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Self-Objectification, Socialization Effects, and Disordered Eating

Self-Objectification and Disordered Eating: A Meta-Analysis

Abstract

Objective—Objectification theory posits that self-objectification increases risk for disordered eating.

Method—The current study sought to examine the relationship between self-objectification and disordered eating using meta-analytic techniques.

Results—Data from 53 cross-sectional studies (73 effect sizes) revealed a significant moderate positive overall effect ($r = 0.39$), which was moderated by gender, ethnicity, sexual orientation, and measurement of self-objectification. Specifically, larger effect sizes were associated with female samples and the Objectified Body Consciousness Scale. Effect sizes were smaller among heterosexual men and African American samples. Age, body mass index, country of origin, measurement of disordered eating, sample type and publication type were not significant moderators.

Discussion—Overall, results from the first meta-analysis to examine the relationship between self-objectification and disordered eating provide support for one of the major tenets of objectification theory and suggest that self-objectification may be a meaningful target in eating disorder interventions, though further work is needed to establish temporal and causal relationships. Findings highlight current gaps in the literature (e.g., limited representation of males, and ethnic and sexual minorities) with implications for guiding future research.

Keywords

Objectification; disordered eating; body surveillance; sociocultural; meta-analysis; review

Introduction

While clinical diagnoses of anorexia nervosa, bulimia nervosa, and binge eating disorder are relatively rare, affecting roughly .09%, 1.5%, and 3.5% of women and .03%, .05%, and 2.0% of men respectively (Hudson, Hiripi, Pope, & Kessler, 2007), engagement in subclinical levels of disordered eating (e.g., skipping meals, use of weight loss pills or extreme diets, subthreshold levels of binging/purging, etc.) is alarmingly common in both

males and females (Mintz & Betz, 1988; Mond et al., 2014). Eating disorders and disordered eating place individuals at risk for a number of negative health outcomes (Fairburn, Cooper, & Waller, 2008; Mehler, Birmingham, Crow, & Jahraus, 2010; Sharp & Freeman, 1993) and are associated with significant psychosocial impairment, high rates of comorbid psychopathology, and elevated mortality rates (Agras, et al., 2004; O'Brien & Vincent, 2003). As current intervention approaches are limited in their efficacy (Berkman et al., 2006; Keel & Haedt, 2008), identification of factors that may contribute to or maintain disturbed eating patterns represents an important area of inquiry with significant implications for intervention.

Research suggests that certain environments may increase vulnerability to the development of eating disorders and disordered eating (Striegel-Moore, Silberstein, & Rodin, 1986). In particular, accruing evidence suggests that environments that promote the importance of physical appearance significantly contribute to disordered eating attitudes and behaviors (Ata, Schaefer, & Thompson, 2014). Objectification theory (Fredrickson & Roberts, 1997), which originated within a feminist framework but has since been expanded to address eating disturbances observed in men, is a sociocultural theory of eating disturbance that attempts to explain how social environments in which the female form is viewed as an aesthetic object to be evaluated by others contributes to the disproportionately high rates of disordered eating observed in girls and women. The theory suggests that females in Western societies commonly experience instances of sexual objectification wherein "a woman's sexual parts or functions are separated out from her person, reduced to status of mere instruments, or else regarded as if they were capable of representing her" (Bartky, 1990, p.35). Examples of such sexually-objectifying experiences include catcalls, leering or sexualized gaze, sexual comments, and media images highlighting the feminine form as an object of pleasure (Calogero, Tantleff-Dunn, & Thompson, 2011).

Objectification theory proposes that repeated exposure to sexually objectifying experiences and the broader societal reinforcement of the acceptability of such practices leads young girls and women to internalize these messages, learning to view their bodies from the external observer's perspective and to conceptualize their own bodies as objects to be scrutinized by others (referred to as self-objectification) (Fredrickson & Roberts, 1997). Women who have adopted such a view are theorized to experience increased self-consciousness regarding their physical appearance, which manifests in heightened levels of self-surveillance or body-monitoring. In other words, sexual objectification refers to the interpersonal experience of having one's body, appearance, or sexuality highlighted by another person or entity; self-objectification refers to a learned self-schema regarding the importance of one's body and appearance relative to other aspects of the self; and self-surveillance represents the cognitive and behavioral manifestation of self-objectification characterized by self-conscious monitoring of one's appearance (Fitzsimmons-Craft, 2011). Fredrickson and Roberts (1997) propose that the continual monitoring and evaluation of one's appearance leads to a number of negative psychological outcomes including body shame and appearance anxiety, which then contribute to the development of disordered eating.

In the years following Fredrickson and Robert's original publication, objectification theory has received a considerable amount of research attention and extant work suggests that the theory may provide a useful framework for understanding the process by which individuals develop disordered eating. Indeed, a growing body of research provides support for the relationship between self-objectification (or its manifestation self-surveillance) and disordered eating (Calogero, 2009; Daubenmier, 2005; Lindner, Tantleff-Dunn, & Jentsch, 2012; Noll & Fredrickson, 1998; Tiggemann & Slater, 2001; Tiggemann & Williams, 2012). Moreover, extant work suggests a possible moderating influence of several demographic and methodological variables.

Objectification theory posits that sexual and self-objectification is largely a female experience. Consistent with theory, research suggests that although boys and men report experiences of sexual objectification, girls and women endorse considerably higher levels of sexual and self-objectification (Bryant 1993; Klonoff, 2000; Kozee, Tylka, Augustus-Horvath, & Denchik, 2007; Murnen & Smolak, 2000; Swim, Hyers, Cohen, & Ferguson, 2001). Women also appear to be more sensitive to objectifying messages, exhibiting greater psychological impact when primed with objectifying words (Roberts & Gettman, 2004). In addition, rates of both diagnosable eating disorders and disordered eating appear to be higher among females than among males (Hudson et al., 2007), and some studies suggest that the relationship between self-objectification and disordered eating may be stronger among females (Calogero, 2009). Therefore, it is possible that gender moderates the relationship between self-objectification and disordered eating.

Although the majority of the work examining self-objectification has utilized college samples, limited work using adolescent and older community samples indicates that levels of objectification among adolescent girls (age 12 to 16) may be similar to levels observed in undergraduate women (Slater and Tiggemann, 2002), but subsequently decline with increasing age (Greenleaf, 2005; Roberts, 2004; Tiggemann & Lynch, 2001). Prevalence of disordered eating appears to increase through adolescence and decline in adulthood (Heatherton, Mahamedi, Striepe, Field, & Keel, 1997; Jones, Bennett, Olmsted, Lawson, & Rodin, 2001; Polivy & Herman, 1985). Further, this is some indication that the relationship between self-objectification and disordered eating may vary with age (Greenleaf, 2005). It is therefore possible that age may moderate the relationship between self-objectification and disordered eating.

Research indicates that rates of eating pathology and associated risk factors may vary across ethnicity (Cachelin, Rebeck, Chung, & Pelayo, 2002; Field, Camargo, Taylor, Berkey, & Colditz, 1999; Gray, Ford, & Kelly, 1987; McKnight Risk Factor Study, 2000; Powell & Kahn, 1995; Wildes, Emery, & Simons, 2001; Winkleby, Gardner, & Taylor, 1996), and accruing evidence suggests that ethnicity may moderate relationships between established risk factors and engagement in disordered eating (Rancourt, Schaefer, Bosson, & Thompson, 2016; Schaefer, Thibodaux, Krenik, Arnold, & Thompson, 2015). Further, available research indicates that levels of self-objectification may differ among ethnic groups (Breitkopft, Littleton, & Berenson, 2007). In light of these findings, it is possible that ethnicity may moderate the relationship between self-objectification and disordered eating.

Objectification theory rests on the assumption that women are viewed as sexual objects for male pleasure. Thus, researchers have suggested that the tenets of objectification theory may apply primarily to heterosexual women who may be more likely to self-objectify in order to garner male attention (Tiggemann, 2011). Investigators have recently begun to examine the role of sexual orientation in self-objectification processes and disordered eating, with findings suggesting that the impact of sexual orientation may differ for men and women (Engeln-Maddox, Miller, & Doyle, 2011; Haines et al., 2008; Kozee and Tylka, 2006). Studies using male samples indicate that homosexual men evidence higher levels of self-objectification (Engeln-Maddox et al., 2011; Kozak, Frankenhauser, & Roberts, 2009) and disordered eating (Boroughs & Thompson, 2002; Martins, Tiggemann, & Kirkbride, 2007) compared to heterosexual men, while studies using female samples generally indicate comparable levels of self-objectification (Lyders, 1999) and disordered eating (Brand, Rothblum, & Solomon, 1992; Striegel-Moore, Tucker, & Hsu, 1990) among homosexual and heterosexual women. Limited research has examined the influence of sexual orientation on the relationships between self-objectification and disordered eating (Lyders, 1999; Martins et al., 2007), however findings suggest that sexual orientation may moderate the relation between self-objectification and disordered eating, especially among males.

Elevated body mass index (BMI) has been shown to predict increases in several risk factors for disordered eating, as well as the onset of bulimic pathology, binge eating, and eating disorder symptoms (Killen et al., 1994; Stice, Presnell, & Spangler, 2002; Vogeltanz-Holm et al., 2000; Wichstrom, 2000). Moreover, BMI has been shown to moderate the relations between several putative risk factors and disordered eating (Rukavina & Pkrajac-Bulian, 2006). As self-objectification is a theorized risk factor for disordered eating, it is possible that BMI may similarly moderate the association between self-objectification and disordered eating such that higher BMI will be associated with a stronger relationship between self-objectification and disordered eating.

Research has demonstrated cross-cultural differences in levels of established risk factors for eating disorders, such as internalization of appearance ideals and appearance related pressures, as well as differences in the strength of associations between putative risk factors and disordered eating (Schaefer et al., 2013). Western appearance ideals emphasize the importance thinness for women, and research suggests that disordered eating behaviors such as binging and purging may be culturally-bound, appearing largely within cultures exposed to Western beauty ideals (Keel & Klump, 2003). Although the majority of the work examining objectification theory has been conducted utilizing samples from the United States, Australia, and Britain (Calogero, 2009; Calogero & Thompson, 2009; Moradi & Huang, 2008), recent work suggests that levels of self-objectification may vary by country (Loughnan et al., 2015). Similar to other environmental risk factors, it is possible that country of origin (here, used as a proxy for one's cultural context) may also moderate the relation between self-objectification and disordered eating such that a stronger association will be observed among samples from Western countries compared with non-Western countries.

Numerous studies conducted within non-clinical samples suggest a relationship between self-objectification and disordered eating (Lindner et al., 2012; Noll & Fredrickson, 1998;

Tiggemann & Slater; 2001). The presence of significant psychopathology, particularly eating pathology, is likely to increase the strength of the association between self-objectification and disordered eating. Therefore, clinical samples comprised of individuals with diagnosed eating disorders may yield stronger effect sizes compared with non-clinical samples. Similarly, as college women have been found to exhibit heightened levels of disordered eating (Hesse-Biber, Marino, & Watts-Roy, 1999; Pyle, Neuman, Halvorson, & Mitchell, 1991), it is possible that the association between self-objectification and disordered eating may be stronger among this group compared to community samples.

Currently, there are two validated and widely-used measures of self-objectification: the Self-Objectification Questionnaire (SOQ; Noll & Fredrickson, 1998) and the Body Surveillance subscale of the Objectified Body Consciousness Scale (OBCS-BS; McKinley & Hyde, 1996). The SOQ assesses the degree to which respondents value observable physical attributes (e.g., physical attractiveness) over non-observable competence-based physical attributes (e.g., physical coordination). The OBCS Body Surveillance subscale assesses the degree to which the respondent engages in habitual monitoring of his or her appearance. Researchers have suggested that "valuing the body as a physical object [as measured by the SOQ] and behaviorally investing in the body as a physical object [as measured by the OBCS] are not the same phenomenon" (Calogero, 2011). Indeed, several studies have demonstrated that self-objectification measured by the SOQ and self-surveillance measured by the OBCS are conceptually and empirically distinguishable concepts that produce different patterns of relations with disordered eating (Greenleaf & McGreer, 2006; Hill & Fischer, 2008; Kozee & Tylka, 2006; Steer & Tiggemann, 2008; Tiggemann & Slater, 2001). Therefore, it is likely that the measure of objectification will moderate the relation between self-objectification and disordered eating.

The Diagnostic and Statistical Manual of Mental Disorders (*DSM-5*; American Psychiatric Association, 2013) currently recognizes three eating disorders with distinct patterns of eating behavior. Anorexia nervosa is characterized by extreme dietary restriction resulting in significantly below average weight. Bulimia nervosa is characterized by recurrent episodes of binge eating followed by compensatory behavior aimed at preventing weight gain (e.g., use of vomiting or laxatives). Finally, binge-eating disorder is characterized by binge eating without compensatory behaviors. Researchers have identified common and unique risk factors associated with these various expressions of disordered eating (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). Therefore, it is possible that measures assessing anorectic, bulimic, binge eating, or global symptoms of eating pathology may exhibit different patterns of relations with self-objectification.

Demonstrated biases towards publishing manuscripts that report significant results (Dickersin, 2005; Easterbrook, Berlin, Gopalan, & Matthews, 1991) may result in differences in effect sizes between unpublished work (e.g., unpublished manuscripts, dissertations, and theses) and published manuscripts. It is therefore possible that published studies demonstrate stronger associations than unpublished studies.

The Current Study

A number of excellent narrative reviews highlight the wealth of evidence linking self-objectification to disordered eating (e.g., Moradi & Huang, 2008; Tiggemann, 2013). However, these findings have not yet been synthesized through a quantitative review. Meta-analysis provides a powerful means of synthesizing results from a series of studies on a given topic. This approach allows researchers to estimate the strength of the relationship between two variables in the population, to examine hypothesized moderators of that association, and to identify research questions that require further attention (Borenstein, Hedges, Higgins, & Rothstein, 2009). Therefore, the primary goal of the current study was to examine the association between self-objectification and disordered eating using meta-analytic techniques. A secondary goal of the study was to examine demographic and methodological characteristics that may modulate this association.

Method

Locating Studies

A literature search was conducted using PsycINFO and ProQuest Dissertations and Theses Online to identify relevant studies that examined the relationship between self-objectification and disordered eating. Search terms included *self-objectification*, *self-surveillance*, *Self-Objectification Questionnaire*, *Objectified Body Consciousness Scale* and *eating disorder**. Reference sections of retained studies were also reviewed to identify additional articles for inclusion. Search returns were screened to eliminate duplicate studies. Next the full text of each study was evaluated to determine eligibility for inclusion in the meta-analysis. Studies were eligible for inclusion in the analysis if they met the following criteria: (1) included a validated measure of self-objectification (i.e., Self-Objectification Questionnaire or Objectified Body Consciousness Scale – Body Surveillance subscale), (2) included a validated measure of disordered eating, and (3) were written in English, although country of origin was not restricted. The current meta-analysis focused exclusively on cross-sectional studies given methodological difficulties associated with assessing disordered eating in laboratory settings, significant variation in the methods used to induce self-objectification, and the limited number of experimental or longitudinal studies available. Finally, all studies were examined for the inclusion of sufficient data to calculate an effect size. If sufficient data was not available from either the text or the corresponding author, the study was excluded from the meta-analysis. Figure 1 provides a flow diagram of the study selection process based on the PRISMA guidelines.

