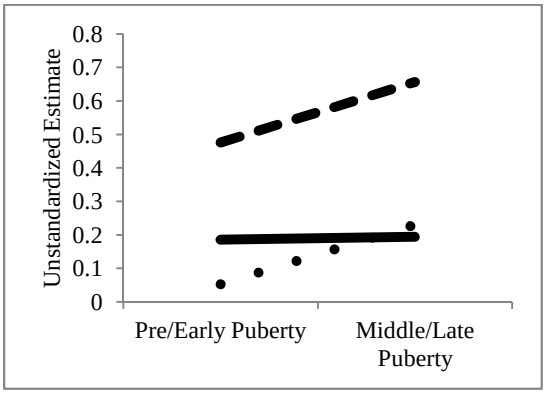


Figure 3. Age Moderation Results. a =additive genetic effects (▬▬), c = shared environmental effects (• • • • •) e = nonshared environmental effects (▬ ▬ ▬). Unstandardized genetic, shared environmental, and nonshared environmental variance components by age. The full models are graphed in panels 3a (controlling for BMI) and 3b (controlling for BMI and disordered eating). Linear models are graphed in panels 3c (controlling for BMI) and 3d (controlling for disordered eating. The no moderation models are indicated in graphs 3e (controlling for BMI) and 3f (controlling for BMI and disordered eating). None of the differences in etiological effects across age (figures 3a – 3d) are statistically significant.

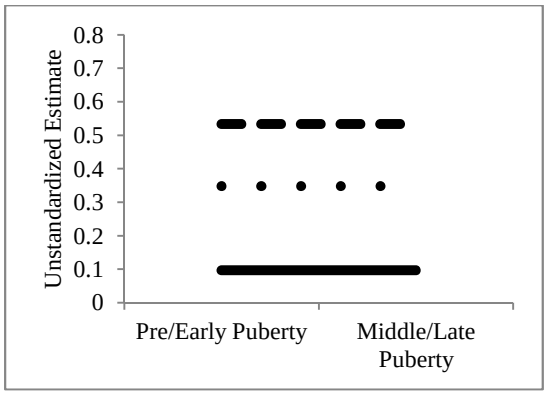
4a.



4b.



4c.



4d.

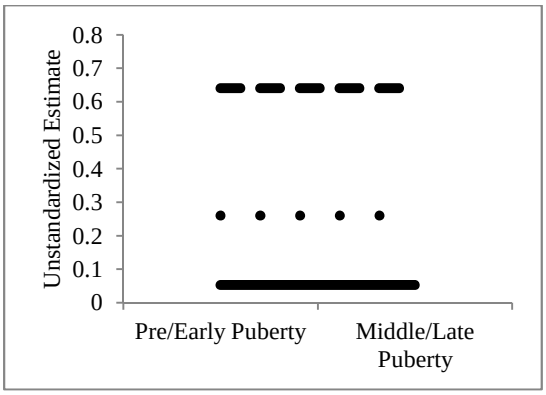


Figure 4. Puberty Moderation Results. a =additive genetic effects (▬▬), c = shared environmental effects (• • • •) e = nonshared environmental effects (- - -). Graphs depict unstandardized genetic, shared environmental, and nonshared environmental variance components by age. The full models are graphed in panels 4a (controlling for BMI) and 4b (controlling for BMI and disordered eating). The no moderation models are indicated in graphs 4c (controlling for BMI) and 4d (controlling for BMI and disordered eating). For models controlling for BMI only, the linear model (Figure 4a) provided the best fit to the data. For models controlling for BMI and disordered eating, the no moderation model (Figure 4d) provided the best fit. In figures 4a and 4b, the only statistically significant change in etiological effects across puberty is the linear increase in additive genetic effects for the model controlling for BMI only (figure 4a).

Figure 5a.

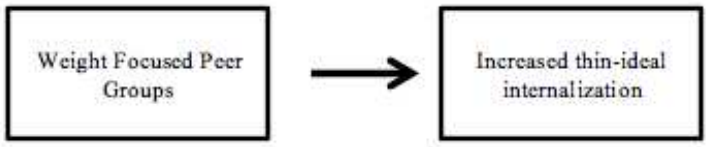


Figure 5b.

