

The Relation Between Body Dissatisfaction and Eating Disorder Symptomatology: An Analysis of Moderating Variables

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Although body dissatisfaction is a strong predictor of disordered eating among women, a majority of women report substantial body dissatisfaction but do not concomitantly report severe levels of eating disorder symptomatology. Third variables, then, may interact with body dissatisfaction to influence its relation to eating disorder symptomatology. Seven theoretically relevant variables were investigated as to whether they moderated this relation within 2 samples ($n = 304$, $n = 373$) of college women. As anticipated, body surveillance, neuroticism, and having a family member and friend with an eating disorder intensified the primary body dissatisfaction–eating disorder symptomatology relation. Contrary to hypotheses, social insecurity and appearance control beliefs did not moderate this relation, and poor impulse regulation weakened the relation between body dissatisfaction and 1 component of eating disorder symptomatology (i.e., drive for thinness).

Although the percentages of women meeting criteria for bona fide clinical eating disorders are rather small (i.e., .5% for anorexia, 1–3% for bulimia, and 2–5% for eating disorder not otherwise specified; *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed.; American Psychiatric Association, 1994; Mulholland & Mintz, 2001), there remains an overwhelming number of young adult women who report engaging in unhealthy weight regulation practices. According to Mintz and Betz (1988), a majority (61%) of college women revealed either occasionally or regularly using extreme measures to control their weight such as fasting, appetite suppressants, diuretics, or purging after eating. More recently, Tylka and Subich (2002a) found that large percentages of high school and college women indicated frequently skipping meals (59.0%), eating fewer than 1,200 calories a day (36.7%), eliminating fats (30.1%) and carbohydrates (26.5%) from their diet, fasting for more than 24 hr (25.9%), using laxatives (7.2%) and diuretics (6.6%), and vomiting after eating (4.8%) specifically to control their weight. Because many women use harmful weight control strategies and do so at various levels, counseling psychologists have focused on preventing and treating the entire spectrum of disordered eating (Hotelling, 2001).

One way that researchers can contribute to the knowledge base for prevention and treatment of disordered eating is to articulate its correlates, protective factors, and risk factors. Fortunately, many concurrent and prospective investigations in this area have begun (e.g., Cattarin & Thompson, 1994; Stice & Agras, 1998) and several factors have been identified. Of these factors, body dissatisfaction is often recognized as the single strongest predictor of

eating disorder symptomatology among women (e.g., Phelps, Johnston, & Augustyniak, 1999; Polivy & Herman, 2002).

Yet, researchers also have found that body dissatisfaction is not unique to individuals with clinical eating disorders and have even suggested that it is so prevalent among women that it is normative (e.g., Mazzeo, 1999; Striegel-Moore, Silberstein, & Rodin, 1986). The following question, then, is raised: Given that high levels of body dissatisfaction are common among women (Klemchuk, Hutchinson, & Frank, 1990), whereas bona fide clinical eating disorders are relatively rare (Striegel-Moore & Cachelin, 2001), could additional variables interact with body dissatisfaction to influence its relation to eating disorder symptomatology? It seems reasonable to argue that certain variables could either accentuate (i.e., in the case of a risk factor) or reduce (i.e., in the case of a protective factor) the strength of the relation between body dissatisfaction and eating disorder symptomatology. An exploration of such moderating variables would address why only a minority of women with high levels of body dissatisfaction report high levels of eating disorder symptomatology.

Investigations in this area seem imperative, as particular variables that weaken or strengthen the body dissatisfaction–eating disorder symptomatology relation would be highlighted. This information would be useful to clinicians, as they would understand which particular variables to assess in order to identify those women, out of the many women with high levels of body dissatisfaction, who are more likely to demonstrate high levels of eating disorder symptomatology. Similarly, once aware of the variables that moderate this relation, professionals can work to prevent high levels of variables that intensify this relation and low levels of variables that buffer this relation. This area of research also attends to requests for researchers to move beyond examining independent correlates of disordered eating toward exploring how variables interact to predict eating disorder symptomatology (Kashubeck-West & Mintz, 2001; Mazzeo & Espelage, 2002). Articulating variables that moderate the body dissatisfaction–eating disorder symptomatology relation, then, would contribute to the literature examining multivariate conceptualizations of eating disorders.

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Curiously, even though professionals have declared a need to investigate variables that interact with body dissatisfaction to predict eating disorder symptomatology (Striegel-Moore et al., 1986), only one published empirical study to date (i.e., Twamley & Davis, 1999) has addressed this area of inquiry by exploring whether self-esteem moderates this relation among college women. The purpose of the present study, then, was to attend to this opening in the research literature by examining whether several additional theoretically relevant variables moderate the body dissatisfaction–eating disorder symptomatology relation.

Study 1

When considering which particular variables could moderate the body dissatisfaction–eating disorder symptomatology relation, it seems logical to explore characteristics thought to intensify women's likelihood of engaging in behavioral (e.g., bingeing, purging, pursuit of thinness) features of eating disorders when dissatisfied with their body shape and size. The general tendency to engage in self-destructive acts and beliefs that others cannot be relied on during times of stress have been argued as such variables that could strengthen the relation between body dissatisfaction and women's disordered eating behaviors (Striegel-Moore et al., 1986). Poor impulse regulation and social insecurity, then, were examined as moderators of this relation. Theoretical support and specific hypotheses for these potential moderators are discussed.