Coding of Study Variables

Each study was coded for the following information: (1) sample size, (2) gender, (3) mean age of sample, (4) primary ethnic group, (5) primary sexual orientation of sample, (6) mean BMI of sample, (7) country of origin, (8) sample type, (9) self-objectification measure, (10) measurement of disordered eating, (11) publication, and (12) effect size (*r*). All studies were double-coded by the primary author and a trained research assistant, allowing for a 100% reliability check on the data. Inter-rater agreement was assessed with the kappa coefficient for categorical variables and with the intraclass correlation coefficient for continuous

variables. Instances where raters did not agree were resolved through discussion and consensus.

As outlined above, several demographic variables were coded for each study. For gender, studies were coded to indicate inclusion of all males, all females, or both genders. If both genders were included in a single study, effect sizes were considered separately for each gender if they were reported separately in the study. If the study included both genders and did not analyze results separately for each gender, the study was coded as including both genders. Age was considered as a continuous variable and the mean age of the sample was coded for each study. Ethnicity was considered as a categorical variable and each study was coded to reflect the dominant ethnic group represented in the sample. Studies were also coded to reflect the dominant sexual orientation of participants in the sample (i.e., heterosexual male/female, homosexual male/female, or mixed/unknown sexual orientation). BMI was considered as a continuous variable and mean sample BMI was recorded for each study. Country of origin was coded to assess the impact of regional/cultural differences on the association between variables of interest.

With regard to methodological moderators, the population from which the sample was drawn was recorded. Specifically, studies were coded as including samples from community, college, or clinical settings. If a study included groups drawn from different populations (e.g., community and clinical), results from each sample were coded separately if possible. All studies were coded to indicate the self-objectification measure utilized. When studies used both the Self-Objectification Questionnaire and Objectified Body Consciousness Scale – Body Surveillance subscale, both effect sizes were recorded and the average effect size calculated for inclusion in the meta-analysis. Consistent with previous meta-analyses examining correlates of disordered eating (Menzel et al., 2010; Stice, 2002), the eating disorder measures utilized were coded to indicate a focus on anorectic symptoms (e.g., Eating Disorder Inventory – Drive for Thinness subscale), bulimic symptoms (e.g., Eating Disorder Inventory – Bulimia subscale), binge eating symptoms (e.g., Binge Eating Scale), or a composite variable representing overall eating disorder symptomatology (e.g., Eating Disorder Examination Questionnaire – Global Scale). When multiple measures were utilized, the effect sizes were averaged for the calculation of the summary effect. Finally, studies were coded to reflect publication type. Both published studies and unpublished theses/dissertations were included in the analysis, however, if any portion of the thesis/ dissertation was published, only the published article was used.

Calculation of Summary Effect Size

As each study included in the meta-analysis was correlational, the Pearson correlation coefficient (r) was used as the index of effect size. If the effect size was not included in the article or could not be calculated from the data presented, authors were contacted in order to obtain the relevant information. If a study yielded more than one independent effect size (e.g., effect size for women and men), these studies were coded separately. Computation of the summary effect size utilized the meta-analytic approach outlined by Hedges and Olkin (1985). Independent effect sizes were converted to z using Fisher's r to z transformation (Fisher, 1970) and weighted by their inverse variance. The summary effect was calculated

using a random-effects model. The random effects model was chosen for two reasons: (1) It was expected that the true effect size would vary across studies due to differences in sample characteristics, and (2) a random effects model would allow the findings of the current meta-analysis to be generalized beyond those studies that were included in the analysis (Borenstein, Hedges, Higgins, & Rothstein, 2009). The obtained summary effect was then transformed back from z to r for interpretation and reporting. According to guidelines set forth by Cohen (1988), effect sizes of .10, .30, and .50 are considered small, medium, and large, respectively. Effect sizes smaller than .05 were considered trivial. All analyses were completed using Comprehensive Meta-Analysis, Version 2.0 (Borenstein, Hedges, Higgins, & Rothstein, 2005).

Moderator Analyses

As discussed, the random-effects model for computing the overall summary effect size presumes that the true effect size may vary from study to study. Between study variation in effect sizes arises from two sources: 1) differences in the true effect size and 2) random error. In meta-analysis, Cochran's heterogeneity statistic Q is used to quantify the amount of between study variation (Cochran, 1954). A statistically significant Q statistic suggests that there is significant heterogeneity in the observed study effect sizes. In other words, the between study variation in effect sizes is greater than one would be expected based on random error alone.

Moderator analyses are conducted to identify sources of between study variation. Moderator analyses for categorical variables were conducted using analog to ANOVA. A mixed effects model, which is generally recommended and allows for some true variation in effects within a subgroup, was utilized (Borenstein, Hedges, Higgins, & Rothstein, 2009). Similar to ANOVA within a single primary analysis, analog to ANOVA analyses provide information regarding both within group variance (Q_{within}) and between group variance (Q_{btwn}). A significant Q_{btwn} suggests that there are significant differences in the true effect size between groups.

Continuous moderators were examined using meta-regression. A random-effects model, which is recommended when it is likely that the impact of the examined moderator captures some but not all of the true variation among effects, was used to examine the impact of the moderator on the relationship between self-objectification and disordered eating. When meta-regression is performed, significant moderation is indicated by a significant slope, similar to regression in a primary study.

Publication Bias

As discussed, research suggests that studies reporting larger effect sizes and/or significant results are more likely to be published compared with studies that report smaller effect sizes and/or non-significant results (Dickersin, 2005). This bias in the published literature is likely to be reflected in meta-analytic procedures, which primarily utilize published data. In the current study, publication bias was assessed via Orwin's (1983) *Fail-safe N* analysis, which determines the number of studies with an effect size of zero that would be needed to reduce the summary effect size to a trivial amount (i.e., $r = .05$). Additionally, the funnel plot, which

presents a visual display of the relationship between effect size and standard error, was also used to assess publication bias. Funnel plots have a symmetrical funnel shape with equal numbers of studies falling above and below the obtained summary effect when publication bias is not present. A lack of symmetry in the plot suggests publication bias. Duval and Tweedie's trim and fill procedure (2000a; 2000b)can be used to correct this bias by imputing "missing" studies and recalculating a new unbiased summary effect.

Results

Fifty-three manuscripts yielded 73 independent effect sizes (N = 15,217). Appendix A presents the effect sizes (r) and study characteristics for each study examined in the current analysis. Inter-rater reliability was good to excellent for all study variables with coefficients ranging from .84 to 1.00. Figure 2 presents the forest plot of the correlations between self-objectification and disordered eating.

Summary Effect

The overall summary effect was r = 0.39, z = 23.78, p < .001 (95% confidence interval [CI]: .36 to .42). According to Cohen's (1988) guidelines, this exceeds the convention for medium effect sizes (.30). The effect was marked by significant heterogeneity, $Q(72)$ = 274.75, p < .001, suggesting the presence of potential moderating factors.

Gender

Sixty-three effect sizes reflected the relationship between self-objectification and disordered eating in females. Ten effect sizes reflected the relationship between self-objectification and disordered eating in males. There were no studies that examined the relationship between self-objectification and disordered eating in mixed gender samples. The point estimate for females was r = 0.41, z = 25.73, p < .001 (95% CI: .38 to .44). The point estimate for males was r = 0.20, z = 4.57, p < .001 (95% CI: .11 to .28). Differences between groups were significant, $Q_{btwn}(1)$ = 46.94, p < .001, suggesting that the relationship between self-objectification and disordered eating is stronger among women.

Age

Sixty-five effect sizes were included in the analysis to examine mean age as a potential moderator of the relationship between self-objectification and disordered eating. Eight studies were excluded from the analysis as they did not report a mean sample age. Results revealed that mean age was not a significant moderator, slope = -.002, SE_{slope} = .001, p = .200. In light of research suggesting that disordered eating and self-objectification may increase through adolescence and decrease in adulthood (Heatherton et al., 1997; Tiggemann & Lynch, 2001), the scatterplot was examined for evidence of a possible u-shaped or non-linear relationship between age and effect size. Visual inspection of the scatterplot did not support a non-linear relationship.

Ethnicity

Many studies (k = 17) did not report sample demographics related to race/ethnicity and were therefore excluded from relevant moderator analyses. Among effect sizes for which race/

ethnicity data was provided (k = 55), 52 effect sizes were computed using primarily Caucasian participants, two effect sizes were computed using primarily African American/ Black participants, and one effect size was computed using Asian American participants. When all genders were included, the point estimate for studies utilizing primarily Caucasian samples was r = 0.41, z = 21.40, p < .001 (95% CI: .38 to .45). The point estimate for studies utilizing primarily African American samples was r = 0.34, z = 11.92, p < .001 (95% CI: .29 to .39). The observed effect for the single study utilizing a primarily Asian American sample was 0.42, z = 5.14, p < .001 (95% CI: .27 to .55). Differences between groups were marginally significant, $Q_{btwn}(2)$ = 5.21, p = 0.074. Given research suggesting that ethnic differences in disordered eating may vary across genders (Croll, Neumark-Stzainer, Story, & Ireland, 2002; Field, Camargo, Taylor, Berkey, & Colditz, 1999; Gray, Ford, & Kelly, 1987; Kelly, Cottern, Tanofsky-Kraff, & Mazzea, 2015; Wildes, Emery, & Simons, 2001), the impact of ethnicity was also examined among males and females separately. When analyses were restricted to include only female samples, ethnicity significantly moderated the association between disordered eating and self-objectification, $Q_{btwn}(2)$ = 6.348, p = 0.039. The point estimate for Caucasian females was r = 0.42, z = 21.96, p < .001 (95% CI: .39 to .45). The point estimate for Asian American females was r = 0.42, z = 5.14, p < .001 (95% CI: .27 to .55). The point estimate for African American females was r = 0.34, z = 11.91, p < .001 (95% CI: .29 to .39). All male samples were primarily Caucasian and therefore moderator analyses were not conducted to examine the impact of ethnicity among men. Results suggest that the relationship between self-objectification and disordered eating is moderated by ethnicity for women. In particular, the relationship was strongest among primarily Caucasian and Asian American samples of women and weakest among primarily African American samples of women.

Sexual Orientation

The majority of studies (k = 58) did not report sample demographics related to sexual orientation and were therefore excluded from relevant moderator analyses. Seven studies utilized samples primarily comprised of heterosexual females, three primarily utilized heterosexual males, two primarily utilized homosexual females, and three studies utilized samples primarily comprised of homosexual males. The point estimate for heterosexual females was r = 0.39, z = 11.30, p < .001 (95% CI: .33 to .45). The point estimate for heterosexual males was r = 0.23, z = 3.51, p < .001 (95% CI: .10 to .35). The point estimate for homosexual females was r = 0.38, z = 5.03, p < .001 (95% CI: .24 to .50). The point estimate for homosexual males was r = 0.32, z = 4.65, p < .001 (95% CI: .19 to .44). Differences between groups were significant, $Q_{btwn}(3)$ = 7.91, p = 0.048, suggesting that the relationship between self-objectification and disordered eating is moderated by sexual orientation. In particular, the relationship appears to be strongest among heterosexual women and weakest among heterosexual males.

Body Mass Index

Forty-eight effect sizes were included in the analysis to examine mean BMI as a potential moderator of the relationship between self-objectification and disordered eating. Twenty-four studies were excluded from the analysis as they did not report a mean sample BMI.

Results reveal that mean BMI was not a significant moderator, slope = −0.01, SE_{slope} = .001, p = .483.

Country of Origin

As all studies reported information regarding the country of origin, all effect sizes (k = 73) were included in the relevant moderator analyses. Forty-four studies utilized samples from the United States, fifteen utilized samples from Australia, seven utilized samples from England, two studies utilized samples from Ireland, two studies utilized samples from Canada, and one study utilized a sample drawn from both the United States and Canada. The point estimate for the United States was r = 0.41, z = 20.62, p < .001 (95% CI: .37 to .44). The point estimate for Australia was r = 0.36, z = 10.41, p < .001 (95% CI: .30 to .42). The point estimate for England was r = 0.27, z = 4.30, p < .001 (95% CI: .15 to .38). The point estimate for Ireland was r = 0.46, z = 7.14, p < .001 (95% CI: .34 to .56). The point estimate for Canada was r = 0.35, z = 3.50, p < .000 (95% CI: .16 to .52). The observed effect size for the combined United States and Canada sample was r = 0.40, z = 7.92, p < .001 (95% CI: . 31 to .49). Differences between groups were not significant, $Q_{btwn}(5)$ = 8.46, p = 0.132, suggesting that the relationship between self-objectification and disordered eating did not differ among the countries assessed in this analysis.

Sample Type

All studies reported information regarding the type of sample utilized and therefore all studies (k = 73) were included in the relevant moderator analyses. Forty-one studies utilized college samples, twenty utilized community samples, nine utilized samples drawn from both college and community populations, two studies utilized samples drawn from eating disorder clinical populations, and one study utilized a sample drawn from a general clinical population. The point estimate for college samples was r = 0.40, z = 14.38, p < .001 (95% CI: .36 to .43). The point estimate for community samples was r = 0.35, z = 14.01 p < .001 (95% CI: .31 to .40). The point estimate for college/community samples was r = 0.39, z = 6.46, p < .001 (95% CI: .28 to .49). The point estimate for eating disorder clinical samples was r = 0.35, z = 6.18, p < .001 (95% CI: .25 to .45). The observed for the general clinical sample was r = 0.57, z = 5.39, p < .000 (95% CI: .39 to .70). Differences between groups were not significant, $Q_{btwn}(4)$ = 6.41, p = 0.171, suggesting that the relationship between self-objectification and disordered eating did not differ among college, community, and clinical samples.

Measurement of Self-Objectification

All studies (k = 73) reported information regarding the measure used to assess objectification. Twenty-five studies utilized the SOQ exclusively, while 24 studies utilized the OBCS-BS exclusively. Twenty-four studies utilized both the OBCS-BS and SOQ yielding an average composite effect. One such study created an objectification composite score and provided the correlation with measures of disordered eating; the remaining 23 studies provided correlations between the individual measures of self-objectification and disordered eating, which were averaged to provide a composite effect. The point estimate for studies using the OBCS-BS was r = 0.48, z = 18.21, p < .001 (95% CI: .43 to .52). The point estimate for studies using the SOQ was r = 0.34, z = 17.99 p < .001 (95% CI: .30 to .37).

The observed effect for the study that utilized a composite of both the OBCS-BS and SOQ was $r = 0.49$, $z = 9.66$, $p < .001$ (95% CI: .40 to .57). Finally, the point estimate for studies in which both the OBCS-BS and SOQ were utilized and the resultant effects averaged for use in the current meta-analysis was $r = 0.34$, $z = 12.01$, $p < .001$ (95% CI: .29 to .40). In order to assess the moderating influence of self-objectification measure, studies that utilized multiple measures were removed from moderator analyses. There was a significant difference in effect sizes obtained from studies utilizing the OBCS-BS compared with studies utilizing the SOQ, $Q_{btwn}(1) = 22.48$, $p < 0.001$. Specifically, larger effect sizes were observed among studies utilizing the OBCS-BS, while smaller effect sizes were observed among studies utilizing the SOQ.