Figure 5c.

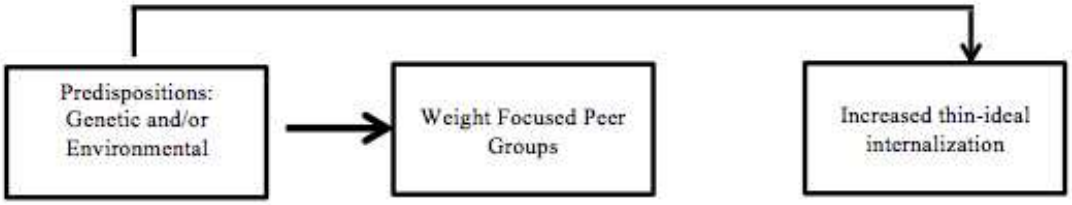


Figure 5. Possible exposure versus selection effects for associations between weight-focused peer groups and thin-ideal internalization. Figure 5a demonstrates an exposure effect, whereby certain experiences (e.g., weight teasing) directly leads to increases in thin-ideal internalization. Figure 5b demonstrates one type of selection effect, whereby genetic and/or environmental selection factors lead to increases in certain types of risk experiences (e.g., weight teasing) as well as increases in the outcome (thin-ideal internalization), but the increase in risk experiences also leads to increases in thin-ideal internalization. Figure 5c demonstrates the second type of selection effect, main effects of the selection factor, whereby preexisting genetic and/or environmental selection factors cause both the risk factor and the outcome (thin ideal internalization).

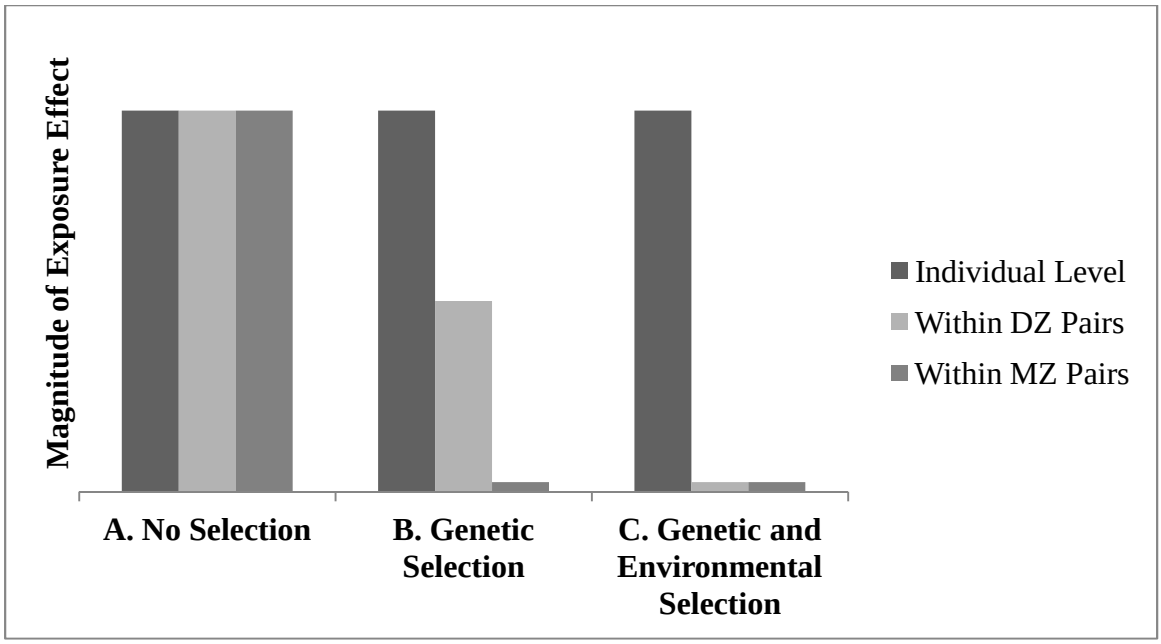


Figure 6. Interpretation of results within a co-twin control design. Graph indicates the hypothetical exposure effect of body-conscious peer groups on thin-ideal internalization when measured at the individual level, within dizygotic (DZ) twins pairs, and within monozygotic (MZ) twin pairs. Individual level effects do not control for any selection processes. Effects in DZ twins control partially for genetic and entirely for shared environmental selection effects. Effects in MZ twins control entirely for genetic and shared environmental selection effects.