Poor Impulse Regulation

Poor impulse regulation reflects the tendency to engage in impulsive, self-destructive, reckless, and hostile acts (Garner, 1991). Researchers have found that college women without eating disorders scored significantly lower than women with anorexia and bulimia on a measure of poor impulse regulation (Sohlberg, Norring, Holmgren, & Rosmark, 1989; Tylka & Subich, 1999). In the present study, it was expected that higher levels of poor impulse regulation would intensify the relation between body dissatisfaction and eating disorder symptomatology. This hypothesis was derived from professionals' (e.g., Garner, 1991) suggestions that women with high levels of both body dissatisfaction and poor impulse regulation may be more likely to engage in disordered eating, as these women may be more likely to attempt self-destructive behaviors (i.e., harmful weight control techniques characteristic of clinical eating disorders) to change their body shape and size. Similarly, in a landmark article articulating risk factors of eating disorders, Striegel-Moore et al. (1986) posited that poor impulse regulation could, in part, explain why some women with high levels of body dissatisfaction report eating disorder symptomatology, whereas other women with high levels of body dissatisfaction do not demonstrate disordered eating.

Social Insecurity

It also was anticipated in the present study that social insecurity (i.e., beliefs that social relationships are disappointing, unrewarding, tense, and generally of poor quality) would strengthen the relation between body dissatisfaction and eating disorder symptomatology. This hypothesis was proposed given the theoretical literature stating that perceptions of security in social relationships

could protect women against psychological distress in general (e.g., Krause, 1986) and eating disorder symptomatology in particular (Berndt & Hestenes, 1996). Relationships perceived as positive and of high quality are proposed to play a salient role in psychological and physical health by helping people maintain a positive outlook and engage in health-promoting behaviors during times of self-doubt and stress (Kessler & McLeod, 1985). Therefore, women who report high body dissatisfaction and who perceive their relationships as poor in quality, tense, insecure, disappointing, and unrewarding may be more likely to allow negative thoughts and feelings about themselves to influence their eating behaviors and health in a negative way.

The first study, then, served to extend the literature on eating disorders by testing whether poor impulse regulation and social insecurity each would intensify the relation between body dissatisfaction and eating disorder symptomatology. In this study, eating disorder symptomatology was operationalized as drive for thinness and bulimic behaviors (i.e., binge eating, purging), as these components are viewed as critical in defining eating disorder symptomatology (Garner, 1991). For each test of moderation, it was expected that the interactions would be enhancing (i.e., in which both body dissatisfaction and the moderating variable independently affect eating disorder symptomatology in a positive direction and together have a stronger than additive effect; Frazier, Tix, & Barron, 2004). Given that effect sizes for interactions are typically small and rarely larger than moderate in size (i.e., Chaplin, 1991), it was anticipated that these interactions would contribute a small to moderate amount of incremental variance in eating disorder symptomatology.

Method

Participants and Procedure

College women ($n = 304$) enrolled in introductory and upper level psychology classes from two large midwestern universities, who ranged in age from 17 to 49 years ($M = 22.2$, $SD = 6.4$), participated. Freshmen were the largest group of participants (38.8%); of the remaining women, 27.6% were sophomores, 11.8% were juniors, 14.8% were seniors, and 6.9% were postbaccalaureate students. The majority of the participants were White (85.9%); 9.5% identified as African American, 3.6% identified as Asian American, 1 participant [.3%] identified as Latina, and 2 participants [.6%] identified as multiracial. Most of the sample (68%) endorsed the middle-class socioeconomic status category; 13% identified as working class, 14% identified as upper middle class, 3% identified as upper class, and 2% did not respond.

Participants were recruited from posted flyers targeting undergraduate psychology classes or verbal announcements of the experiment within their psychology classes. Women were tested in small groups of 2–15 participants. Prior to receiving the questionnaire packet, they were told that the study's purpose was to examine the associations between their eating habits, body attitudes, and other personality characteristics. Participants were instructed to fill out the entire Eating Disorder Inventory-2 (EDI-2; Garner, 1991) to avoid confusion as to which items to answer. After signing a consent form, they filled out a short demographic questionnaire and then completed the EDI-2. In exchange for their participation, all women received extra credit applied toward their psychology classes that was awarded by the psychology departments' organized research programs.

Instrument

EDI-2 (Garner, 1991). The EDI-2 is a 91-item self-report measure containing 11 subscales. Two subscales (i.e., Drive for Thinness and Bulimia) are used to determine behavioral eating disorder symptomatology, and the remaining 9 subscales do not assess behavioral symptoms of eating disorders but measure personality variables correlated with eating disturbances (for a full review of the EDI-2 subscales, see Garner, 1991). It is recommended by Garner (1991) that researchers and practitioners use each of the 11 subscales individually and do not combine them to form an overall index of eating pathology, as each subscale represents a separate construct. Indeed, the average intercorrelation between these subscales is .33 among college women and .26 among women with eating disorders, suggesting that they assess relatively distinct constructs (Garner, 1991). As a result, it is commonplace for scholars (e.g., Brookings & Wilson, 1994; Pike, 1995; Tylka & Subich, 1999, 2002b) to have each subscale represent an independent construct within their research.

For the present study, select EDI-2 (Garner, 1991) subscales were used to assess participants' levels of the predictor variable body dissatisfaction, the moderating variables (i.e., difficulty controlling impulses and social insecurity),¹ and the criterion variables representative of eating disorder symptomatology (i.e., drive for thinness and bulimia). For all subscales, respondents rated each item on a 6-point scale ranging from *always true of me to never true of me*. Consistent with EDI-2 scoring criteria (Garner, 1991), the responses "never true of me," "rarely true of me," and "sometimes true of me" received a score of 0, and the responses "often true of me," "usually true of me," and "always true of me" received scores of 1, 2, and 3, respectively. Raw scores were summed and converted into percentile ranks as specified in the EDI-2 manual (Garner, 1991).