Measurement of Disordered Eating

All studies ($k = 73$) were included in the moderator analyses to examine possible differences in effect size across type of eating disorder symptoms. Twelve studies assessed anorectic/restrictive symptoms, two studies assessed bulimic symptoms, and 59 studies assessed global eating disorder symptoms (i.e., restrictive behaviors, binge/purge behaviors, and body image concerns). The point estimate for studies assessing anorectic/restrictive symptoms was $r = 0.37$, $z = 8.96$, $p < .001$ (95% CI: .29 to .44). The point estimate for studies assessing bulimic symptoms was $r = 0.33$, $z = 2.65$, $p = .008$ (95% CI: .09 to .53). The point estimate for studies assessing global eating disorder symptoms was $r = 0.39$, $z = 21.67$, $p = .008$ (95% CI: .36 to .42). There was not a significant difference among effect sizes observed in studies assessing anorectic/restrictive, bulimic, or global eating disorder symptoms $Q_{btwn}(2) = 0.64$, $p = 0.726$.

Publication Type

All studies ($k = 73$) were included in the moderator analyses to assess the effect of publication type on the relationship between self-objectification and disordered eating. Fifty-eight journal articles and 15 dissertations were examined. The point estimate for journal articles was $r = 0.39$, $z = 20.03$, $p < .001$ (95% CI: .35 to .42). The point estimate for dissertations was $r = 0.38$, $z = 12.74$ $p < .001$ (95% CI: .33 to .43). Effect sizes extracted from journal articles were not significantly different from those extracted from dissertations $Q_{btwn}(1) = 0.07$, $p = 0.793$.

Publication Bias

Orwin's *Fail-safe N* analysis indicated that 547 additional studies with a mean effect of zero would be needed to reduce the summary effect to a trivial effect size (i.e., $r = .05$). Examination of the funnel plot (Figure 3) indicates that the effect sizes were largely symmetrically distributed around the summary effect. Duval and Tweedie's trim and fill procedure did not identify any "missing" studies. Therefore, the adjusted or unbiased summary effect computed using a random effects model was equivalent to the observed summary effect.

Discussion

Objectification theory implicates self-objectification in the etiology of disordered eating and numerous studies have sought to examine the relationship between these constructs empirically. Results from the current meta-analysis suggest a positive, moderate, and significant (r = .39) bivariate relationship such that greater objectification of one's body is related to higher levels of disordered eating attitudes and behaviors. Importantly, the magnitude of this effect is similar to effect sizes observed in meta-analyses examining other established risk factors and correlates of disordered eating, including appearance comparisons (Meyers & Crowther, 2009), weight teasing (Menzel et al., 2010), perceived pressures for thinness, and thin ideal internalization (Stice, 2002).

Moderators of the Relationship Between Self-Objectification and Disordered Eating

The overall summary effect was characterized by significant heterogeneity. Subsequent analyses indicated that several moderator variables including gender, ethnicity, sexual orientation, and the measurement of self-objectification may partially account for this heterogeneity. Mean sample age, mean sample BMI, country of origin, sample type, measurement of disordered eating, and publication type were not significant moderators.

As expected, there was a stronger relationship between self-objectification and disordered eating for women compared with men. These results may not be surprising in light of research indicating that women are more consistently objectified in media, report more objectifying experiences and self-objectification, more frequently feel evaluated solely based on their physical appearance, and experience a greater impact when exposed to objectifying messages (Archer, Iritani, Kimes, & Barrios, 1983; Bryant 1993; Fredrickson & Roberts, 1997; Klonoff, 2000; Kozee, Tylka, Augustus-Horvath, & Denchik, 2007; Murnen & Smolak, 2000; Swim, Hyers, Cohen, & Ferguson, 2001; Unger & Crawford, 1996). Indeed, objectification theory derives from a feminist framework that attempts to explain how women's gendered experiences contribute to their elevated rates of disordered eating. Importantly, although considerably fewer studies examined the relationships of interest in male samples, the cumulative results suggest the potential role of self-objectification in disordered eating among men as well as women. In particular, moderator analyses suggest that this association may be especially strong for homosexual males (further discussed below). The current analysis highlights the importance of self-objectification processes in disordered eating among women, the potential role of these processes among men, and the need for further work among males.

Although previous meta-analyses examining the relationship between proposed risk factors (e.g., appearance comparisons, exposure to thin media images, weight-related teasing) and disordered eating have found a moderating influence of age, such that effect sizes are larger among younger samples (Groesz, Levine, & Murnen, 2002; Menzel et al, 2010; Myers & Crowther, 2009), age was not a significant moderator in the current study. While the range of mean sample age was somewhat limited (lowest mean sample age: 13.40, highest mean sample age: 48.95), results suggest that self-objectification is as strongly related to disordered eating among adolescents as it is among young and middle-aged adults. Limited research among adult women aged 18 to 84 has found that levels of self-objectification and

disordered eating decrease with age, and that self-objectification may mediate the relationship between age and disordered eating (Tiggemann & Lynch, 2001). This finding, paired with the current meta-analytic results, suggest that changes in self-objectification may play an important role in the etiology, maintenance, and remittance of disordered eating across age groups, though longitudinal work is needed to examine these prospective associations. Additional work utilizing adolescent and older adult samples is also encouraged.

Ethnicity emerged as a marginally significant moderator when male and female samples were considered together, with primarily Caucasian and Asian American samples evidencing the largest effect sizes and African American samples evidencing smaller effect sizes. When the moderating influence of ethnicity was examined among female samples alone, ethnicity significantly moderated the association between self-objectification and disordered eating. Again, Caucasian and Asian American women evidenced the largest effect sizes, with African American women evidencing smaller effect sizes. As all male samples were primarily Caucasian, moderator analyses were not conducted for males. Importantly, very few studies ($k = 3$) were conducted within primarily non-Caucasian (i.e., African American and Asian American) samples, and samples representing additional ethnic groups (e.g., Hispanic) were not available. Therefore, results must be interpreted in light of these significant limitations. On a methodological level, many studies did not report information regarding race/ethnicity. As research increasingly suggests the importance of examining ethnicity in the etiology, maintenance, and treatment of disordered eating (Bucchianeri, Fernandes, Loth, Hannan, Eisenberg, & Neumark-Sztainer, 2015; Kelly et al., 2015), inclusion of information regarding the ethnic composition of the sample is encouraged. Overall, findings indicate a possible influence of ethnicity and highlight the need for further work examining the constructs of interest in ethnic minority groups.

Sexual orientation significantly moderated the relationship between self-objectification and disordered eating. Point estimates were highest for heterosexual and homosexual women, followed by homosexual males. Effect sizes were weakest among heterosexual males. Previous research suggests that heterosexual and homosexual women report comparable levels of disordered eating and results from the current study suggest that that self-objectification may play an equally important role in understanding the etiology and maintenance of maladaptive eating patterns in these groups. Interestingly, results among males suggest that self-objectification may play a larger role in disordered eating attitudes and behaviors for homosexual men compared with heterosexual men. Consistent with Objectification Theory's assertion that "male gaze" contributes to experiences of sexual and self-objectification among women (Fredrickson & Roberts, 1997), it is possible that this same phenomenon similarly contributes to objectification experiences among homosexual men. As homosexual men evidence higher levels of both disordered eating and self-objectification compared to heterosexual men (Boroughs & Thompson, 2002; Engeln-Maddox et al., 2011; Kozak et al., 2009; Tiggemann et al., 2007), prevention and treatment interventions aimed at this population may consider self-objectification as a particularly relevant treatment target. Results also suggest the importance of reporting such demographic information in research related to the constructs of interest.

BMI did not moderate the association between self-objectification and disordered eating, suggesting that a tendency to self-objectify and engage in habitual body monitoring is moderately related to disordered eating across the BMI spectrum. Notably, the majority of studies reported mean BMIs in the normal range (18.5 to 25.0) and only one study utilized a sample comprised of obese individuals, restricting the range of BMI examined in the current analysis. As elevated BMI is associated with disordered eating and its risk factors (Killen et al., 1994; Stice et al., 2002; Vogeltanz-Holm et al., 2000; Wichstrom, 2000), further evaluation in individuals with overweight and obesity may be warranted.

Country of origin was not a significant moderator in the current study. Notably, however, all studies were conducted with samples drawn from Western industrialized countries (i.e., United States, Australia, England, Ireland, Canada) where rates of disordered eating and associated risk factors (e.g., emphasis on a thin body ideal) are relatively homogeneous (Mautner, Owen, & Furnham, 2000; Swami et al., 2010). As research indicates that rates of disordered eating and associated risk factors are typically higher in Western compared with Eastern societies (Keel & Klump, 2003; Miller & Pumariega, 2001), it is possible that effect sizes may differ in non-Western samples. Therefore, examination of the relationship between self-objectification and disordered eating in non-Western cultures is an area for future investigation.

Sample type was also not a significant moderator in the current analysis, suggesting that the relationship between self-objectification and disordered eating may be consistent across college, community, clinical eating disorder, and general clinical samples. It is therefore possible that self-objectification processes are equally impactful regardless of the presence of an eating disorder diagnosis. Alternatively, it is possible that results may be due to methodological artifact. Only two studies examined the constructs of interest within samples of individuals with diagnosed eating disorders. Both of these studies assessed self-objectification via the SOQ, which moderator analyses suggest is associated with smaller effect sizes. Among college, community, and general clinical samples, self-objectification was often assessed via the OBCS-BS, which may yield higher effect sizes. Future work with clinical samples may seek to include the OBCS-BS in order to capture the self-monitoring aspect of self-objectification, which appears to be more closely associated with disordered eating.

Consistent with researchers' assertions that an objectifying orientation towards the self and habitual body monitoring represent distinct constructs (Calogero, 2011), the OBCS-BS and SOQ produced significantly different mean effect sizes in the current meta-analysis. Specifically, the OBCS-BS was associated with larger effect sizes compared with the SOQ. These results suggest that the degree to which an individual engages in habitual monitoring of his or her appearance is more predictive of disordered eating than the degree to which an individual values his or her observable physical attributes over non-observable competence-based physical attributes. It is possible that the attitudinal aspect of self-objectification (as measured by the SOQ) developmentally precedes the behavioral manifestation of self-objectification (as measured by the OBCS-BS), such that habitual monitoring represents a more proximal etiological factor. Longitudinal data and meditational models are needed to examine this potential etiological model.

Previous meta-analytic work suggests that associations with some proposed risk factors may vary across anorectic, bulimic, and binge eating pathology (Stice, 2002), while other risk factors demonstrate consistent associations with distinct forms of eating pathology (Menzel et al., 2010). In the current study, measurement of disordered eating did not emerge as a significant moderator, suggesting that self-objectification is comparably related to disordered eating when assessed via measures of anorectic, bulimic, and global eating pathology. Notably, most effect sizes reflected general eating pathology symptoms either through the use of a global measure (e.g., the Eating Disorder Examination Questionnaire) or through the averaging of measures reflecting anorectic and bulimic symptoms (e.g., Eating Disorder Inventory – Drive for Thinness subscale and Eating Disorder Inventory – Bulimia subscale), which may have obscured possible differences in effect sizes. Future work should examine this possibility. Additionally, there were no studies that examined associations with measures of binge eating symptoms, highlighting a complementary area for future investigation. As binge eating disorder may be less closely associated with overvaluation of shape/weight and preoccupation with the body (core features of anorexia nervosa and bulimia nervosa) (American Psychiatric Association, 2013; Grilo et al., 2009) it is possible that measures of binge eating may produce weaker associations with self-objectification.

Given the tendency for studies with significant results to reach publication, while studies with non-significant results often go unpublished, moderator analyses examining publication type may serve as a signal of potential publication bias. Additionally, as journal articles undergo extensive peer-review prior to publication, publication type may also be viewed as a proxy for methodological rigor. In the current study, publication type was not a significant moderator – studies published in peer-reviewed journals and unpublished dissertations exhibited similar mean effect sizes. These results suggest an absence of publication bias. Further, formal publication bias analyses including *Fail-safe N* analyses, trim and fill procedures, and examination of the funnel plot suggest minimal publication bias in the obtained summary effect.

Limitations and Future Directions

There are several limitations to the current study. First, it is likely that variables not examined in the present study operate as moderators of the relationship between self-objectification and disordered eating. Candidate variables for future research include appearance importance, appearance ideal internalization, body dissatisfaction, relationship status, neurobiological factors, and genetic factors. Second, although the current study attempted to include numerous potential theoretical and methodological moderators, proposed analyses may have been underpowered for some variables (e.g., country of origin, measurement of disordered eating). Relatedly, often studies did not include important demographic information regarding participant ethnicity or sexual orientation. As both variables emerged as significant moderators, future studies should seek to collect and report these demographics, and to conduct further work in ethnic and sexual minority groups. Although country of origin did not significantly moderate the summary effect, it is notable that countries representing non-Western cultures were not available for inclusion in the current analysis. Future work should seek to examine this association in non-Western samples. Although measurement of disordered eating did not moderate the summary effect,

most studies assessed global eating disorder symptoms, which may have obscured potential differences, and no studies examined associations with symptoms of binge eating disorder. Further work may seek to examine potential differences in associations with anorectic, bulimic, and binge eating phenomena. An additional point to consider relates to the measurement of both disordered eating and self-objectification. The SOQ, OBCS and numerous measures of disordered eating were developed utilizing primarily Caucasian heterosexual female samples. It is therefore possible that these measures may more readily assess experiences of self-objectification and disordered eating most relevant to those groups. To date, no study has examined measurement invariance for the SOQ or OBCS among individuals of different genders, sexual orientation, or ethnic backgrounds. Therefore, caution should be exercised when interpreting findings using male, non-Caucasian, and non-heterosexual samples. Finally, the cross-sectional nature of all included studies precludes the ability to establish temporal precedence or causality. Therefore, we are not able to draw conclusions regarding the role of self-objectification as a casual risk or maintenance factor for disordered eating based solely on the current meta-analysis. However, a small body of work suggests that experimentally-induced changes in state self-objectification (manipulated by instructing participants to wear bulky versus revealing clothing in front of a mirror) may relate to decreased consumption of snack foods in a laboratory setting (Fredrickson, Roberts, Noll, Quinn, & Twenge, 1998; Martins et al., 2007), although this effect has not been consistently demonstrated (Hebl, King, & Lin, 2004). Future research should continue to examine the relationship between self-objectification and disordered eating using a range of methodologies including longitudinal and experimental designs. In particular, as objectification theory proposes that self-objectification serves as a risk factor for disordered eating, examination of the temporal relationships between these variables represents an important area of inquiry. Accordingly, prospective studies that examine the ability of baseline self-objectification levels to predict the emergence of disordered eating or growth in symptoms are recommended to examine the role of self-objectification as a risk factor for disordered eating. Additionally, longitudinal studies examining self-objectification as a predictor of symptom persistence versus remittance among initially ill individuals would provide information regarding the role of self-objectification as a possible maintenance factor for disordered eating. Finally, experimental intervention studies targeting self-objectification are needed to provide important information regarding potential causal relationships between self-objectification and disordered eating. Such work would also serve to clarify whether self-objectification represents a modifiable and meaningful treatment target within disordered eating interventions.