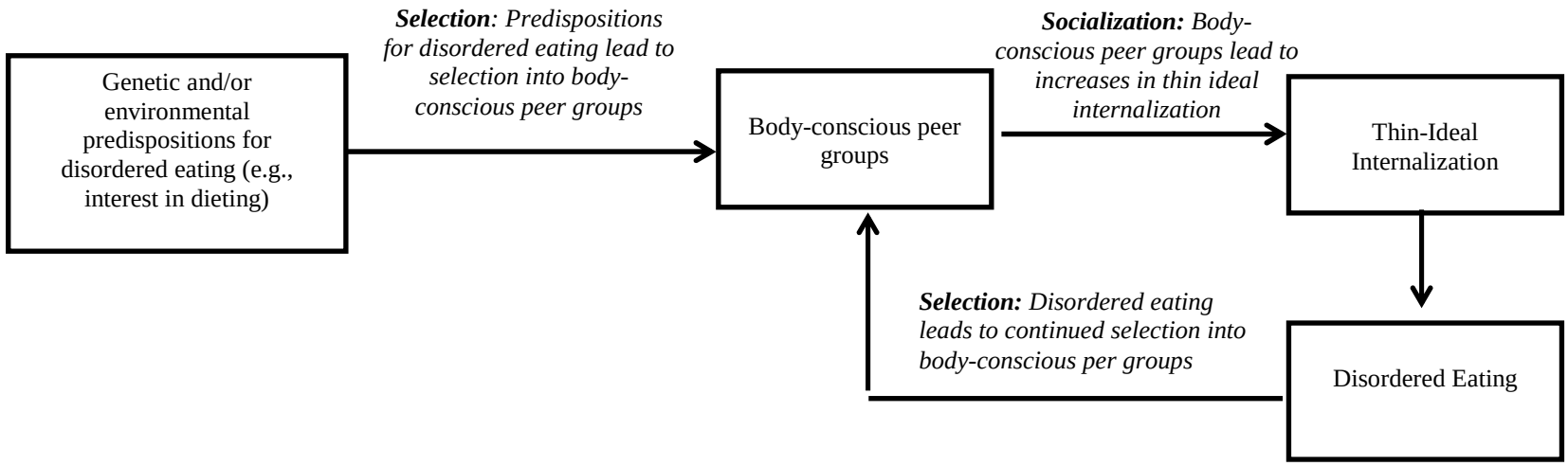


Figure 7. Proposed Interplay among Socialization and Selection Processes. This model integrates results from O'Connor et al (in preparation), which suggest that selection factors account for associations between body-conscious peer groups and disordered eating, and results from the current study, which suggest that socialization effects account for associations between body-conscious peer groups and thin-ideal internalization.

REFERENCES

REFERENCES

Becker, C. B., Bull, S., Schaumberg, K., Cauble, A., & Franco, A. (2008). Effectiveness of peer-led eating disorders prevention: a replication trial. *Journal of Consulting and Clinical Psychology, 76*(2), 347.

Becker, C. B., Smith, L. M., & Ciao, A. C. (2006). Peer-facilitated eating disorder prevention: A randomized effectiveness trial of cognitive dissonance and media advocacy. *Journal of Counseling Psychology, 53*(4), 550.

Burt, S. A. (2009). Rethinking environmental contributions to child and adolescent psychopathology: A meta-analysis of shared environmental influences. *Psychological Bulletin, 135*(4), 608-637. doi: 10.1037/a0015702

Burt, S. A., Donnellan, M. B., Humbad, M. N., Hicks, B. M., McGue, M., & Iacono, W. G. (2010). Does Marriage Inhibit Antisocial Behavior?: An Examination of Selection vs Causation via a Longitudinal Twin Design. *Archives of General Psychiatry, 67*(12), 1309.