Body dissatisfaction. The Body Dissatisfaction subscale contains 9 items assessing women's levels of dissatisfaction with their overall body shape (i.e., "I am satisfied with the shape of my body" [reverse scored]) as well as specific body parts (e.g., "I think my stomach is too big"). Higher scores are indicative of greater body dissatisfaction. Among college women, scores on the Body Dissatisfaction subscale have garnered evidence of internal consistency reliability ($\alpha = .91$; Brookings & Wilson, 1994) and test-retest reliability over a 3-week period ($r = .97$; Wear & Pratz, 1987), and are related to other measures of body dissatisfaction (e.g., Body Shape Questionnaire; $r = .82$; Garner, 1991). For the present sample, alpha was .91.

Moderators. The Impulse Regulation subscale (11 items) measures impulsivity, self-destructiveness, recklessness, and substance abuse. A sample item is "I am prone to outbursts of anger or rage." Higher scores reflect poorer impulse regulation. Among samples of college women, it demonstrates adequate internal consistency reliability ($\alpha = .82$; Tylka & Subich, 1999) and is related to measures similar in content (i.e., impulsivity items on the Borderline Syndrome Index; $r = .59$; Garner, 1991). Other reliability and validity information has not been reported for scores on this subscale among nonclinical samples of women. Alpha was .82 among the present sample.

The 8-item Social Insecurity subscale was used to assess women's perceptions of feeling insecure within their relationships. Overall, this subscale measures the belief that relationships are disappointing and poor in quality (e.g., "People understand my real problems" [reverse scored]). Higher scores indicate greater levels of social insecurity. Its internal consistency reliability has been adequate among samples of college women ($\alpha = .80$), and it is related with items assessing relationship quality on the Borderline Syndrome Index ($r = .70$; Garner, 1991). Other reliability and validity information has not been reported for scores on this subscale among college women. For the present study, alpha was .80.

Eating disorder symptomatology. Two subscales (i.e., Drive for Thinness and Bulimia) were used to assess eating disorder symptomatology, as they have been found to reflect an eating disorders factor (Klemchuk et al., 1990). The Drive for Thinness subscale measures the core psychopathology of both anorexia and bulimia (Garner, 1991): attitudes and behaviors

reflecting an individual's motivation to become thinner. It contains 7 items (e.g., "I eat sweets and carbohydrates without feeling nervous" [reverse scored]). Higher subscale scores are indicative of a higher drive for thinness. It has garnered evidence of internal consistency reliability ($\alpha = .90$) and test-retest reliability over a 3-week period ($r = .92$; Wear & Pratz, 1987). Alpha was .87 for the present sample of participants. Brookings and Wilson (1994) found the relation between this subscale and the Eating Attitudes Test-26 (EAT-26) to be .84, supporting its convergent validity.

The Bulimia subscale reflects the tendency to engage in bingeing and purging behaviors common in bulimia and some types of anorexia. It contains 7 items such as "I have gone on eating binges where I felt that I could not stop." Higher subscale scores reflect greater levels of bulimic symptomatology. Scores on this subscale have been shown to be internally consistent ($\alpha = .86$; Brookings & Wilson, 1994) and reliable over a 3-week period ($r = .90$; Wear & Pratz, 1987). For the present sample, alpha was .85. Although this subscale solely examines behaviors related to bingeing and purging, it is correlated with other measures of general eating disorder symptomatology, such as the EAT-26 ($r = .55$; Brookings & Wilson, 1994). It also is strongly related to EAT-26 items that reflect bulimic behaviors ($r = .72$; Garner, 1991).

Results

Means, standard deviations, and intercorrelations for the examined variables are presented in Table 1. Data were screened for bivariate outliers in the relations between (a) body dissatisfaction and drive for thinness and (b) body dissatisfaction and bulimia. Mahalanobis distance was in the acceptable range for all cases. Therefore, no case was excluded.

I tested whether each potential moderator variable interacted with body dissatisfaction to predict eating disorder symptomatology (i.e., drive for thinness and bulimia) using hierarchical moderated regression (HMR; Evans, 1991). This analysis has been argued (e.g., Aiken & West, 1991; Frazier et al., 2004) as the preferred statistical strategy for identifying the presence and nature of moderating effects. For HMR, the sample size of this study (i.e., $n = 304$) was sufficient to detect a small to moderate effect size (i.e., $\Delta R^2 = .02-.15$), with an alpha level of .05 and a power of .80 (Cohen, 1992). As recommended (e.g., Cronbach, 1987), scale scores for the predictor and moderator variables were centered to reduce multicollinearity between the main effect and interaction terms.

The potential moderators were analyzed first individually (i.e., within separate analyses) and then collectively (i.e., combined within the same analysis) to determine whether they moderated the body dissatisfaction-eating disorder symptomatology relation. Both types of analyses were thought to provide useful information for the present study. Because of very limited knowledge of particular variables that moderate the body dissatisfaction-eating disorder symptomatology relation, it first seems important to conduct individual analyses to gain insight into which variables moderate this primary relation. If combined analyses were performed exclusively, potential interactions may be overlooked because of their overlap with predictors and interactions on the criterion (Tabachnick & Fidell, 1996). Yet, in conjunction with individual

¹ Other subscales assessed by the EDI-2 (Garner, 1991) were not examined as moderators of the body dissatisfaction-eating disorder symptomatology relation because no theoretical rationale has been offered for arguing why they would moderate this relation.

Table 1
Study 1 Means, Standard Deviations, and Correlations for EDI-2 Subscales Assessing Eating Disorder Symptomatology, Body Dissatisfaction, and Proposed Moderators (n = 304)

Variable	1	2	3	4	5
1. Drive for Thinness	—				
2. Bulimia	.44*	—			
3. Body Dissatisfaction	.74*	.41*	—		
4. Impulse Regulation	.33*	.30*	.23*	—	
5. Social Insecurity	.31*	.32*	.26*	.62*	—
<i>M</i>	61.46	65.71	62.49	63.65	57.34
<i>SD</i>	26.37	27.44	28.98	21.18	27.20

Note. The means and standard deviations presented were derived from the noncentered variables. EDI-2 = Eating Disorder Inventory-2.