Clinical Implications

Eating disorders and disordered eating are multifactorial problems likely arising from a complex interplay between biological, psychological, and environmental variables. The current study indicates a moderate relationship between disordered eating and the psychological experience of self-objectification. Moreover, the strength of this association appears to be comparable to that of other well-established risk factors (e.g., pressures for thinness, thin ideal internalization). Further, a small body of work suggests that changes in self-objectification may be causally related to increases in restrained eating behaviors

(Fredrickson et al., 1998; Martins et al., 2007). Therefore, there is growing evidence to suggest that self-objectification may represent an important element to consider in work related to the conceptualization, treatment, and prevention of disordered eating (Tiggemann, 2013). We have suggested avenues for additional research examining the temporal and causal significance of self-objectification, as well its relevance to disordered eating interventions. Should self-objectification emerge as a causal risk or maintenance factor for disordered eating, existing interventions may seek to address both the cognitive and behavioral manifestations of this experience using empirically-supported techniques. For example cognitive restructuring and dissonance-based approaches may be used to challenge self-schemas that emphasize the importance of appearance over other self-attributes. Mindfulness-based approaches may be used to encourage attention to internal experiences and reduce negative appearance-based judgements regarding oneself and others (Tiggemann, 2013).

Conclusions

In sum, objectification theory posits that self-objectification acts as a risk factor in the development of disordered eating among women. Following the introduction of the theory in 1997, the association between self-objectification and disordered eating has drawn considerable research interest. The current meta-analysis represents the first quantitative review of the resulting 20 years of scientific inquiry. Results suggest a significant moderate positive correlation between self-objectification and disordered eating. Significant heterogeneity in observed effects may be at least partially explained by gender, sexual orientation, ethnicity, and measurement of self-objectification. Although the current study is limited by the use of cross-sectional data and therefore cannot explicitly identify self-objectification as a risk or maintenance factor (Stice, 2002), the results suggest that self-objectification processes (particularly high levels of body surveillance) are highly related to eating disordered attitudes and behaviors. Given this relationship, interventions aiming to reduce levels of self-objectification may also positively impact disordered eating (Menzel, 2013). Future work should continue to clarify the potential causal role of self-objectification in the development of disordered eating and further examine demographic and methodological moderators of this relationship.

Appendix A: Characteristics of all studies included in the meta-analysis

Author(s), year	Sample size	Sample gender	Mean age	Primary ethnic group	Primary sexual orientation	Mean BMI	C
Augustus-Horvath & Tylka, 2009							
18–24 y/o	329	Female	19.90	Caucasian	N/A	24.13	
25–68 y/o	330	Female	29.74	Caucasian	N/A	26.74	
Calogero, 2009							
Females	139	Female	N/A	Caucasian	N/A	N/A	

Author(s), year	Sample size	Sample gender	Mean age	Primary ethnic group	Primary sexual orientation	Mean BMI	C
Males	113	Male	N/A	Caucasian	N/A	N/A	
Calogero, 2004	104	Female	N/A	Caucasian	N/A	21.71	
Calogero, Davis, & Thompson, 2005	209	Female	22.90	Caucasian	N/A	19.70	
Calogero & Pina, 2011							
Study 1	225	Female	21.60	Caucasian	Heterosexual	21.92	
Study 2	80	Female	21.89	Caucasian	Heterosexual	22.01	
Calogero & Thompson, 2009							
Study 1	104	Female	18.63	Caucasian	N/A	21.71	
Study 2	111	Female	22.00	N/A	N/A	21.89	
Choma, Shove, Busseri, Sandava, & Hosker, 2009	104	Female	19.12	Caucasian	N/A	23.60	
Clarke, Murnen, & Smolak, 2010	98	Female	N/A	Caucasian	N/A	N/A	
Cottingham, 2008	161	Female	19.83	Caucasian	N/A	N/A	
Daubenmier, 2005							
Study 1	139	Female	37.16	Caucasian	N/A	N/A	
Study 2	133	Female	20.46	Asian	N/A	22.1	
Eshkevari, Rieger, Longo, Haggard, & Treasure, 2012							
Clinical	78	Female	23.50	N/A	N/A	18.50	
Healthy Control	61	Female	24.00	N/A	N/A	21.50	
Funk, 2008	202	Female	20.10	Caucasian	N/A	21.83	
Gianini, 2012	204	Female	19.44	Caucasian	N/A	23.55	
Greenleaf, 2005							
College	200	Female	20.96	Caucasian	N/A	22.98	
Community	194	Female	48.95	Caucasian	N/A	25.16	
Greenleaf & McGreer, 2006	185	Female	20.52	Caucasian	N/A	23.05	
Grupski, 2010	539	Female	20.43	Caucasian	Heterosexual	23.16	
Haines, Erchull, Liss, Turner, Nelson, Ramsey, & Hurt, 2008	126	Female	35.12	Caucasian	Homosexual	N/A	
Hallsworth, Wade, Tiggemann, 2005	83	Male	27.59	N/A	N/A	35.79	
Harrison & Fredrickson, 2003	374	Female	13.40	African American	N/A	N/A	
Kaplan, 2012	125	Female	19.86	Caucasian	N/A	22.78	
Kaptein, 2008	263	Female	21.40	Caucasian	N/A	22.53	
Kelly, Mitchell, Gow, Trace, Lydecker, Bair, & Mazzeo, 2012							
African American	741	Female	N/A	African American	N/A	N/A	
Caucasian	1467	Female	N/A	Caucasian	N/A	N/A	
Kittler, 2003	328	Female	18.70	Caucasian	N/A	22.10	

Author(s), year	Sample size	Sample gender	Mean age	Primary ethnic group	Primary sexual orientation	Mean BMI	C
Langdon & Petracca, 2010	77	Female	22.91	Caucasian	N/A	N/A	
Lindner, Tantleff-Dunn, & Jentsch, 2012	549	Female	19.78	Caucasian	Heterosexual	23.23	
Lyders, 1999							
Heterosexual female	40	Female	37.18	Caucasian	Heterosexual	24.57	
Heterosexual male	40	Male	37.00	Caucasian	Heterosexual	26.44	
Homosexual female	40	Female	35.43	Caucasian	Homosexual	25.75	
Homosexual male	40	Male	35.75	Caucasian	Homosexual	25.03	
Martins, Tiggemann, & Kirkbridge, 2007							
Heterosexual male	103	Male	27.59	N/A	Heterosexual	25.38	
Homosexual male	98	Male	27.61	N/A	Homosexual	24.54	
Mitchell, 2009	893	Female	19.09	Caucasian	N/A	23.96	
Moret, 1999	74	Female	28.04	Caucasian	N/A	N/A	
Morrison & Sheahan, 2009							
College	140	Female	20.42	N/A	N/A	21.93	
Community	76	Female	23.61	N/A	N/A	22.28	
Morry & Staska, 2001							
Females	89	Female	18.80	N/A	N/A	N/A	
Males	61	Male	19.80	N/A	N/A	N/A	
Muehlenkamp & Saris-Baglama, 2002	396	Female	19.50	Caucasian	N/A	N/A	
Mussap, 2009	189	Female	27.34	N/A	N/A	23.59	
Myers & Crowther, 2008	195	Female	19.60	Caucasian	N/A	23.30	
Noffsinger-Frazier, 2004	345	Female	30.52	Caucasian	Heterosexual	25.90	
Noll & Fredrickson, 1998							
Study 1	93	Female	18.80	Caucasian	N/A	21.40	
Study 2	111	Female	18.30	Caucasian	N/A	22.10	
Oehlhof, 2012							
Normal weight	212	Female	19.17	Caucasian	N/A	21.02	
Overweight	201	Female	19.71	Caucasian	N/A	30.69	
Peat & Muehlenkamp, 2011	214	Female	20.09	Caucasian	N/A	23.24	
Prichard & Tiggemann, 2008	571	Female	35.99	N/A	N/A	23.80	
Prichard & Tiggemann, 2005	157	Female	32.15	N/A	N/A	22.63	
Rolnik, Engeln-Maddox, & Miller, 2010	127	Female	18.14	Caucasian	N/A	N/A	
Serpa, 2005							
Heterosexual male	96	Male	33.97	Caucasian	Heterosexual	N/A	
Homosexual male	96	Male	37.45	Caucasian	Homosexual	N/A	

Author(s), year	Sample size	Sample gender	Mean age	Primary ethnic group	Primary sexual orientation	Mean BMI	C
Slater & Tiggemann, 2002							
Dancer	38	Female	14.50	Caucasian	N/A	18.60	
Non-dancer	45	Female	14.10	Caucasian	N/A	20.50	
Slater & Tiggemann, 2012	141	Female	14.45	Caucasian	N/A	N/A	
Smolak & Murnen, 2011							
Females	148	Female	N/A	Caucasian	N/A	N/A	
Males	76	Male	N/A	Caucasian	N/A	N/A	
Tiggemann & Kuring, 2004							
Females	171	Female	21.92	N/A	N/A	N/A	
Males	115	Male	22.43	N/A	N/A	N/A	
Tiggemann & Lynch, 2001	332	Female	45.02	N/A	N/A	25.37	
Tiggemann & Slater, 2001							
Dancer	50	Female	20.30	N/A	N/A	21.50	
Non-dancer	51	Female	19.40	N/A	N/A	22.70	
Tiggemann & Williams, 2012	146	Female	20.40	Caucasian	N/A	23.61	
Tylka & Hill, 2004	460	Female	21.40	Caucasian	N/A	N/A	
Van Diest & Perez, 2013	177	Female	19.25	Caucasian	N/A	21.28	
Woolley, 2009	395	Female	N/A	Caucasian	Heterosexual	N/A	

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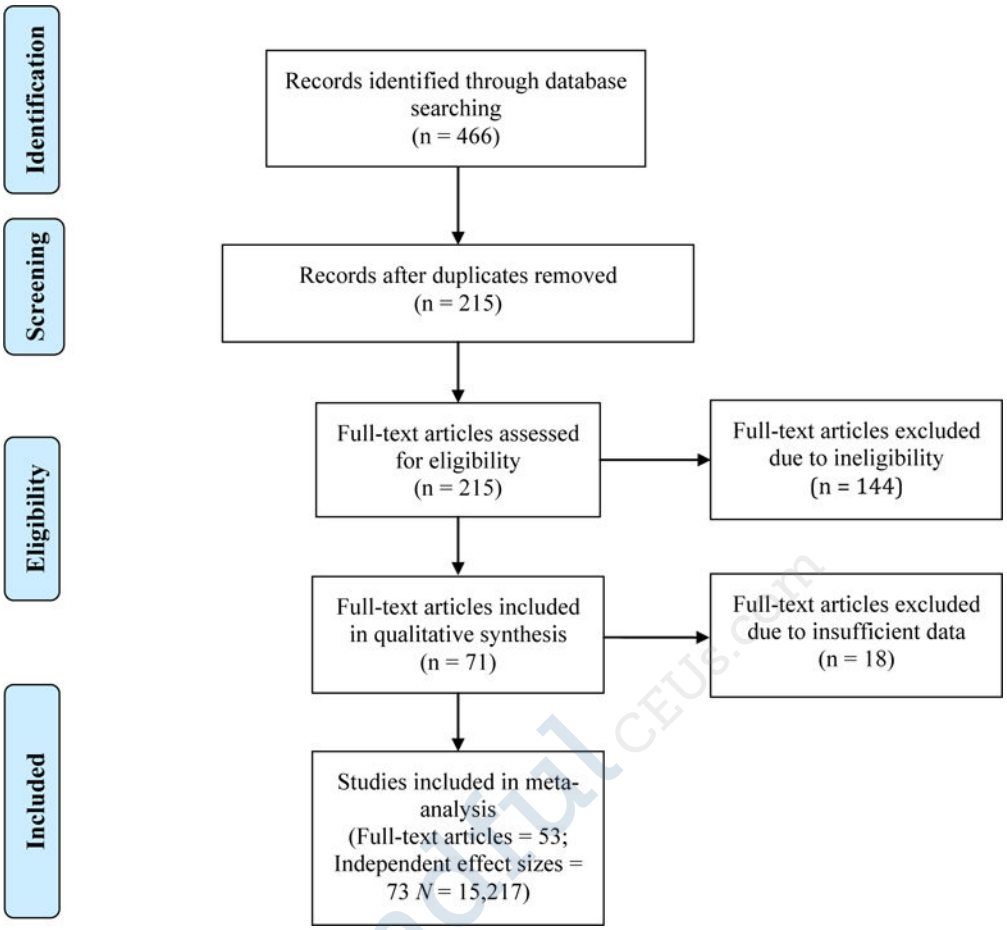

Figure 1.
Flow Diagram of Study Selection

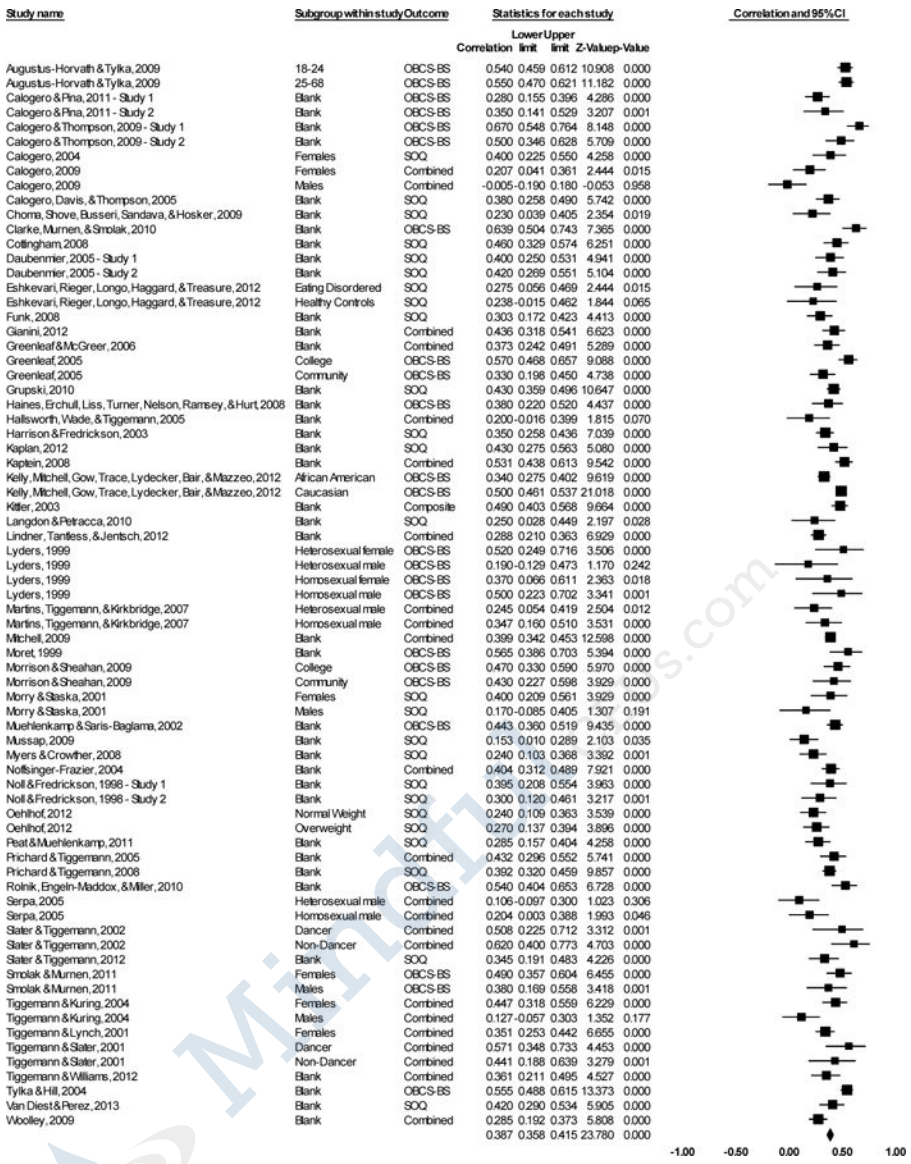

Figure 2.
Forest plot of the correlations between self-objectification and disordered eating