Burt, S. A., & Klump, K. L. (2012). The Michigan State University Twin Registry (MSUTR): An update. *Twin Research and Human Genetics, 16*, 344-350.

Cafri, G., Yamamiya, Y., Brannick, M., & Thompson, J. K. (2005). The Influence of Sociocultural Factors on Body Image: A Meta‐Analysis. *Clinical Psychology: Science and Practice, 12*(4), 421-433.

Calogero, R. M., Davis, W. N., & Thompson, J. K. (2004). The Sociocultural Attitudes Toward Appearance Questionnaire (SATAQ-3): reliability and normative comparisons of eating disordered patients. *Body Image, 1*(2), 193-198. doi: 10.1016/j.bodyim.2004.01.004

Chumlea, W. C., Schubert, C. M., Roche, A. F., Kulin, H. E., Lee, P. A., Himes, J. H., & Sun, S. S. (2003). Age at menarche and racial comparisons in US girls. *Pediatrics, 111*(1), 110-113.

Clark, L., & Tiggemann, M. (2006). Appearance Culture in Nine‐to 12‐Year‐Old Girls: Media and Peer Influences on Body Dissatisfaction. *Social Development, 15*(4), 628-643.

Coughlin, J. W., & Kalodner, C. (2006). Media literacy as a prevention intervention for college women at low-or high-risk for eating disorders. *Body Image, 3*(1), 35-43.

Cronk, N. J., Slutske, W. S., Madden, P. A., Bucholz, K. K., Reich, W., & Heath, A. C. (2002). Emotional and behavioral problems among female twins: An evaluation of the equal environments assumption. *Journal of the American Academy of Child and Adolescent Psychiatry, 41*(7), 829-837.

Culbert, K. M., Burt, S. A., McGue, M., Iacono, W. G., & Klump, K. L. (2009). Puberty and the genetic diathesis of disordered eating attitudes and behaviors. *Journal of Abnormal Psychology, 118*, 788-796.

Durkin, S. J., & Paxton, S. J. (2002). Predictors of vulnerability to reduced body image satisfaction and psychological wellbeing in response to exposure to idealized female media images in adolescent girls. *Journal of Psychosomatic Research, 53*(5), 995-1005.

Gordon, R. A., Lahey, B. B., Kawai, E., Loeber, R., STOUTHAMER‐LOEBER, M., & Farrington, D. P. (2004). Antisocial behavior and youth gang membership: selection and socialization. *Criminology, 42*(1), 55-88.

Harriger, J. A., Calogero, R. M., Witherington, D. C., & Smith, J. E. (2010). Body size stereotyping and internalization of the thin ideal in preschool girls. *Sex Roles, 63*(9), 609-620.

Hay, P. J., Mond, J., Buttner, P., & Darby, A. (2008). Eating disorder behaviors are increasing: findings from two sequential community surveys in South Australia. *PLoS One, 3*(2), e1541.

Herman-Giddens, M. E., Slora, E. J., Wasserman, R. C., Bourdony, C. J., Bhapkar, M. V., Koch, G. G., & Hasemeier, C. M. (1997). Secondary sexual characteristics and menses in young girls seen in office practice: a study from the Pediatric Research in Office Settings network. *Pediatrics, 99*(4), 505-512.

Hermes, S. F., & Keel, P. K. (2003). The influence of puberty and ethnicity on awareness and internalization of the thin ideal. *International Journal of Eating Disorders, 33*(4), 465-467.

Hudson, J. I., Hiripi, E., Pope Jr, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry, 61*(3), 348-358.

Jones, D. C., Vigfusdottir, T. H., & Lee, Y. (2004). Body Image and the Appearance Culture Among Adolescent Girls and Boys An Examination of Friend Conversations, Peer Criticism, Appearance Magazines, and the Internalization of Appearance Ideals. *Journal of adolescent research, 19*(3), 323-339.

Keery, H., Boutelle, K., Van Den Berg, P., & Thompson, J. K. (2005). The impact of appearance-related teasing by family members. *Journal of Adolescent Health, 37*(2), 120-127.