* $p < .01$.

analyses, combined analyses would underscore the particular interactions that contribute unique variance in eating disorder symptomatology when other predictors and interactions are considered.

For the individual analyses, the predictor variable at Step 1 was body dissatisfaction. The proposed moderator variable was entered as a predictor at Step 2 of the analysis. Next, at Step 3, the predictor was an interaction term reflecting the product of body dissatisfaction and the proposed moderating variable (i.e., Body Dissatisfaction \times Poor Impulse Regulation, or Body Dissatisfaction \times Social Insecurity). Separate analyses were conducted for each criterion variable representing eating disorder symptomatology (i.e., drive for thinness, bulimia). The criterion variables and steps for the combined analyses were similar to the individual analyses. The only differences were that, for the combined analyses, all moderator variables were entered at Step 2 and all interactions were entered at Step 3. Evidence for a moderator effect is noted at Step 3 by a statistically significant increment in R^2 (i.e., ΔR^2) and beta weight. Because regression analyses are sensitive to sample size (McClelland & Judd, 1993), the size of the ΔR^2 value also was evaluated. Following the recommendations of Cohen (1992), it was determined that ΔR^2 values of .02 and above would signify unique contributions to the overall variance. Table 2 presents the findings for these analyses.

Individual HMR Analyses

Poor impulse regulation. Unexpectedly, poor impulse regulation did not moderate the relation between body dissatisfaction and bulimic symptomatology ($\beta = -.04$, $t(303) = -0.68$, *ns* ($\Delta R^2 = .00$). Poor impulse regulation moderated the relation between body dissatisfaction and drive for thinness ($\beta = -.11$, $t(303) = -2.64$, $p < .01$; however, this effect was very small ($\Delta R^2 = .01$) and was in an opposite direction than expected. Poor impulse regulation, therefore, slightly weakened rather than strengthened the relation between body dissatisfaction and drive for thinness. This finding was particularly surprising given that both body dissatisfaction ($\beta = .74$, $t(303) = 19.29$, $p < .01$, and poor impulse regulation ($\beta = .17$, $t(303) = 4.29$, $p < .01$, as independent predictors, were found to predict higher levels of drive for thinness.

The regression slopes of this significant interaction were plotted in a graph (see Figure 1) using predicted values for drive for thinness calculated from representative groups at the mean, 1

standard deviation above the mean, and 1 standard deviation below the mean on body dissatisfaction and poor impulse regulation. Following convention (e.g., Aiken & West, 1991; Frazier et al., 2004), these predicted values were obtained by multiplying the respective unstandardized regression coefficients for each centered variable by its appropriate value (i.e., 1 standard deviation or -1 standard deviation of the predictor for the first term, 1 standard deviation or -1 standard deviation of the moderator for the second term, and the product of the standard deviations of the predictor and moderator for the interaction term), summing these products, and then adding the constant value. An analysis of the significance of the simple slopes (Aiken & West, 1991) showed that body dissatisfaction strongly predicted drive for thinness for women 1 standard deviation below the mean on poor impulse regulation, producing a very large effect ($\beta = .80$, $t(303) = 15.38$, $p < .001$). Body dissatisfaction still predicted drive for thinness for women 1 standard deviation above the mean on poor impulse regulation; however, the effect was large rather than very large ($\beta = .61$, $t(303) = 11.53$, $p < .001$).

Social insecurity. Contrary to hypotheses, social insecurity was not found to interact with body dissatisfaction when predicting drive for thinness ($\beta = -.06$, $t(303) = -1.47$, *ns* ($\Delta R^2 = .00$) or bulimic symptomatology ($\beta = -.07$, $t(303) = -1.37$, *ns* ($\Delta R^2 = .00$).

Combined HMR Analyses

When combined into the same analysis, both poor impulse regulation ($\beta = -.10$, $t(303) = -1.78$, *ns*, and social insecurity ($\beta = -.02$, $t(303) = -0.34$, *ns*, did not moderate the relation between body dissatisfaction and drive for thinness ($\Delta R^2 = .01$). Similarly, when examined together, poor impulse regulation ($\beta = .04$, $t(303) = 0.57$, *ns*, and social insecurity ($\beta = -.12$, $t(303) = -1.69$, *ns*, did not moderate the relation between body dissatisfaction and bulimia ($\Delta R^2 = .01$).

Study 2

Other variables not considered in the first study have been articulated within the theoretical literature (e.g., Striegel-Moore et al., 1986) as possible moderators of the relation between body dissatisfaction and eating disorder symptomatology. In this second

Table 2

Study 1 Hierarchical Multiple Regression Analyses Predicting Eating Disorder Symptomatology (Drive for Thinness, Bulimia) From Body Dissatisfaction, Hypothesized Moderator Variables, and Interactions (n = 304)