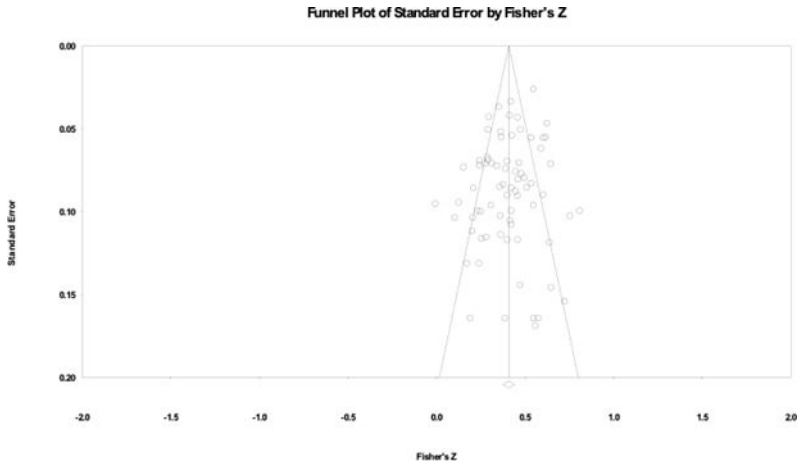

Figure 3.
Funnel plot of observed effects

What Drives the Association between Weight Conscious Peer Groups and Disordered Eating? Disentangling Genetic and Environmental Selection from Pure Socialization Effects

Abstract

Previous studies suggest strong associations between exposure to weight conscious peer groups and increased levels of disordered eating. This association has been attributed to socialization effects (i.e., membership leads to disordered eating); however, selection effects (i.e., selecting into peer groups based on genetic and/or environmental predispositions toward disordered eating) could contribute to or even account for these associations. The current study was the first to use a co-twin control design to disentangle these types of selection factors from socialization effects. Participants included 610 female twins (ages 8–14) drawn from the Michigan State University Twin Registry. To comprehensively examine a range of eating pathology, several disordered eating attitudes and behaviors (e.g., body dissatisfaction, binge eating) were examined via self-report questionnaires. Questionnaires also were used to assess peer group emphasis on body weight and shape. Replicating previous results, significant individual-level associations were found between membership in weight conscious peer groups and disordered eating. However, co-twin control analyses indicated that these associations were largely due to genetic and/or shared environmental selection factors rather than pure socialization effects. Importantly, results remained unchanged when controlling for pubertal status, suggesting that effects do not vary across developmental stage. Overall, these findings question whether associations between weight conscious peer groups and disordered eating are due entirely to socialization processes. Future studies are needed to identify the specific genetic and/or shared environmental factors that may drive selection into weight conscious peer groups.

Keywords

selection; socialization; co-twin control; disordered eating; weight conscious peer groups

Prior research has demonstrated significant associations between weight conscious peer groups (e.g., groups that emphasize thin body weights/shapes, dieting, and appearance) and levels of disordered eating. Multiple cross-sectional studies have shown associations between peer groups' emphasis on body weight and excessive weight concerns, dieting and body dissatisfaction in girls (Levine & Smolak, 1992; Taylor et al., 1998; Vander Wal & Thelen, 2000; Wertheim, Paxton, Schutz, & Muir, 1997). Likewise, body image concerns, dietary restraint, the use of extreme weight-loss behaviors (Paxton, Schutz, Wertheim, & Muir, 1999), and binge eating (Goldschmidt et al., 2014) have been shown to be similar within friendship groups. Longitudinal studies have corroborated cross-sectional effects by demonstrating significant, prospective associations between peer groups' initial level of weight concerns and disordered eating symptoms 6 months to 10 years later (Crandall, 1988; Haines, Neumark-Sztainer, Eisenberg, & Hannan, 2006; Keel, Forney, Brown, & Heatherton, 2013; Myer & Waller, 2001).

However, the mechanisms underlying these significant associations remain largely unknown. Researchers have proposed socialization effects (Crandall, 1988; Zalta & Keel, 2006), such that disordered eating attitudes and behaviors are learned through observing these cognitions/behaviors within peer groups. Two studies have indirectly examined these social explanations by exploring the convergence versus divergence of disordered eating within peer groups. Crandall (1988) found that, at the beginning of the academic year, a female college student's level of binge eating was equally similar to her sorority friends as to her sorority as a whole, whereas at the end of the year, the student's level of binge eating was significantly more correlated with members of her sorority friendship group. Likewise, Zalta and Keel (2006) found that college roommates who cohabited during the academic year became less similar to each other in bulimic symptoms (i.e., the EDI bulimia subscale; Garner, 1991) over their summer break away. These studies indirectly support socialization theories, as they suggest that time together increases similarity in disordered eating symptoms amongst peers (Crandall, 1988), while time apart decreases peers similarity in these symptoms (Zalta & Keel, 2006).

However, other studies suggest that pre-existing factors (i.e., selection effects - see Burt et al., 2010; McGue, Osler & Christesen, 2010) may also contribute to associations between weight conscious peer groups and increased disordered eating. Selection occurs when individuals seek out peer groups that are in line with their own attitudes and beliefs. In the case of disordered eating, girls who strongly value thin body weights and shapes might seek out peer groups who also focus on these characteristics. These weight conscious peer groups may then reinforce and strengthen their underlying beliefs and lead to increased disordered eating within the group. In this case, selection effects may drive the association between weight conscious peers and disordered eating by grouping like-minded individuals, rather than this association being driven solely by exposure to a particular set of peers.

Three studies have examined this possibility, and two found evidence for possible selection effects. Myer and Waller (2001) investigated convergence/divergence of bulimic symptoms in unselected college roommates across three time points (i.e., 1 week after move in, and then 10 and 24 weeks later). Unselected roommates did not become more similar to each other in bulimic symptoms; in fact, they became more dissimilar, as evidenced by a

divergence in bulimic symptoms over the course of the study (Myer & Waller, 2001). Rayner et al. (2013) examined whether adolescents' (ages 12 to 14) similarity to their peer groups in weight/shape concerns was prospectively associated with an increased likelihood of staying in that peer group across 12 and 24 months. Results indirectly support the presence of selection effects, as girls were more likely to stay in friendship groups that were more similar in body dissatisfaction and bulimic behaviors. Importantly, one study failed to find peer selection effects, as female college students who selected their roommates were not more similar in their levels of drive for thinness, body dissatisfaction, or bulimic symptoms than students who were unselected (i.e., randomly paired; Gilbert & Myer, 2004).

Overall, findings are mixed as to whether socialization or selection accounts for peer group/ disordered eating associations, as an equal number of studies support each type of process (N = 2 for each). However, a relatively small number of studies have been conducted, and most have not simultaneously examined both socialization and selection factors. Indeed, although Crandall (1988) and Zalta and Keel (2006) showed socialization effects, they did not examine/control for initial selection into those peer groups. Thus, it remains unknown whether socialization alone, or socialization that is dependent upon initial selection into like-minded peer groups, accounts for associations between weight conscious peer groups and disordered eating.

One approach for simultaneously examining both processes is the co-twin control study. The co-twin control design is based on the counterfactual model (McGue et al., 2010) that states that the best way to determine whether a risk factor (e.g., exposure to weight conscious peers) is causal for a particular outcome (versus due to selection processes) is to examine the outcome when the individual is exposed to the risk factor and when the individual is not exposed. If the outcome is the same regardless of whether the individual is exposed or not-exposed, then the risk factor is not causal (i.e., it does not affect the outcome). However, if the individual's outcome is different depending on whether he/she was exposed, then it is likely that the risk factor does lead to the outcome and risk factor/outcome associations are not due to selection factors. Unfortunately, for most risk factors, it is impossible to observe an outcome for both exposure and non-exposure simultaneously in one person. Consequently, the counterfactual model attempts to get as close as possible to this ideal situation by comparing exposed and unexposed groups who are matched on as many key characteristics and potential selection factors as possible. This matching ensures that exposure to the risk factor is not based on selection via pre-existing traits, as the two groups are matched on traits that could lead to selection into the exposed group.

Importantly, the co-twin control method provides a valuable extension of the counterfactual model by using *co-twin discordance* on a risk factor as a predictor of an outcome. This method eliminates the need for matching since twins are already matched on key demographic characteristics (i.e., age, socioeconomic status, rearing family, etc.). Moreover, the model improves upon the typical counterfactual model by matching twins on shared environmental influences (i.e., environmental influences shared by siblings, e.g., parental divorce, parental discipline, etc.) and genetic predispositions, as twins reared together share 100% of their shared environment, and ~50% (for dizygotic [DZ] twins) or 100% (for monozygotic [MZ] twins) of their segregating genes. The ability to control for and model

genetic risk is particularly important for disordered eating, as twin and adoption studies show significant genetic influences (i.e., heritability ≥ 50%) on clinical eating disorders and their symptoms (Bulik, Sullivan & Kendler, 1998; Kaye, Klump, Frank, & Strober, 2000; Klump, Burt, Spanos, McGue, Iacono, & Wade, 2010; Klump et al., 2003; Klump, Miller, Keel, McGue, & Iacono, 2001).

Capitalizing on the use of twins, the co-twin control study compares three different sets of regression results: (1) individual-level effects (i.e., the extent to which a twin's exposure to weight conscious peers predicts her own level of disordered eating), (2) effects in DZ twin pairs only (i.e., the extent to which co-twin discordance on exposure to weight conscious peers predicts each DZ twin's level of disordered eating), and (3) effects in MZ twins only (i.e., the extent to which co-twin discordance on exposure to weight conscious peers predicts each MZ twin's level of disordered eating). The presence of significant individual-level effects would be similar to what has been shown in previous cross-sectional research - they simply show significant associations between the exposure and outcome, not differentiating socialization from selection effects.

By contrast, the MZ and DZ twin pair estimates help determine if individual-level associations are due to socialization or selection effects. Because MZ and DZ co-twins share 100% of their shared environment, discordance between them on the exposure variable cannot be due to shared environmental selection effects. Thus, if shared environmental factors (e.g., socioeconomic status, parenting style) are important for selection into weight conscious peer groups, significant associations between exposure and disordered eating in either discordant MZ or DZ twins will not be observed[1]. If genetic factors are important for selection into weight conscious peer groups, then significant associations between exposure to weight conscious peers and disordered eating in discordant DZ twins will be observed, since these twins only share 50% of their genes. By contrast, an association in discordant MZ twin pairs would not be observed, since these twins share 100% of their genes – in this case, discordance in exposure to weight conscious peers cannot be due to differences in genetic factors between MZ co-twins.

Figure 1 provides a summary of expected results if socialization and/or selection effects are present. Scenario A shows expected results if associations between exposure to weight conscious peers and disordered eating are due entirely to socialization processes. In this case, there are significant associations with similar magnitude within individual-level and within discordant MZ and DZ twin pairs, suggesting that exposure to weight conscious peers increases disordered eating on the individual level and within-twin pairs even when shared environmental and genetic selection processes are controlled. Scenarios B and C show expected results if selection is present. In both cases, the association between exposure to weight conscious peer groups and disordered eating in discordant twins is attenuated, suggesting that the association is due to either genetic and/or shared environment selection effects. Scenario B would suggest genetic selection effects only, as there is still an

[1]Unfortunately, the co-twin control design is unable to control for non-shared environmental factors (e.g., twins participating in different sports or after-school activities). These factors are unique to each co-twin, making it is impossible to match twins on these experiences and thus, these factors cannot be controlled for within the co-twin control models.

association for DZ twins who share less genetic material than MZ twins. Scenario C would suggest shared environmental and/or genetic and shared environmental selection effects, since associations are no longer significant or are reduced in magnitude when controlling for shared environmental and genetic selection processes. Since no portion of our sample controls for 100% of genetic influences and 0% of shared environmental influences (MZ twins reared apart would be one such sample), we are unable to detect if selection effects identified in Scenario C are due completely to shared environmental effects or to both genetic and shared environmental effects. Nonetheless, the ability to separate socialization, selection due to genetic effects only, and selection due to genetic and/or shared environmental effects represents a significant advance over prior studies.

Using the co-twin control method, the present study directly explored socialization versus selection effects in associations between weight conscious peer groups and disordered eating in pre-adolescent and young adolescent female twins. Although previous research has primarily focused on college-aged women, peer groups become more developmentally important in early adolescence (e.g., ages 10–14; Steinberg & Monahan, 2007) when disordered eating often begins. Our focus on this younger age group therefore may highlight patterns of effects that are unique to the early (and critical) stages of disordered eating development. Nonetheless, because of our younger age range, we focus our analyses on a variety of disordered eating attitudes and behaviors (rather than clinical disorders) in order to maximize power and examine the full spectrum of eating pathology.

Methods

Participants

The current study used archival twin data (N = 610 female twins ages 8–14 years, M = 11.63, SD = 2.10; 52.8% MZ, 47.2% DZ) from the *Twin Study of Mood, Behavior, and Hormones during Puberty (MBHP).* This study recruits all of its twins from the Michigan Twins Project (MTP), a population-based recruitment database within the Michigan State University Twin Registry (MSUTR; Burt & Klump, 2013; Klump & Burt, 2006) that recruits twins ages 3–25 and 30–50 years using birth records in collaboration with the Michigan Department of Community Health (see Burt & Klump (2013) for recruitment details). Although the MTP is an on-going project, the current response rate (57%) is on par or better than that of other twin registries using similar recruitment methods (Burt & Klump, 2013; Iacono & McGue, 2002), and participating twins are representative of the broader population in terms of racial/ethnic diversity, family income, parental education, and a range of other variables (including emotional problems – see Burt & Klump, 2013).

The primary aim of the on-going MBHP study is to investigate the influence of ovarian hormones on phenotypic and genetic risk for disordered eating during puberty. Thus, several inclusion/exclusion criteria were used (e.g., no recent psychotropic, steroid, or other medication use that is known to influence hormone functioning) that could conceivably alter the composition and disordered eating characteristics of the recruited sample. Notably, however, twins who were assessed for the MBHP were not significantly different from non-participating MTP families in terms of overall disordered eating symptoms (e.g., body dissatisfaction, dieting, binge eating) ($t(391) = -0.95$ $p = 0.35$) or BMI ($t(375) = -0.84$ $p =$

0.40). The recruited MBHP was also highly representative of the MTP sample and the general population of Michigan in terms of ethnic/racial distributions, with 4% of pairs identifying as Hispanic and 81.5% identifying as Caucasian, 8.3% as African American, 0.2% as American Indian/Alaskan Native, and 9.3% as multiple races.

Zygosity Determination

The MSUTR determines zygosity using a well-validated physical similarity questionnaire (Lykken, Bouchard Jr, McGue, & Tellegen, 1990) that has been shown to be 95% accurate when compared to genotyping (Peeters, Van Gestel, Vlietinck, C. Derom, & R. Derom, 1998). For the MSUTR sample used in this study, both twins, their mother, and two research assistants evaluated the physical similarities independently. Reports were then compared and any discrepancies were resolved through review of questionnaire data and twin photographs by one of the principal investigators (KLK) or by examination of DNA markers (Burt & Klump, 2013; Klump & Burt, 2006).