Keery, H., van den Berg, P., & Thompson, J. K. (2004). An evaluation of the Tripartite Influence Model of body dissatisfaction and eating disturbance with adolescent girls. *Body Image, 1*(3), 237-251. doi: 10.1016/j.bodyim.2004.03.001

Keski-Rahkonen, A., Hoek, H., Linna, M., Raevuori, A., Sihvola, E., Bulik, C., . . . Kaprio, J. (2010). Incidence and outcomes of bulimia nervosa: a nationwide population-based study. *Psychological Medicine, 39*(5), 823.

Klump, K. L., Bulik, C. M., Kaye, W. H., Treasure, J., & Tyson, E. (2009). Academy for Eating Disorders position paper: Eating disorders are serious mental illnesses. *International Journal of Eating Disorders, 42*(2), 97-103. doi: 10.1002/eat.20589

Klump, K. L., & Burt, S. A. (2006). The Michigan State University Twin Registry: Genetic, environmental, and neurobiological influences on behavior across development. *Twin Research, 9*, 971-977. doi: 10.1375/twin.9.6.971

Klump, K. L., Burt, S. A., McGue, M., & Iacono, W. G. (2007). Changes in genetic and environmental influences on disordered eating across adolescence: A longitudinal twin study. *Archives of General Psychiatry, 64*, 1409-1415.

Klump, K. L., Burt, S. A., Spanos, A., McGue, M., Iacono, W. G., & Wade, T. D. (2010). Age differences in genetic and environmental influences on weight and shape concerns. *International Journal of Eating Disorders*.

Klump, K. L., Keel, P. K., Racine, S. E., Burt, S. A., Sisk, C. L., Neale, M., . . . Hu, J. Y. (2013). The interactive effects of estrogen and progesterone on changes in binge eating across the menstrual cycle. *Journal of Abnormal Psychology, 122*, 131-137. doi: 10.1037/a0029524

Klump, K. L., Keel, P. K., Sisk, C., & Burt, S. A. (2010). Preliminary evidence that estradiol moderates genetic influences on disordered eating attitudes and behaviors during puberty. *Psychological Medicine, 40*(10), 1745-1753.

Klump, K. L., McGue, M., & Iacono, W. G. (2000). Age differences in genetic and environmental influences on eating attitudes and behaviors in preadolescent and adolescent female twins. *Journal of Abnormal Psychology, 109*, 239-251.

Klump, K. L., McGue, M., & Iacono, W. G. (2003). Differential heritability of eating attitudes and behaviors in prepubertal versus pubertal twins. *International Journal of Eating Disorders, 33*, 287-292. doi: 10.1002/eat.10151

Klump, K. L., Perkins, P. S., Burt, S. A., McGue, M., & Iacono, W. G. (2007). Puberty moderates genetic influences on disordered eating. *Psychological Medicine, 37*, 627-634. doi: 10.1017/S0033291707000189

Klump, K. L., Suisman, J. L., Burt, S. A., McGue, M., & Iacono, W. G. (2009). Genetic and environmental influences on disordered eating: An adoption study. *Journal of Abnormal Psychology, 118*(4), 797-805. doi: 10.1037/a0017204

Little, R. J. A., & Rubin, D. B. (1987). *Statistical Analysis with Missing Data*. New York: Wiley.

Lykken, D. T., Bouchard, T. J., McGue, M., & Tellegen, A. (1990). The Minnesota Twin Family Registry: Some initial findings. *Acta Gemellogicae et Medicae, 39*, 35-70.

Lynam, D. R., Hoyle, R. H., & Newman, J. P. (2006). The perils of partialling cautionary tales from aggression and psychopathy. *Assessment, 13*(3), 328-341.

Machado, P. P. P., Machado, B. C., Gonçalves, S., & Hoek, H. W. (2007). The prevalence of eating disorders not otherwise specified. *International Journal of Eating Disorders, 40*(3), 212-217.

Malouff, J. M., Rooke, S. E., & Schutte, N. S. (2008). The heritability of human behavior: results of aggregating meta-analyses. *Current Psychology, 27*(3), 153-161.

Markland, D., & Oliver, E. J. (2008). The sociocultural attitudes towards appearance questionnaire-3: a confirmatory factor analysis. *Body Image, 5*(1), 116-121.