Variable	<i>B</i>	<i>SE B</i>	β	Cumulative R^2	Adj. R^2	Incremental R^2	t^a
Dependent variable: Drive for thinness							
Step 1							
Body dissatisfaction (BD)	2.24	0.12	.74	.55	.55	.55	19.29*
Step 2							
Impulse regulation (IR)	1.10	0.26	.17	.57	.57	.02	4.29*
Step 3							
BD \times IR interaction	-0.07	0.03	-.11	.59	.58	.01	-2.64*
Overall $F(3, 300) = 142.42^*$							
Dependent variable: Bulimia							
Step 1							
Body dissatisfaction (BD)	0.81	0.11	.41	.16	.16	.16	7.69*
Step 2							
Impulse regulation (IR)	0.97	0.23	.22	.21	.21	.05	4.21*
Step 3							
BD \times IR interaction	-0.01	0.03	-.04	.21	.20	.00	-0.68
Overall $F(3, 300) = 26.81^*$							
Dependent variable: Drive for thinness							
Step 1							
Body dissatisfaction (BD)	2.24	0.12	.74	.55	.55	.55	19.29*
Step 2							
Social insecurity (SI)	1.00	0.32	.13	.57	.57	.02	3.27*
Step 3							
BD \times SI interaction	-0.05	0.03	-.06	.57	.57	.00	-1.47
Overall $F(3, 300) = 132.85^*$							
Dependent variable: Bulimia							
Step 1							
Body dissatisfaction (BD)	0.81	0.11	.41	.16	.16	.16	7.69*
Step 2							
Social insecurity (SI)	1.16	0.27	.23	.22	.22	.06	4.26*
Step 3							
BD \times SI interaction	-0.04	0.03	-.07	.22	.22	.00	-1.37
Overall $F(3, 300) = 27.57^*$							
Dependent variable: Drive for thinness							
Step 1							
Body dissatisfaction (BD)	2.11	0.12	.74	.55	.55	.55	19.29*
Step 2							
Impulse regulation (IR)	0.92	0.32	.14	.58	.58	.03	2.88*
Social insecurity (SI)	0.35	0.38	.05				0.92
Step 3							
BD \times IR Interaction	-0.06	0.04	-.10	.59	.58	.01	-1.77
BD \times SI Interaction	-0.02	0.04	-.02				-0.34
Overall $F(5, 298) = 85.38^*$							
Dependent variable: Bulimia							
Step 1							
Body dissatisfaction (BD)	0.81	0.11	.41	.16	.16	.16	7.69*
Step 2							
Impulse regulation (IR)	0.60	0.29	.14	.22	.22	.06	2.08*
Social insecurity (SI)	0.74	0.34	.14				2.18*
Step 3							
BD \times IR interaction	0.02	0.03	.04	.23	.22	.01	.57
BD \times SI interaction	-0.07	0.04	-.12				-1.69
Overall $F(5, 298) = 17.91^*$							

Note. Adj. = adjusted.

^a $df = 303$.

* $p < .05$.

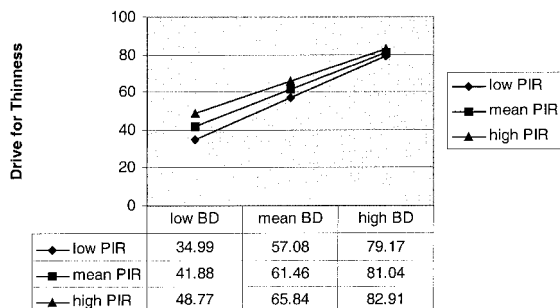


Figure 1. Study 1: Plot of significant Body Dissatisfaction × Poor Impulse Regulation interaction. BD = body dissatisfaction; PIR = poor impulse regulation; low = predicted value for women 1 standard deviation below the mean; mean = predicted value for women at the mean; high = predicted value for women 1 standard deviation above the mean.

study, I therefore sought to explore whether an additional five variables (i.e., body surveillance and appearance control beliefs [reflecting objectified body consciousness], neuroticism, and presence of a family member and friend with an eating disorder) moderated this relation. Furthermore, the second study addressed two limitations of the first study. A more global measure of eating disorder symptomatology (i.e., EAT-26; Garfinkel & Garner, 1979) was used in the second study in lieu of two separate indicators of eating disorder symptomatology (EDI-2 Drive for Thinness and Bulimia subscales; Garner, 1991). Although these two EDI-2 subscales have adequate psychometric evidence for their use as measures of eating disorder symptomatology, they were not strongly related to one another (i.e., $r = .44$), and it is not recommended that they be combined into a single measure (Garner, 1991). The EAT-26 has psychometric evidence for its use as a single measure of eating disorder symptomatology among college women (e.g., Mazzeo, 1999). Also, in the second study, subscales of the same instrument were not used to assess body dissatisfaction, the hypothesized moderating variables, and eating disorder symptomatology. The results of the first study may have been confounded by the overlapping variance caused by using subscales of the same instrument to assess the predictors, moderators, and criterion variables. A rationale of why and how each potential moderating variable was thought to interact with body dissatisfaction when predicting eating disorder symptomatology (i.e., EAT-26 scores) is presented.

Objectified Body Consciousness

Objectified body consciousness is defined as the degree to which a woman internalizes an outsider’s perspective of herself and focuses more on observable attributes (e.g., appearance) rather than internal attributes (e.g., feelings), and it is often manifested in women using a considerable amount of energy to monitor their appearance (McKinley & Hyde, 1996). I predicted that two characteristics of objectified body consciousness (i.e., body surveillance and beliefs that individuals can change their appearance) would strengthen the relation between body dissatisfaction and eating disorder symptomatology. The other recognized component of objectified body consciousness (i.e., body shame) was not

investigated, as it was thought to overlap conceptually with body dissatisfaction.

The rationale for investigating whether body surveillance and appearance control beliefs moderate the body dissatisfaction–eating disorder symptomatology relation was based on feminist literature (e.g., Fallon, Katzman, & Wooley, 1994; Maine, 2000). According to these writings, the relation between body dissatisfaction and eating disorder symptomatology can be explained, at least in part, by the extent to which women internalize objectifying messages present in Western culture that relate to body size and shape. Two such messages are that (a) women should base their self-worth on their external appearance rather than internal characteristics (e.g., feelings, personality, intellect, body functionality) and need to constantly monitor their appearance for this reason, and (b) women’s bodies can be treated as objects to be molded into society’s thin-ideal prototype.