Measures

All measures were completed by the twins. Although we did not have parental reports of weight-conscious peer groups, we did have parental reports of twin disordered eating (via a parent report version of the Minnesota Eating Behavior Survey see description below). However, similar to previous studies (Kolko & Kazdin, 1993), the parental reports did not significantly correlate with the twin's report of disordered eating (r's mainly <.16), and individual-level associations between parental reported disordered eating and twin reported weight-conscious peer groups were small in magnitude (average r = .13). These non-significant associations likely reflect the lack of information parents have about their child's eating disorder symptoms, particularly the cognitive symptoms (e.g., weight preoccupation) and behavioral symptoms that children often hide (e.g., purging). Thus, in the current study, we focus on twin reported symptoms in all analyses.

Exposure to Weight Conscious Peer Groups—As described below, we used several measures to assess exposure to weight conscious peer groups, many of which seem to tap related constructs. Although intercorrelations between scores on peer exposure questionnaires were significant and positive (rs = .31–.56, Mean = .42), all correlations were small-to-moderate in magnitude, with only 10–31% shared variance. This relatively limited amount of shared variance suggests that the questionnaires tap different aspects of weight conscious peer group exposure. Thus, all analyses were conducted separately for each questionnaire. Notably, these questionnaires have been studied much less frequently than those used to assess disordered eating, and so psychometric data is limited (but is still described below).

The *Perceived Friend Preoccupation with Weight and Dieting Scale* (Schutz, Paxton, & Wertheim, 2002) is a 9-item questionnaire aimed at assessing twins' perceptions of the frequency of weight- and dieting- related thoughts and behaviors among their friends. This self-report questionnaire has participants rate their response on a 5-point scale from 1 (never) to 5 (very often), with high scores representing more perceived importance of weight and dieting among friendship groups. Factor analysis found all items load on a single factor

with item-total correlations ≥ .52 (Schutz et al., 2002). Cronbach's alpha in a sample of adolescent girls in grades 7, 8, and 10 was excellent at .87 (Schutz et al., 2002). Similarly, Cronbach's alpha in the present study was excellent at .86.

The *Appearance Conversations with Friends* (Jones, Vigfusdottir, & Lee, 2004) is a 5-item questionnaire modified from the original Magazines as a Source of Influence Scale (Levine, Smolak, & Hayden, 1994). The current scale assesses the frequency of discussions about current and desired body shape with friends (e.g., "My friends and I talk about what we would like our bodies to look like"). Cronbach's alpha of .85 indicated excellent reliability for a sample of adolescent females in grades 7, 8, 9, and 10 (Jones et al., 2004). Cronbach's alpha in the present study was .88.

The *Friends as a Source of Influence Scale* (Paxton et al. 1999) is a 5-item questionnaire that asks participants to rate how important their friends are in influencing their opinions of the perfect body, diet products, exercise, and dieting (e.g. "Your idea of the perfect body") on a 5-point scale from 1 (Not at all important) to 5 (Very important). Cronbach's alpha of .87 indicates good internal consistency in a study of 10th grade girls (mean age = 15.5) (Paxton et al., 1999). In the present study, the Cronbach's alpha was .84.

The *Peer Attribution Scale* (Lieberman, Gauvin, Bukowski, & White, 2001) includes 8-items assessing appearance-related attributions (e.g., "My friends would like me more if I lost weight) from friends on a 6-point Likert scale (ranging from "false" to "true"). The original scale includes items referring to same-sex and opposite-sex friends; however, in the current sample, we used a modified version (Shroff & Thompson, 2006) that refers to any friend (same- or opposite-sex) and deletes items that are specific to opposite-sex friends only (e.g., "If I was thinner, boys would be more attracted to me"). This modified scale exhibited a Cronbach's alpha of .85 in past research (Shroff & Thompson, 2006) and .80 in the current sample.

Disordered Eating—The *Minnesota Eating Behavior Survey (MEBS;* von Ranson, Klump, Iacono & McGue, 2005)[2] is a 30-item questionnaire made up of true/false questions that assesses a range of disordered eating symptoms. This measure was developed for use with children as young as 10-years-old. Previous factor analyses (Klump, McGue, & Iacono, 2000; von Ranson et al., 2005) produced four factors: Body Dissatisfaction (i.e., assessing discontent with body size and shape), Compensatory Behaviors (i.e., assessing the use of, and thoughts of using, self-induced vomiting and other inappropriate compensatory behaviors to control weight), Binge Eating (i.e., assessing thinking about binge eating as well as engaging in binge eating and/or secretive eating) and Weight Preoccupation (i.e., assessing preoccupation with weight, eating, and dieting). The current study focused on the Body Dissatisfaction, Binge Eating and Weight Preoccupation subscales, as well as the MEBS Total Score (i.e., a measure of overall levels of disordered eating that is the sum of all

[2]The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory (M-EDI)) was adapted and reproduced by special permission of Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, & Polivy (1983) Copyright 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

30 items on the questionnaire). The Compensatory Behaviors subscale was excluded from analyses due to the low prevalence of compensatory behaviors in the sample (i.e., 90% of participants scored a "0" on this subscale).

The MEBS subscales that were examined demonstrate good three-year stability (r = 0.32–0.59) in adolescents (age 11–14) with the Total score being the most stable (r = 0.59) followed by the scales measuring attitudes (i.e., Weight Preoccupation and Body Dissatisfaction; r = 0.51 and r = 0.53, respectively) and then behaviors (i.e., Binge Eating; r = 0.32) (von Ranson et al., 2005). The MEBS subscales also demonstrate adequate convergent validity with similar types of scales from the Eating Disorder Examination Questionnaire (EDE-Q; Fairburns & Beglin, 1994), with inter-scale correlations ranging from .74–.83 (von Ranson et al., 2005). Good criterion-related validity is present for the MEBS as well, as girls with eating disorders (i.e., either anorexia nervosa [AN] or bulimia nervosa [BN]) were shown to have significantly higher scores on the Body Dissatisfaction subscale, Weight Preoccupation subscale, and Total Score than controls (von Ranson et al., 2005). Additionally, participants with BN had significantly higher scores on the Binge Eating subscale than controls (von Ranson et al. 2005). Finally, internal consistencies in previous studies have ranged from 0.70–0.85 (von Ranson et al., 2005) and were 0.68–0.88 in the current study.

The *Eating in the Absence of Hunger for Children and Adolescents (EAH-Child;* Tanofsky-Kraff et al., 2008) is a 14-item questionnaire developed to assess precipitants to eating when not hungry in children and adolescents ages 6 through 19. This questionnaire includes five emotional precipitants to eating when one is not hungry (i.e., feeling sad or depressed, angry or frustrated, anxious or nervous, tired, bored) and two external precipitants (i.e., sensory cues, social cues). Using a 5-point Likert scale with answers ranging from 1 ("Never") to 5 ("Always"), participants are asked to select the frequency in which they eat beyond satiation and in the absence of hunger in response to each of the 7 precipitants. Factor analysis generated three subscales on this measure Negative Affect (i.e., eating in the absence of hunger in response to feeling sad or depressed, angry or frustrated, or anxious or nervous), External Eating (i.e., eating in absence of hunger when food looks, tastes, or smells good and when others are eating), and Fatigue/Boredom (i.e., eating in absence of hunger when feeling tired or bored) (Tanofsky-Kraff et al., 2008). Significant test-retest correlations (r = 0.65–0.70) have been observed for these scales across 5–565 days (M = 150 days, SD = 130), and they show good convergent validity with interview-based reports of loss of control over eating (Tanofsky-Kraff, et al., 2008). Finally, internal consistency was excellent for all subscales in past studies (0.80–0.88; Tanosky-Kraff et al., 2008) and the current sample (0.75–0.84).

The *Emotional Eating Scale- Adapted for Children and Adolescents (EES-C;* Tanofsky-Kraff et al., 2007) is a 26-item, self-report questionnaire designed to assess the urge to cope with negative affect through eating. Participants are presented with a list of emotions (e.g., resentful, discouraged, worn out) and asked to rate their desire to eat in the presence of each emotion on a 5-point scale from "I have no desire to eat" to "I have a very strong desire to eat." A factor analysis generated three subscales that included: 1) eating in response to anxiety, anger, and frustration (e.g., furious, worried); 2) eating in response to depressive

symptoms (e.g., down, sad); and 3) eating in response to feeling unsettled (e.g., excited, resentful). Good temporal stability was observed over an average of three months with intraclass correlations ranging from 0.59–0.74, depending upon the subscale (Tanofsky-Kraff et al., 2007). All subscales exhibited excellent internal consistency in prior work (0.83–0.93; Tanosky-Kraff et al, 2007) and the current study (0.72–0.92).

Covariate—Pubertal status was included in the models to ensure that associations between exposure to weight conscious peer groups and disordered eating were not due to this potentially confounding factor[3]. Indeed, past research has demonstrated that pubertal development is related to both phenotypic (Klump et al., 2013) and genetic risk (Culbert, Burt, McGue, Iacono, & Klump, 2009; Klump et al., 2000; Klump, Burt, McGue, & Iacono, 2007; Klump, McGue, & Iacono, 2003) for disordered eating symptoms. Further, a positive association was found between pubertal development and disordered eating within the current study (average $r = 0.15$), as well as pubertal development and exposure to weight conscious peer groups (average $r = 0.28$).

Pubertal development was assessed using the self-report *Pubertal Development Scale (PDS*; Peterson, Crockett, Richards, & Boxer, 1988). The PDS which asks participants to assess their pubertal development based on physical markers of puberty (i.e., height spurts, body hair growth, skin changes, breast development, onset of menarche). Participants rated the development of these physical markers on a 4-point scale: (1) development has not yet begun; (2) development has barely started; (3) development is definitely underway; and (4) development seems completed. An exception to this 4-point scale was the coding for menses, which was coded dichotomously as either absent (1) or present (4). The ratings of each physical marker are summed and averaged to obtain an overall PDS score, with higher scores representing more advanced pubertal development. The PDS correlates highly ($r = .61$–$.67$) with physician ratings of pubertal development (Peterson et al., 1988) and exhibited good internal consistency (alpha = .77) in the present sample.

Statistical Analyses

Data Preparation—Disordered eating and weight conscious peer group data were log transformed prior to analyses to control for positive skew. All scores were also standardized for the co-twin control analyses in order to allow for comparisons of effects across measures and analyses.

Initial Associations—Before examining associations between exposure to weight conscious peer groups and disordered eating, we first examined the extent of "discordance" in peer group exposure in MZ and DZ twins to ensure adequate variability in the independent variable. We calculated within-pair difference scores (i.e., absolute value of the difference between Twin 1's and Twin 2's score) for the peer exposure variables and compared the scores for MZ versus DZ twins using independent samples t-tests.

[3]To ensure that results also did not vary by age or body weight, we ran analyses controlling for age (which correlates highly with pubertal development, $r = .79$) and body mass index (calculated from height and weight measurements). Results were identical to those described below, and thus, only analyses with pubertal development as a covariate are included herein.

Pearson correlations were used to investigate within-person associations between exposure to weight conscious peer groups and disordered eating. These analyses were essentially replications of cross-sectional studies that examine whether individuals with weight conscious peer groups have significantly higher levels of disordered eating.

Co-Twin Control Analyses—Co-twin control analyses were then used to examine whether phenotypic associations between exposure to weight conscious peer groups and disordered eating symptoms were due to purely socialization factors (i.e., higher weight conscious peer groups lead to higher disordered eating) or selection (i.e., individuals who are more inclined towards disordered eating are more likely to choose weight conscious peer groups). We used mixed linear models (MLMs) to examine these possibilities and also to control for the non-independence of the twin data (by nesting a level-1 variable (individual twin) within a level-2 unit (twin pair)).

Separate MLMs were used to examine individual-level effects (i.e., associations between exposure to weight conscious peer groups and disordered eating within each twin) versus within-twin pair effects (i.e., associations between co-twin discordance in exposure to weight conscious peer groups and disordered eating in each twin). More specifically, the individual-level effects were estimated in models that regressed the disordered eating scores onto the exposure variable using the following equation:

$$Y_{ij}=\beta_0+\beta_1 x_{ij}+\varepsilon_{ij}$$

where Y_{ij} is the observed outcome (i.e., disordered eating) for the jth twin (j= 1 or 2) in the ith twin pair (i= 1,2,…, N), β_0 is the intercept term, β_1 is the individual-level effect of exposure (i.e., weight conscious peer groups) on outcome (i.e., disordered eating), x_{ij} is the level of exposure for the jth twin in the ith twin pair, and ε_{ij} is the residual (correlated across two members of a twin pair).

By contrast, the within-pair effects were modeled using both a within-pair (β_W) and a between-pair (β_B) effect that were estimated using the following regression model:

$$Y_{ij}=\beta_0+\beta_W\,(x_{ij}-x_i)+\beta_B x_i+\varepsilon_{ij}$$

where, β_W is the within-pair effect of exposure to weight conscious peer groups, x_{ij} is the level of exposure for the jth twin in the ith twin pair, x_i is the mean exposure index for the ith twin pair, β_B is the between-pair effect of exposure. In order to appropriately model all of these effects, the MLMs for the within-pair analyses included five predictor variables and one covariate (i.e., pubertal status): 1) each twin's score on the exposure to weight conscious peers scale (i.e., the individual-level effect); 2) the difference between each co-twin's score on the weight conscious peers scale and the pair's mean on the scale (i.e., the within-pair effect); 3) a dummy coded zygosity variable that identifies MZ versus DZ twins; 4) pubertal status; 5) an interaction between the zygosity variable and the twin's score on the weight conscious peers scale; 6) an interaction between the zygosity variable and the within-twin pair difference score. These latter interaction variables estimated the between-family effects

(i.e., zygosity x exposure to weight-conscious peers) and tested for significant differences in within-pair effects between MZ and DZ twins (i.e., zygosity x within-twin pair difference scores). Notably, in order to obtain estimates of all effects in MZ and DZ twins separately, we ran two sets of MLMs – one in which zygosity was coded with the DZ twins as the control (i.e., MZ twins = 2 and DZ twins = 1), and the other with MZ twins as the control (i.e., MZ twins = 1, DZ twins = 2). The first model provided estimates of within-pair exposure for the MZ twins, while the second model provided these same estimates for DZ twins.

We conducted individual models for each of the disordered eating outcome variables (e.g., Body Dissatisfaction, Binge Eating, etc.) with each peer group exposure questionnaire (e.g., Perceived Friend Preoccupation with Weight and Dieting, etc.). Due to the relatively large number of models examined, a conservative p value of .01 was used for all analyses.

Results

Preliminary Analyses

A range of disordered eating attitudes and behaviors and exposure to weight conscious peer groups was present in our sample (see Table 1). A total of 3.9% of twins scored above the clinical cut-off (score = 15.55) for the MEBS Total Score (von Ranson et al., 2005), which is a relatively high percentage, given the lower age range of our sample (M = 11.63; SD = 2.10). Moreover, a wide range of scores were present on the exposure to weight conscious peer groups scales, as 10.9%–14.4% of twins scored ≥1 SD above the mean on these scales.

Within-pair difference scores for peer exposure variables are presented in Table 2. Interestingly, and similar to previous research (Rushton & Bons, 2005), significantly smaller differences in exposure to weight conscious peer groups were observed in MZ as compared to DZ pairs. However, effect sizes were small (d's = 0.11 to 0.21), suggesting that overall, the degree of co-twin "discordance" in peer groups was relatively similar in MZ and DZ twins.