Markon, K. E., & Krueger, R. F. (2004). An empirical comparison of information-theoretic selection criteria for multivariate behavior genetic models. *Behavior Genetics, 34*(6), 593-610.

Martin, N. G., Eaves, L. J., Kearsey, M. J., & Davies, P. (1978). The power of the classical twin study. *Heredity, 40*, 97-116.

McGue, M., Osler, M., & Christensen, K. (2010). Causal Inference and Observational Research: The Utility of Twins. *Perspectives on Psychological Science, 5*(5), 546-556.

Mintz, L. B., & Betz, N. E. (1988). Prevalence and correlates of eating disordered behaviors among undergraduate women. *Journal of Counseling Psychology, 35*(4), 463.

Neale, M. C. (1995). Mx: Statistical Modeling (Version 3rd Edition). Richmond, VA: Department of Psychology.

O'Connor, S., Burt, S. A., & Klump, K. L. (in preparation). What drives the association between body-conscious peer groups and disordered eating? Disentangling genetic and environmental selection from pure socialization effects.

Parker, J. G., Rubin, K. H., Erath, S. A., Wojslawowicz, J. C., & Buskirk, A. A. (2006). Peer relationships, child development, and adjustment: A developmental psychopathology perspective. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology, Vol 2: Risk, Disorder, and Adaptation* (pp. 421-477). Hoboken, NJ, US: John Wiley and Sons, Inc.

Paxton, S. J., Schutz, H. K., Wertheim, E. H., & Muir, S. L. (1999). Friendship clique and peer influences on body image concerns, dietary restraint, extreme weight-loss behaviors, and binge eating in adolescent girls. *Journal of Abnormal Psychology, 108*(2), 255.

Peeters, H., Van Gestel, S., Vlietinck, R., Derom, C., & Derom, R. (1998). Validation of a telephone zygosity questionnaire in twins of known zygosity. *Behavior Genetics, 28*, 159-163. doi: 10.1023/A:1021416112215

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence, 17*, 117-133. doi: 10.1007/BF01537962

Posthuma, D., & Boomsma, D. I. (2000). A note on the statistical power in extended twin designs. *Behavior Genetics, 30*(2), 147-158.

Purcell, S. (2002). Variance components models for gene-environment interaction in twin analysis. *Twin Research, 5*, 554-571.

Rideout, V. J., Foehr, U. G., & Roberts, D. F. (2010). Generation M: Media in the Lives of 8-to 18-Year-Olds. *Henry J. Kaiser Family Foundation*.

Rubin, D. B. (2007). The design versus the analysis of observational studies for causal effects: parallels with the design of randomized trials. *Statistics in Medicine, 26*(1), 20-36.

Rubin, D. B. (2008). For objective causal inference, design trumps analysis. *The Annals of Applied Statistics*, 808-840.

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype environment effects. *Child Development, 54*(2), 424-435.

Schutz, H. K., Paxton, S. J., & Wertheim, E. H. (2002). Investigation of Body Comparison Among Adolescent Girls. *Journal of Applied Social Psychology, 32*(9), 1906-1937.

Shroff, H., & Thompson, J. K. (2006a). Peer influences, body-image dissatisfaction, eating dysfunction and self-esteem in adolescent girls. *Journal of Health Psychology, 11*(4), 533-551.

Shroff, H., & Thompson, J. K. (2006b). The tripartite influence model of body image and eating disturbance: A replication with adolescent girls. *Body Image, 3*(1), 17-23. doi: 10.1016/j.bodyim.2005.10.004

Slevec, J. H., & Tiggemann, M. (2011). Predictors of body dissatisfaction and disordered eating in middle-aged women. *Clinical Psychology Review, 31*(4), 515-524.

Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin, 128*(5), 825.

Stice, E., Becker, C. B., & Yokum, S. (2013). Eating disorder prevention: Current evidence‐base and future directions. *International Journal of Eating Disorders, 46*(5), 478-485.

Stice, E., Butryn, M., Rohde, P., & Shaw, H. (2013). *Pilot trial of a group dissonance-based treatment for DSM-5 eating disorders*. Paper presented at the Eating Disorder Research Society, Bethesda, Maryland.