Specifically, among women with high levels of body dissatisfaction, those who constantly monitor their bodies, are more concerned about how their bodies appear to others, and are more likely to think of their bodies as objects will spend more time being aware of how much they dislike their bodies (Fredrickson & Roberts, 1997). Because these women have less regard for their bodies, they may minimize the danger of using weight control techniques that are harmful and use these strategies in an attempt to lose weight when dissatisfied with their weight or shape (Striegel-Moore et al., 1986). Furthermore, among women with high levels of body dissatisfaction, those who view their bodies as objects that can be molded into the thin-ideal societal stereotype may be more likely to use harmful weight control techniques, as they believe their bodies can be changed (McKinley & Hyde, 1996). In contrast, women who have high levels of body dissatisfaction and do not believe their bodies can be altered would be less likely to use these weight control techniques.

Neuroticism

Neuroticism also was investigated as a possible moderator of the body dissatisfaction–eating disorder symptomatology relation, such that it was expected to intensify this relation. The rationale for exploring neuroticism as a moderator was derived, first, from scholars who have declared that neuroticism has the greatest utility in psychological research as a moderator variable used alongside other variables when predicting psychological disorders (Claridge & Davis, 2001). With many psychological disorders, it is suggested that neuroticism modulates the role of other personality influences in behavior (i.e., the “bad mood” that constitutes high neuroticism may exacerbate the negative elements in other features of the individual and thus intensify maladaptive behaviors characteristic of psychological disorders; Claridge & Davis, 2001, p. 395). Furthermore, specialists in eating disorders (Striegel-Moore et al., 1986) have proposed that features of neuroticism (e.g., affective instability, a fragile sense of self) could differentiate women with high levels of both body dissatisfaction and eating disorder symptomatology from women with high levels of body dissatisfaction who do not engage in disordered eating.

Presence of Family Member and Friend With an Eating Disorder

Last, the presence of a family member and friend with an eating disorder each was expected to strengthen the relation between body dissatisfaction and eating disorder symptomatology within the present study. Reasoning for examining these variables as moderators was based, in part, on the premise of social cognitive theory (Bandura, 1986). According to this theory, people often model the behaviors learned from watching or listening to others they view as similar to them. Women with high levels of body dissatisfaction who interact with family members or friends who engage in eating disordered behaviors may learn these behaviors and use them in an attempt to change their body size or shape. Indeed, research has found that exposure to individuals with clinical eating disorders has been found to worsen eating disorder symptomatology among women with high levels of body dissatisfaction. For instance, some eating disorder prevention and treatment programs, including discussions of eating disordered behaviors, actually have been found to worsen disordered eating, as members reported learning new strategies for weight loss from other group members (Mann, 1995). This hypothesis also was specified, as it is consistent with theory (i.e., Striegel-Moore et al., 1986) that suggests that the presence of family members and friends who model weight preoccupation and disordered eating may strengthen the relation between body dissatisfaction and eating disorder symptomatology among women.

The second study, then, explored whether body surveillance, appearance control beliefs, neuroticism, and the presence of a family member and friend with an eating disorder each strengthened the body dissatisfaction–eating disorder symptomatology relation. For each test of moderation, body dissatisfaction and the proposed moderating variable were expected to predict eating disorder symptomatology in a positive direction and together have a stronger than additive effect (i.e., producing an enhancing interaction). These interactions were anticipated to predict a small to moderate amount of incremental variance in eating disorder symptomatology, because significant interactions, when they occur, are most often in this range (Chaplin, 1991).

Method

Participants and Procedure

The sample included 373 women who ranged in age from 17 to 58 years ($M = 23.74$, $SD = 7.69$). Women were recruited from either undergraduate psychology classes ($n = 290$) or campus sororities ($n = 83$) at two large midwestern universities. Nineteen percent were freshmen, 22.8% were sophomores, 29.0% were juniors, 24.7% were seniors, and 4.6% were either postbaccalaureate or graduate students. Most of the participants identified as White (79.9%), followed by African American (9.9%), Latina (5.9%), Asian American (1.9%), Native American (.5%), and multiracial (.3%); 6 participants (1.6%) left the question blank. In terms of socioeconomic status, 55.7% endorsed middle class, 25.1% endorsed upper middle class, 16.9% endorsed working class, and 2.2% endorsed upper class.

Psychology students were recruited via posted flyers advertising the experiment outside of a psychology research lab or through verbal announcements given in their undergraduate psychology class. Sorority women were recruited at the beginning of their chapter meeting. Prior to their participation, women were told that the purpose of the study was to examine the associations between eating habits and other psychological

variables. They either filled out the packet of questionnaires in small groups of 2–10 participants (99% returned it fully completed), completed the packet during their sorority chapter meeting (96% returned it fully completed), or took the packet home and brought it back to the experimenter (93% returned it fully completed). Participants in psychology classes received extra credit that was applied toward their class grade. This credit was awarded via a research program organized through the university's Psychology department. Sororities were awarded a small monetary donation (\$3) per completed packet.

Instruments

Body dissatisfaction. The EDI-2 Body Dissatisfaction subscale (Garner, 1991) was used to assess participants' levels of body dissatisfaction. This 9-item measure was described in depth in the first study. For this sample of women, the alpha was .92.