Initial Phenotypic Associations

Within-person, phenotypic associations between weight conscious peer groups and disordered eating are presented in Table 3. Significant positive associations were found between all disordered eating and peer group scores; however, correlations varied in magnitude depending upon the questionnaires. Stronger correlations were observed between weight conscious peer groups and the disordered eating symptoms assessed with the MEBS (r's = .25–.48) as compared to symptoms assessed with the EAH-C and EES-C scales (r's = .10–.32).

Co-Twin Control Analyses

Overall, findings from the co-twin control analyses suggested that the vast majority of within-person associations observed in Table 3 were accounted for by effects that are consistent with genetic and/or environmental selection (i.e., Scenario C). Table 4 displays results for all associations showing Scenario C. In many cases, the MZ and/or DZ within-

pair estimates were not significantly different from zero, suggesting minimal associations between twin discordance in weight-focused peer groups and disordered eating when controlling for genetic and/or shared environmental factors. At times, a within-pair estimate was statistically significant (e.g., the MZ within-pair estimate for associations between MEBS Body Dissatisfaction and Perceived Friend Preoccupation), but in all cases, there were no significant differences between the MZ and DZ within-pair estimates (i.e., the "Ex x Zyg" interaction was non-significant), *and* all within-pair estimates were significantly reduced in magnitude ($p < .05$) as compared to the individual-level estimates (see Cumming (2009) for methods for beta comparisons). All of these results suggest the presence of Scenario C (i.e., genetic and/or shared environmental selection), as associations between exposure to weight-focused peers and disordered eating are significantly diminished when controlling for genetic and/or shared environmental factors.

Notably, there were a few associations that showed Scenario B (genetic selection), or a mix of Scenario A and C (socialization and selection) (see Table 5). One association showed strong evidence in support of Scenario B - the association between the Emotional Eating Scale- Unsettled and Appearance Conversations with Friends (see double lined cell in Table 5). A significant DZ within-pair effect and no significant MZ within-pair effect was observed for this association, the within-pair DZ effect was significantly greater than the MZ effect, and the within-pair DZ effect was similar in magnitude to the DZ individual-level estimate. These findings suggest genetic selection effects only, as when genetic effects were completely controlled (i.e., within MZ twins), the association was not observed.

By contrast, a few associations showed a mix of Scenarios A and C (see solid line cells in Table 5). In some of these cases (e.g., associations between Weight Preoccupation and Friends as a Source of Influence), the within-pair estimates were significant for both MZ and DZ twins, and these estimates were not significantly different from each other (e.g., the Ex x Zyg interaction was non-significant), suggesting Scenario A. However, the MZ and DZ within-pair associations were either significantly reduced in magnitude ($p < .05$) as compared to their corresponding individual-level effects, or they were substantially reduced (e.g., nearly half of the within-individual estimate – see MEBS Binge Eating with the Appearance Conversations with Friends scale). These reductions suggest that Scenario C is also present, since controlling for genetic and shared environmental selection factors attenuates associations between weight conscious peer groups and disordered eating (see Figure 2). Another example of associations that resembled both Scenarios A and C included situations where the MZ twin estimate was statistically significant, the DZ twin estimate was non-significant, *and* there were significant differences between the MZ and DZ estimates (see the MEBS Weight Preoccupation with Perceived Friend Preoccupation with Weight and Dieting). In these cases, the non-significant DZ pair estimate suggests the presence of selection (i.e., Scenario C), but the ability for exposure to predict results even when controlling for genetic and/or shared environmental influences with the MZ twins also suggests that socialization effects may be present (i.e., Scenario A).

Finally, there were a small number of associations in which the effects were more difficult to interpret (see dotted line cells in Table 5). In all of these cases, results were trending toward Scenario B, or genetic selection effects, in that the DZ within-pair estimate was statistically

significant and it was not significantly reduced ($p > .05$) from the DZ within-individual estimate. Moreover, the MZ within-pair estimates were not statistically significant, and in some cases, even the MZ within-individual estimates were not significant. These results would clearly suggest Scenario B, except that the Ex x Zyg interaction was non-significant, leading to ambiguity around whether the MZ and DZ within-pair estimates were very different from each other. However, given the difficulty in detecting statistically significant interactions in regression and MLM models, and the bulk of evidence in support of Scenario B for these associations, we tentatively interpret these results to show Scenario B, genetic selection effects.

Discussion

Findings from this study are the first to suggest that associations between weight conscious peer groups and disordered eating may be due to genetic and/or shared environmental selection factors rather than pure socialization effects. Results showed that girls who exhibit more disordered eating (either due to genetic or shared environmental predispositions) appear to select into weight conscious peer groups rather than socialization within these peer groups leading to increased disordered eating. Support for these types of genetic and/or shared environmental selection effects persisted even when controlling for pubertal status. This pattern was consistently observed across multiple disordered eating constructs (i.e., body dissatisfaction, weight preoccupation, binge eating, eating in the absence of hunger, emotional eating) and multiple measures of peer group exposure. Overall, these findings question whether associations between weight conscious peer groups and disordered eating are due entirely to socialization processes.

Previously, researchers have suggested that socialization may be the mechanism at work in the association between weight conscious peer groups and disordered eating. Studies examining the convergence or divergence of disordered eating behaviors with exposure to peer groups have provided support for socialization, in that individuals who spend more time together become more similar in their level of disordered eating (Crandall, 1988; Zalta & Keel, 2006). However, these studies were unable to entirely control for selection effects, since neither study accounted for whether the individuals were friends prior to living together in the sorority (Crandall, 1988) or living together as roommates (Zalta & Keel, 2006). When peer groups were completely unselected, Myer and Waller (2001) observed a lack of convergence in bulimic behavior. Additional support for selection effects was found when Rayner et al. (2013) examined girls' friendship groups across time and noted that girls tend to stay in friendship groups that are more similar to their own body dissatisfaction and bulimic behavior.

Results from our study corroborate these latter findings by supporting the presence of genetic and/or shared environmental selection effects. When genetic and shared environmental influences were controlled for using the co-twin control method, the association between exposure to weight conscious peer groups and disordered eating was either eliminated or significantly reduced. Aggregating findings across all studies then, it is possible that genetic and/or shared environmental selection effects may drive who one chooses to affiliate with initially (e.g., who decides to join a sorority), and then socialization

may work to increase the similarity within a particular peer group (VanHuysse et al, submitted). This hypothesis would reconcile previous inconsistent findings and would help account for the small number of associations (~20%) in our study that showed both socialization and selection effects (i.e., Scenarios A and C).

Interestingly, another recent co-twin control study from the MSUTR examined associations between weight conscious peer groups and *thin-ideal internalization* in a sample that overlapped with the current one (i.e., 92% overlap; VanHuysse et al., submitted). This study found stronger support for socialization effects, although results also highlighted a complimentary role of selection (VanHuysse et al, submitted). Integration of the findings from VanHuysse et al. (submitted) and the current study seem to support the presence of both mechanisms. Specifically, genetic and/or environmental risk for disordered eating (and to a lesser extent thin-ideal internalization) may lead young girls to select into weight conscious peer groups. Within these peer groups, socialization effects may then work to increase thin-ideal internalization and, to a lesser extent, disordered eating symptoms (VanHuysse et al., submitted).

Moving forward, it will be important to identify the genetic and/or shared environmental influences contributing to selection into weight conscious peer groups. Although our co-twin control design advances prior research by providing evidence for selection effects, the design is unable to determine the degree to which genetic and/or shared environmental effects contribute to the selection processes. Future studies could use multivariate models (e.g., Cholesky decomposition models) to disentangle these genetic/shared environmental influences and advance our understanding of the underlying selection processes.

Ideally, these studies would also assess and examine the specific factors that may contribute to genetically and/or environmentally mediated selection into weight conscious peer groups. The current study controlled for pubertal development, suggesting that the selection effects observed in this study are independent of pubertal stage.[3] These results were somewhat unexpected, as prior research has demonstrated increased genetic (and decreased shared environmental) risk for disordered eating with more advanced pubertal development (Culbert et al., 2009; Klump et al., 2000; Klump et al., 2007). Thus, we thought we might observe stronger genetic (and weaker shared environmental) selection effects in pubertal girls as opposed to pre-pubertal girls. The lack of differences suggests that there are stable genetic and/or shared environmental factors that drive selection into weight-conscious peer groups across development.

Two specific factors that might contribute to selection into weight focused peer groups are perfectionism and maternal disordered eating. Perfectionism is a personality trait that tends to develop early in life (prior to peer group selection), it is significantly heritable (.39 to .58; Jang, Livesley, Vernon, & Jackson, 1996; Tellegen et al., 1988), and it exhibits significant associations with a range of eating disorder symptoms (e.g., fasting, binge behaviors) and

[3]In order to further ensure that results do not differ by pubertal status, we also examined pubertal status as a moderator (instead of covariate) in analyses. The sample was divided into pre-pubertal and pubertal groups (with a mean score of 2.5 on the PDS used as a cut-off). Co-twin control models were run separately in each group. The overall results were consistent between the pre-pubertal and pubertal groups, as they both continued to favor Scenario C, suggesting selection effects do not seems to vary by pubertal status.

diagnoses (Forbush, Heatherton & Keel, 2007). As noted by Zalta and Keel (2006), it is possible that young girls who are raised in an environment with excessively high standards and/or who are genetically predisposed to perfectionistic qualities may select into peer groups with other girls who are self-critical and have high standards. These high standards could translate into an emphasis on society's notion of the "ideal" body and an increased rate of disordered eating symptoms. Maternal disordered eating also could serve as a genetic and/or shared environmental selection factor, as girls who inherit genes of risk from their mother and/or observe maternal disordered eating could select into peer groups who also exhibit or emphasize these behaviors.

Despite the many strengths of this study (i.e., the ability to test for selection effects, the examination of multiple disordered eating measures, etc.), this study was not without limitations. First, the co-twin control study design is unable to control for every factor that may be important in selection into weight-focused peer groups (e.g., consumption of media that emphasizes the thin-ideal). Propensity score matching (Caliendo & Kopeinig, 2008) within a twin study design or other designs may be a promising approach for exploring additional matching variables. Moreover, the co-twin control design is unable to control for environmental factors that are different between twins, such as non-shared environmental factors that are specific to each co-twin (e.g., twins participating in different sports or after school activities). Since these unique experiences vary across the twin pair, it is impossible to match twins on these experiences and thus, they cannot be controlled for within the models. It is possible that these non-shared environmental factors are causing selection into peer groups with differing levels of focus on body weight and body shape (i.e., one co-twin plays soccer [a less weight-focused sport] while another takes ballet classes [a more weight-focused sport]).

Second, we used data from only one informant (e.g., twin's self report). Associations between weight conscious peer groups and disordered could be inflated by shared method variance (e.g., similarities in response styles may lead to stronger associations between exposure and outcome variables; Podsakoff et al., 2003). Additional research is needed to explore associations using data from other informants (e.g., parental reports, peer reports) to ensure that results are unchanged. Further, the use of informant reports is also important given the possibility that individuals high in disordered eating might erroneously report a stronger emphasis on weight and shape in their peer groups than individuals lower in disordered eating.

Third, our participants were younger than those in most previous studies (Crandall, 1988 and Zalta & Keel, 2006; for an exception, see Rayner et al. (2013) who also identified selection effects in this age group). Although we would argue that adolescence is a critical time period when young girls are beginning to rely more on their peer groups (Steinberg, 1990), it remains unknown whether differences in results (between ours and previous studies' results) are due to possible age differences in effects (e.g., selection effects being more important in adolescence; socialization being more important in young adulthood). Future studies should examine this possibility by examining effects across adolescent and young adult ages.

Fourth, since this study was conducted using a non-clinical sample, it is unknown if the findings from this study generalize to a clinical population. However, the disordered eating symptoms that were examined are precursors to full clinical eating disorders (Killen, et al., 1996; Stice & Shaw, 2002), which suggests it is likely that similar results might be found in a clinical sample. One challenge in conducting the present study using a clinical sample would be finding a sufficient number of twins with clinical eating disorders to conduct a well-powered twin analysis. Nonetheless, future research is needed to assess whether findings translate to individuals with clinical eating disorders.

Finally, our data are cross-sectional, and we therefore are limited in our ability to confirm causal associations. Indeed, while this study investigated whether peer group's emphasis on weight and shape predicted disordered eating, it is possible that an individual's disordered eating could lead to greater emphasis on body weight and shape within the peer group. Future longitudinal studies are needed to assess whether individuals at higher risk for disordered eating symptoms prospectively select into weight conscious peer groups. This design could then assess whether, after selecting into these peer groups, exposure leads to enhanced disordered eating symptoms across time (i.e., demonstrating socialization effects). This longitudinal design may also allow for identification of specific selection factors that may drive selection into weight conscious peer groups (e.g., perfectionism, maternal dieting, etc.).

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General Scientific Summary

This study suggests that selection effects may contribute to the association between weight conscious peer groups and increased levels of disordered eating, such that girls with a genetic and/or environmental predisposition for disordered eating may select into peer groups who are more body or weight focused.

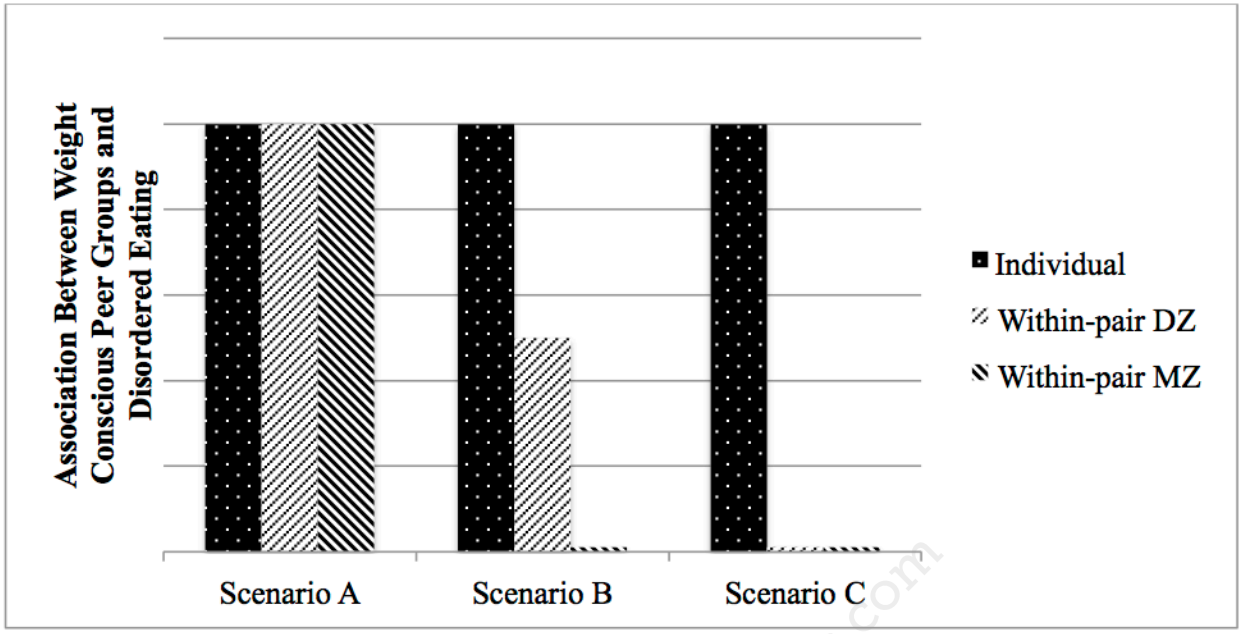

Figure 1. Summary of Potential Results of Co-Twin Control Analysis

Scenario A would indicate that the association between weight conscious peer groups and disordered eating are due completely to socialization processes, as there are significant associations on the individual-level and within discordant MZ and DZ twin pairs. Scenario B would indicate genetic selection effects only, as there is still an association for DZ twins who share less genetic material (approximately 50%) than MZ twins (approximately 100%). Scenario C would suggest genetic and/or shared environment selection effects, since when controlling for these types of influences, associations are non-significant or reduced in magnitude.