Stice, E., Chase, A., Stormer, S., & Appel, A. (2001). A randomized trial of a dissonance‐based eating disorder prevention program. *International Journal of Eating Disorders, 29*(3), 247-262.

Stice, E., Mazotti, L., Weibel, D., & Agras, W. S. (2000). Dissonance prevention program decreases thin‐ideal internalization, body dissatisfaction, dieting, negative affect, and bulimic symptoms: A preliminary experiment. *International Journal of Eating Disorders, 27*(2), 206-217.

Stice, E., & Shaw, H. (2004). Eating Disorder Prevention Programs: A Meta-Analytic Review. *Psychological Bulletin, 130*(2), 206.

Stice, E., Shaw, H., & Marti, C. N. (2007). A meta-analytic review of eating disorder prevention programs: encouraging findings. *Annu. Rev. Clin. Psychol., 3*, 207-231.

Suisman, J. L., O'Connor, S. M., Sperry, S., Thompson, J. K., Keel, P. K., Burt, S. A., . . . Klump, K. L. (2012). Genetic and environmental influences on thin-ideal internalization. *International Journal of Eating Disorders, 45*(8), 942-948. doi: 10.1002/eat.22056

Suisman, J. L., Thompson, J. K., Burt, S. A., Keel, P. K., Neale, M., Boker, S., . . . Klump, K. L. (in preparation). Common genetic influences between thin-ideal internalization and disordered eating.

Suisman, J. L., Thompson, J. K., Keel, P. K., Burt, S. A., Neale, M., Boker, S., . . . Klump, K. L. (submitted). Genetic and environmental influences on thin-ideal internalization across development.

Thompson, J. K., Heinberg, L. J., Altabe, M. N., & Tantleff-Dunn, S. (1999). *Exacting beauty: Theory, assessment, and treatment of body image disturbance*: American Psychological Association.

Thompson, J. K., Shroff, H., Herbozo, S., Cafri, G., Rodriguez, J., & Rodriguez, M. (2007). Relations among multiple peer influences, body dissatisfaction, eating disturbance, and self-esteem: A comparison of average weight, at risk of overweight, and overweight adolescent girls. *Journal of Pediatric Psychology, 32*(1), 24-29.

Thompson, J. K., & Stice, E. (2001). Thin-ideal internalization: Mounting evidence for a new risk factor for body-image disturbance and eating pathology. *Current Directions in Psychological Science, 10*(5), 181-183.

Thompson, J. K., van den Berg, P., Roehrig, M., Guarda, A. S., & Heinberg, L. J. (2004). The sociocultural attitudes towards appearance scale-3 (SATAQ-3): Development and validation. *International Journal of Eating Disorders, 35*(3), 293-304.

Thornton, L. M., Mazzeo, S. E., & Bulik, C. M. (2011). The heritability of eating disorders: methods and current findings *Behavioral neurobiology of eating disorders* (pp. 141-156): Springer.

van den Berg, P., Thompson, J. K., Obremski-Brandon, K., & Coovert, M. (2002). 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, 53*(5), 1007-1020.

van der Sluis, S., Posthuma, D., & Dolan, C. V. (2012). A Note on False Positives and Power in G× E Modelling of Twin Data. *Behavior Genetics*, 1-17.

von Ranson, K. M., Klump, K. L., Iacono, W. G., & McGue, M. (2005). The Minnesota Eating Behavior Survey: A brief measure of disordered eating attitudes and behaviors. *Eating Behaviors, 6*, 373-392. doi: 10.1016/j.eatbeh.2004.12.002

Yamamiya, Y., Cash, T. F., Melnyk, S. E., Posavac, H. D., & Posavac, S. S. (2005). Women's exposure to thin-and-beautiful media images: body image effects of media-ideal internalization and impact-reduction interventions. *Body Image, 2*(1), 74-80.

Yamamiya, Y., Shroff, H., & Thompson, J. K. (2008). The tripartite influence model of body image and eating disturbance: a replication with a Japanese sample. *International Journal of Eating Disorders, 41*(1), 88-91. doi: 10.1002/eat.20444