Moderators. The first two moderating variables, body surveillance and appearance control beliefs, were assessed via two subscales of the Objectified Body Consciousness scale (OBC; McKinley & Hyde, 1996) that reflect these constructs. The 8-item Body Surveillance subscale measures the degree to which a woman watches her body and thinks of her body in terms of how it appears to others rather than how it feels or functions. It does not examine body dissatisfaction. Items (e.g., "I think that it is more important that my clothes are comfortable than whether they look good on me" [reverse scored]) are rated on a scale ranging from 1 (*strongly disagree*) to 7 (*strongly agree*) and averaged. Higher scores indicate greater levels of body surveillance. For this subscale, McKinley and Hyde (1996) reported an alpha of .89, evidence that participants respond in a consistent fashion over a 2-week period ($r = .79$), and support for its relation to a measure of similar content (i.e., public self-consciousness, $r = .73$). For the present study, alpha was .88.

The Appearance Control Beliefs subscale of the OBC (McKinley & Hyde, 1996) contains 8 items that reflect the assumption that women, given enough effort, can comply with cultural standards to control how their bodies look. A sample item is "I can weigh what I'm supposed to when I try hard enough." Items are averaged; higher scores reflect the belief that appearance can be more easily controlled. This subscale has demonstrated evidence of internal consistency reliability (i.e., $\alpha = .72$), test–retest reliability over a 2-week period ($r = .73$), and was related to several independent measures of appearance control behaviors such as dieting ($r = .38$) and wearing makeup ($r = .22$), supporting its construct validity (McKinley & Hyde, 1996). For the present sample of women, alpha was .76.

Neuroticism was assessed using the Neuroticism subscale of the NEO-Five Factor Inventory (NEO-FFI; Costa & McCrae, 1992). This subscale contains 12 items that are each scored on a 5-point scale ranging from 0 (*strongly disagree*) to 4 (*strongly agree*). A sample item is "I am not a worrier" (reverse scored). Raw scores were transformed into *T* scores as specified in the NEO-FFI manual. Higher scores are indicative of higher levels of neuroticism. Its alpha estimates (.84–.92; Costa & McCrae, 1992; Tylka & Subich, 1999) and test–retest reliability estimates over a 3-month period ($r = .79$; Costa & McCrae, 1992) are adequate. For the present study, its internal consistency alpha reliability was found to be .89. This subscale is related with the revised version of the NEO Personality Inventory Neuroticism domain scale ($r = .89$) and self-report adjective factors of neuroticism ($r = .92$), supporting its convergent validity (Costa & McCrae, 1992).

Presence of a family member and friend with an eating disorder was assessed by asking participants the following two questions specifically designed for this study: "Do you have anyone in your *family of origin* (e.g., sister, mother, aunt, and so forth) who either has or has had an eating disorder?" and "Do you have a *friend* who either has or has had an eating disorder?" Participants indicated either no or yes to these questions.

Eating disorder symptomatology. The EAT-26 (Garfinkel & Garner, 1979) contains 26 items designed to measure salient characteristics of

eating disorder symptomatology within clinical and/or nonclinical populations (Mazzeo, 1999). Sample items are “I am terrified about being overweight” and “I have the impulse to vomit after meals.” Individuals are instructed to rate each item on a continuous scale ranging from 1 (*never*) to 6 (*always*). Responses of never, rarely, and sometimes receive a score of 0, and the responses of often, usually, and always receive scores of 1, 2, and 3, respectively. Items are summed to arrive at a total score (i.e., ranging from 0 to 78). Higher scores are reflective of more severe symptomatology. For samples of college women, alpha estimates have been found to be high (e.g., .91; Mazzeo, 1999), it has been shown to be related to other measures of eating disorder symptomatology such as the Drive for Thinness ($r = .84$) and Bulimia ($r = .55$) subscales of the EDI-2 (Garner, 1991), and it correctly classifies women with eating disorders from those without eating disorders (Garner, Olmsted, Bohr, & Garfinkel, 1982). For the present study, alpha was .89.

Results

Means and standard deviations were calculated for each continuous measure, and their relations with other measures were examined. This information is presented in Table 3. A phi coefficient was used to express the relation between the presence of a family member with an eating disorder and the presence of a friend with an eating disorder, as they are dichotomous variables, and point-biserial coefficients were used to express the relations of these categorical variables with the continuous measures. These coefficients can be interpreted similarly to Pearson product-moment correlations. Twenty-four percent of the women indicated that they have or have had a family member with an eating disorder, and 63% reported that they have or have had a friend with an eating disorder. These variables were dummy coded so that 0 = *no family member (friend) with an eating disorder*, and 1 = *presence of a family member (friend) with an eating disorder*. The assumption of the homogeneity of error variances was met for each categorical variable (Aguinis, Petersen, & Pierce, 1999). Mahalanobis distance was used to screen for bivariate outliers in the relation between body dissatisfaction and eating disorder symptomatology. No case was excluded, as all cases were in the acceptable range.

Similar to Study 1, HMR analyses were used to test whether the proposed moderator variables interacted with body dissatisfaction to predict eating disorder symptomatology. First, individual analyses were performed to observe whether each proposed moderator indeed moderated the primary body dissatisfaction–eating disorder symptomatology relation. Then, all predictors and interactions were examined within a combined analysis to examine the particular interactions that accounted for unique variance in eating disorder symptomatology. In all regression analyses, body dissatisfaction was entered at Step 1, the proposed moderator(s) was entered at Step 2, and the interaction(s) of body dissatisfaction with the proposed moderator(s) was entered at Step 3. Statistically significant ΔR^2 and beta weight(s) at Step 3 were used to determine whether a moderator effect was present. Furthermore, the ΔR^2 values were evaluated on the basis of their size; values of .02 and above are recommended (Cohen, 1992). The sample size of this study (i.e., $n = 373$) was determined to be a sufficient number of participants needed to detect a small to moderate effect size (i.e., $\Delta R^2 = .02-.15$), with an alpha level of .05 and a power of .80. For each analysis, scale scores for the predictor and moderator variables were centered (e.g., Cronbach, 1987) to reduce multicollinearity. Table 4 presents the findings from the individual and combined analyses.