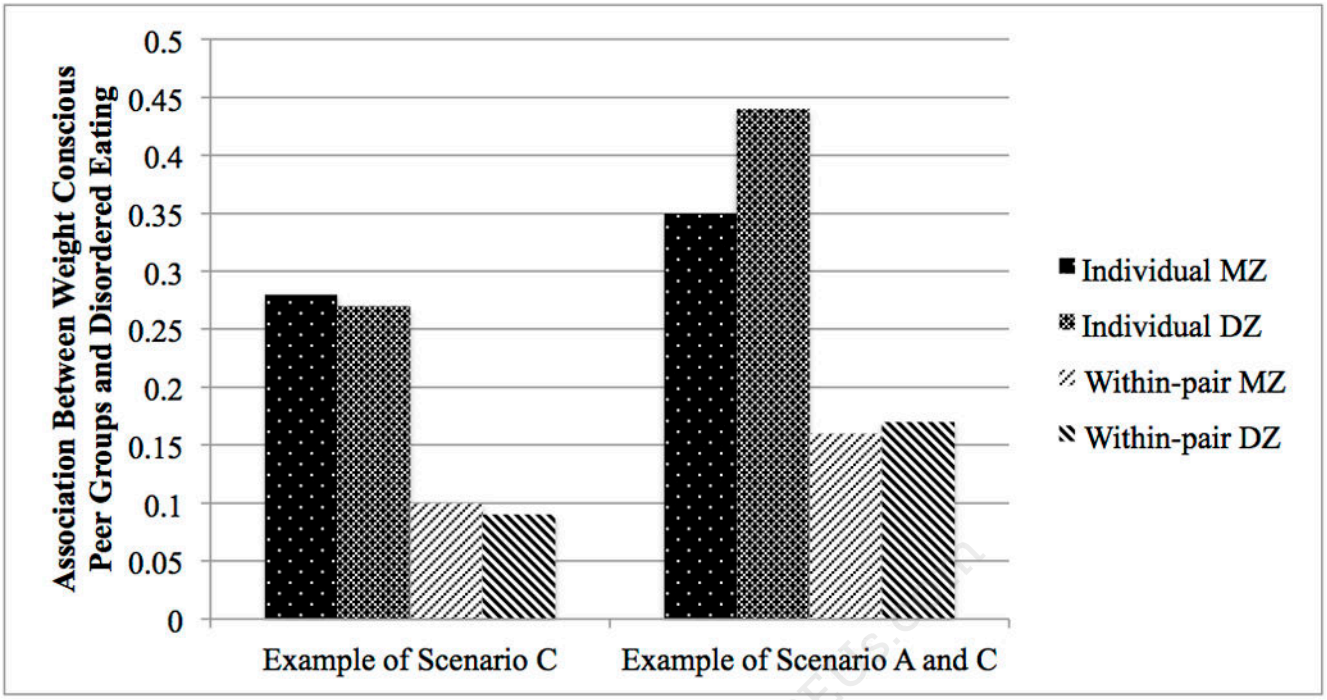

Figure 2. Examples of Scenario C and a Mixture of Scenarios A and C

Associations between Friends as a Source of Influence and Eating in the Absence of Hunger-Negative Affect are displayed to demonstrate effects that resemble Scenario C (i.e., significant individual-level effects and non-significant DZ and MZ within-pair effects). Associations between Peer Attribution Scale and the Minnesota Eating Behavior Survey-Total Score are displayed to represent associations that resemble a mix of Scenario A and C (i.e., the within-pair associations are significant resembling Scenario A, but reduced compared to the individual-level effects resembling Scenario C).

Table 1

Descriptive Statistics for Disordered Eating and Peer Exposure Measures (N = 538–609).

Measures	Mean	SD	Range in Sample
Disordered Eating Measures			
Minnesota Eating Behavior Survey (MEBS)			
Total Score (Possible range 0–30)	4.29	4.67	0–27
Body Dissatisfaction (Possible range 0–6)	0.94	1.50	0–6
Binge Eating (Possible range 0–7)	0.84	1.30	0–7
Weight Preoccupation (Possible range 0–8)	1.66	1.98	0–8
Eating in the Absence of Hunger (EAH)			
Negative Affect (Possible range 1–5)	1.19	0.41	1.00–3.50
External Eating (Possible range 1–5)	2.03	0.74	1.00–4.75
Fatigue/Boredom (Possible range 1–5)	1.44	0.61	1.00–5.00
Emotional Eating Survey(EES)			
Anxiety, Anger, & Frustration (Possible range 1–5)	1.46	0.60	1.00–4.25
Depression (Possible range 1–5)	1.65	0.69	1.00–4.57
Unsettled (Possible range 1–5)	1.59	0.67	1.00–5.00
Peer Exposure Questionnaires			
Perceived Friend Preoccupation with Weight and Dieting (Possible range 9–45)	14.45	5.49	9–37
Appearance Conversations with Friends (Possible range 5–25)	9.00	4.28	5–25
Friends as a Source of Influence (Possible range 5–25)	7.54	3.51	5–25
Peer Attribution Scale (Possible range 4–24)	6.04	3.47	4–24

Note. MEBS = Minnesota Eating Behaviors Survey; EAH = Eating in the Absence of Hunger; EES= Emotional Eating Scale

Table 2

Means and Standard Deviations for Co-Twin Difference Scores on Weight Conscious Peer. Group Scales (N=519–629)

Peer Exposure Questionnaires:	**MZ Pairs (N = 322)**	**DZ Pairs (N = 288)**	***t (df)***	***p***	**Cohen's *d***
Perceived Friend Preoccupation with Weight and Dieting	3.47 (4.15)	3.91 (3.57)	−1.26 (493)	0.21	0.11
Appearance Conversation with Friends	2.68 (2.94)	3.30 (3.07)	−2.44 (542.2)	0.02	0.21
Friends as a Source of Influence	2.45 (3.22)	2.82 (3.00)	−1.40 (547)	0.16	0.12
Peer Attribution Scale	1.92 (2.83)	2.36 (2.96)	−1.77 (550)	0.08	0.15

Note. Values are the mean (standard deviation) of the co-twin difference scores for the weight conscious peer group scales. Difference scores were calculated by taking the absolute value of Twin 1-Twin 2.

Table 3

Pearson Correlations examining Within-Person Associations between Weight Conscious Peer Groups and Disordered Eating.

	Weight Conscious Peer Group Measures			
Disordered Eating Measures	**Perceived Friend Preoccupation with Weight and Dieting (N=535–561)**	**Appearance Conversations with Friends (N=569–596)**	**Friends as a Source of Influence (N=566–592)**	**Peer Attribution Scale (N=566–593)**
Minnesota Eating Behavior Survey (MEBS)				
Total Score	0.43**	0.36**	0.43**	0.50**
Weight Preocc.	0.37**	0.31**	0.41**	0.41**
Body Diss.	0.39**	0.31**	0.27**	0.44**
Binge Eating	0.35**	0.29**	0.32**	0.36**
Eating in the Absence of Hunger (EAH)				
Negative Affect	0.27**	0.23**	0.30**	0.26**
External Eating	0.27**	0.22**	0.21**	0.20**
Fatigue/Boredom	0.31**	0.24**	0.23**	0.30**
Emotional Eating Survey (EES)				
Anxiety, Anger and Frustration	0.13*	0.18**	0.22**	0.18**
Depression	0.25**	0.27**	0.25**	0.25**
Unsettled	0.12*	0.11*	0.16**	0.13*

* p< 0.05,

** p<0.01

Table 4

Results from Co-Twin Control Results that Resemble Scenario C (i.e., Genetic and/or Environmental Selection).

	Exposure Questionnaires																			
	Perceived Friend Preoccupation with Weight and Dieting (N=535–561)					Appearance Conversations with Friends (N=569–596)					Friends as a Source of Influence (N=566–592)					Peer Attribution Scale (N=566–593)				
Outcome Measures	**Ind. MZ (β)**	**Ind. DZ (β)**	**Within-Pair MZ (β)**	**Within-Pair DZ (β)**	**Ex x Zyg (β)**	**Ind. MZ (β)**	**Ind. DZ (β)**	**Within-Pair MZ (β)**	**Within-Pair DZ (β)**	**Ex x Zyg (β)**	**Ind. MZ (β)**	**Ind. DZ (β)**	**Within-Pair MZ (β)**	**Within-Pair DZ (β)**	**Ex x Zyg (β)**	**Ind. MZ (β)**	**Ind. DZ (β)**	**Within-Pair MZ (β)**	**Within-Pair DZ (β)**	**Ex x Zyg (β)**
Minnesota Eating Behaviors Survey																				
Total Score	--	--	--	--	--	**0.35***	**0.23***	**0.19***	0.07	0.11	**0.35***	**0.35***	**0.15***	0.09	0.07	--	--	--	--	--
Body Diss	**0.29***	**0.23***	**0.13***	<0.01	0.13	**0.23***	0.14†	0.10	<0.01	0.10	**0.21***	**0.25***	0.08	0.02	0.06	**0.26***	**0.46***	0.07	**0.14***	0.07
Binge Eating	**0.37***	**0.25***	**0.17***	0.06	0.11	--	--	--	--	--	**0.30***	**0.30***	0.11	**0.13***	0.01	**0.18***	**0.43***	0.10	**0.19***	0.09
Weight Preocc	--	--	--	--	--	**0.35***	**0.23***	**0.18***	0.07	0.11	--	--	--	--	--	--	--	--	--	--
Eating in the Absence of Hunger																				
Neg Affect	**0.29***	**0.24***	0.12	0.10	0.03	*0.15*†	**0.23***	0.08	0.13	0.05	**0.28***	**0.27***	0.10	0.09	<0.01	--	--	--	--	--
External Eating	**0.23***	**0.28***	0.01	0.12	0.11	0.16†	**0.24***	0.10	0.07	0.02	**0.16***	**0.26***	<0.01	0.12	0.12	--	--	--	--	--
Fatigue	**0.35***	0.17†	0.14	−0.02	0.15	**.18***	0.16†	0.01	0.06	0.05	**0.20***	**0.21***	0.08	0.04	0.04	--	--	--	--	--
Emotional Eating Scale																				
Anger, Anx, & Frustration	0.16†	0.15†	0.07	0.10	0.03	**0.19***	**0.22***	0.10	0.11	0.01	--	--	--	--	--	--	--	--	--	--
Depression	**0.24***	**0.21***	0.09	0.10	<0.01	**0.18***	**0.27***	0.08	0.12	0.04	--	--	--	--	--	**0.15***	**0.34***	0.10	**0.17***	0.08
Unsettled	0.15†	0.12	0.04	0.07	0.03	--	--	--	--	--	**0.19***	0.17†	0.13	0.10	0.04	--	--	--	--	--

Note. β = fixed effects beta estimates from the mixed linear models that index how effectively exposure to weight conscious peer groups can predict the outcome (i.e., disordered eating) in each set of analysis; "Ind. MZ" = individual-level associations between weight conscious peer groups and disordered eating for MZ pairs; "Ind. DZ" = individual-level associations between weight conscious peer groups and disordered eating for DZ pairs. "Within-Pair MZ" = within-pair association (i.e., the ability of within-pair differences in peer exposure to weight conscious peer groups to predict each twin's level of disordered eating) for MZ pairs; "Within-Pair DZ = within-pair associations for DZ pairs only; "Ex x Zyg" = test of whether there are significant differences in within-pair effects for MZ versus DZ twins.

* $p < .01$,

† $p < .05$

Table 5

Results from Co-Twin Control Results that Resemble Scenario B (Genetic Selection) or Mixture of Scenarios A and C (Socialization and Selection).

	Exposure Questionnaires																			
	Perceived Friend Preoccupation with Weight and Dieting (N=535-561)					Appearance Conversations with Friends (N=569-596)					Friends as a Source of Influence (N=566-592)					Peer Attribution Scale (N=566-593)				
Outcome Measures	Ind. MZ (β)	Ind. DZ (β)	Within-Pair MZ (β)	Within-Pair DZ (β)	Ex x Zyg (β)	Ind. MZ (β)	Ind. DZ (β)	Within-Pair MZ (β)	Within-Pair DZ (β)	Ex x Zyg (β)	Ind. MZ (β)	Ind. DZ (β)	Within-Pair MZ (β)	Within-Pair DZ (β)	Ex x Zyg (β)	Ind. MZ (β)	Ind. DZ (β)	Within-Pair MZ (β)	Within-Pair DZ (β)	Ex x Zyg (β)
Minnesota Eating Behaviors Survey																				
Total Score	**0.42***	**0.29***	**0.20***	0.03	**0.16***	--	--	--	--	--	--	--	--	--	--	**0.35***	**0.44***	**0.16***	**0.17***	<.01
Body Diss	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Binge Eating	--	--	--	--	--	**0.34***	**0.23***	**0.19***	**0.11***	0.08	--	--	--	--	--	--	--	--	--	--
Weight Preocc	**0.39***	**0.29***	**0.18***	0.02	**0.16***	--	--	--	--	--	**0.35***	**0.39***	**0.15***	**0.12***	0.03	**0.36***	**0.34***	**0.20***	**0.12***	0.08
Eating in the Absence of Hunger																				
Neg Affect	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	**0.16***	**0.30***	0.10	**0.16***	0.06
External Eating	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	**0.19***	**0.20***	0.05	**0.15***	0.10
Fatigue	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	**0.25***	**0.31***	0.14	**0.18***	0.04
Emotional Eating Scale																				
Anger, Anx, & Frustration	--	--	--	--	--	--	--	--	--	--	**0.24***	**0.21***	**0.19***	0.11	0.07	.12	**0.29***	0.12	**0.19***	0.07
Depression	--	--	--	--	--	--	--	--	--	--	**0.21***	**0.26***	**0.19***	0.13	0.07	--	--	--	--	--
Unsettled	--	--	--	--	--	0.03	**0.21***	-0.07	**0.15***	**0.22***	--	--	--	--	--	.14	**0.18***	0.03	**0.15***	0.13

Note. β = fixed effects beta estimates from the mixed linear models that index how effectively exposure to weight conscious peer groups can predict the outcome (i.e., disordered eating) in each set of analysis; "Ind. MZ" = individual-level associations between weight conscious peer groups and disordered eating for MZ pairs; "Ind. DZ" = individual-level associations between weight conscious peer groups and disordered eating for DZ pairs; "Within-Pair MZ" = within-pair association (i.e., the ability of within-pair differences in peer exposure to weight conscious peer groups to predict each twin's level of disordered eating) for MZ pairs; "Within-Pair DZ" = within-pair associations for DZ pairs only; "Ex x Zyg" = test of whether there are significant differences in within-pair effects for MZ versus DZ twins. Cells that are outlined with a single line resemble a mix of Scenarios A and C, whereas cells that are outlined with a double line resemble Scenario B. Cells outlined in a dotted line were cases where the interpretation is slightly ambiguous, however, these cases were interpreted as Scenario B.

* $p < .01$