Individual HMR Analyses

Objectified body consciousness-body surveillance. As anticipated, body surveillance intensified the relation between body dissatisfaction and eating disorder symptomatology ($\beta = .25$), $t(372) = 5.97$, $p < .001$; this interaction accounted for 6% (i.e., $\Delta R^2 = .06$) of the overall variance in eating disorder symptomatology (i.e., a small to moderate effect). I plotted the regression slopes of this significant interaction in a graph (see Figure 2) using the predicted values for eating disorder symptomatology estimated from representative groups at the mean, 1 standard deviation above the mean, and 1 standard deviation below the mean on body dissatisfaction and body surveillance (a discussion of how pre-

Table 3
Study 2 Means, Standard Deviations, and Correlations for Measures Assessing Eating Disorder Symptomatology, Body Dissatisfaction, and Proposed Moderators (n = 373)

Variable	1	2	3	4	5	6	7
1. EAT-26	—						
2. EDI-2-Body Dissatisfaction	.46*	—					
3. OBC-Body Surveillance	.54*	.48*	—				
4. OBC-Appearance Control Beliefs	.02	-.04	-.03	—			
5. NEO-FFI Neuroticism	.45*	.37*	.49*	-.24*	—		
6. Family member with ED	.27*	.15*	.16*	.08	.18*	—	
7. Friend with ED	.11*	.02	.05	.01	.12*	.12*	—
<i>M</i>	9.88	63.27	4.75	4.52	54.03	—	—
<i>SD</i>	10.33	27.55	1.15	0.95	11.51	—	—

Note. The means and standard deviations presented were derived from the noncentered variables. As the Family member with ED and Friend with ED variables are nominal categorical variables (each coded as 0 = *no presence of family member [or friend] with ED*, 1 = *presence of family member [or friend] with ED*), the relation between them is expressed as a phi coefficient, their relations with continuous variables are expressed as point-biserial coefficients, and their means and standard deviations were not calculated (as indicated by the dashes). EAT-26 = Eating Attitudes Test-26; EDI-2 = Eating Disorder Inventory-2; OBC = Objectified Body Consciousness scale; NEO-FFI = NEO Five-Factor Inventory; ED = eating disorder.
* $p < .05$.

Table 4

Study 2 Hierarchical Multiple Regression Analyses Predicting Eating Disorder Symptomatology From Body Dissatisfaction, Hypothesized Moderator Variables, and Interactions ($n = 373$)

Variable	<i>B</i>	<i>SE B</i>	β	Cumulative R^2	Adj. R^2	Incremental R^2	t^a
Step 1							
Body dissatisfaction (BD)	3.89	0.39	.46	.21	.21	.21	9.92*
Step 2							
OBC body surveillance (BS)	3.71	0.43	.42	.34	.34	.13	8.61*
Step 3							
BD \times BS interaction	1.59	0.27	.25	.40	.40	.06	5.97*
Overall $F(3, 369) = 81.89^*$							
Step 1							
Body dissatisfaction (BD)	3.89	0.39	.46	.21	.21	.21	9.92*
Step 2							
OBC appearance control (AC)	0.43	0.50	.04	.21	.21	.00	0.85
Step 3							
BD \times AC interaction	-0.34	0.43	-.04	.21	.21	.00	-0.79
Overall $F(3, 369) = 33.19^*$							
Step 1							
Body dissatisfaction (BD)	3.89	0.39	.46	.21	.21	.21	9.92*
Step 2							
Neuroticism (N)	0.37	0.05	.29	.30	.30	.09	7.11*
Step 3							
BD \times N interaction	0.16	0.04	.18	.33	.33	.03	4.03*
Overall $F(3, 369) = 61.68^*$							
Step 1							
Body dissatisfaction (BD)	3.89	0.39	.46	.21	.21	.21	9.92*
Step 2							
Family member w/ED (FAM)	4.95	1.09	.21	.25	.24	.04	4.55*
Step 3							
BD \times FAM interaction	2.29	0.88	.12	.27	.26	.02	2.61*
Overall $F(3, 369) = 44.36^*$							
Step 1							
Body dissatisfaction (BD)	3.89	0.39	.46	.21	.21	.21	9.92*
Step 2							
Friend w/ED (FR)	2.07	0.97	.10	.22	.21	.01	2.11*
Step 3							
BD \times FR interaction	1.72	0.78	.10	.23	.22	.01	2.21*
Overall $F(3, 369) = 36.59^*$							
Step 1							
Body dissatisfaction (BD)	3.89	0.39	.46	.21	.21	.21	9.92*
Step 2							
OBC body surveillance (BS)	2.73	0.45	.31	.41	.40	.20	6.08*
OBC appearance control (AC)	1.06	0.46	.10				2.31*
Neuroticism (N)	0.25	0.06	.22				4.46*
Family member w/ED (FAM)	3.46	0.99	.14				3.49*
Friend w/ED (FR)	0.93	0.87	.04				1.07
Step 3							
BD \times BS interaction	1.10	0.30	.17	.47	.45	.06	3.61*
BD \times AC interaction	0.13	0.37	.02				0.36
BD \times N interaction	0.07	0.04	.08				1.64
BD \times FAM interaction	1.26	0.78	.07				1.61
BD \times FR interaction	1.03	0.67	.06				1.55
Overall $F(11, 361) = 28.70^*$							

Note. Family member w/ED (eating disorder) was dummy coded so that 0 = no family member with an eating disorder, and 1 = the presence of a family member with an eating disorder. Similarly, Friend w/ED was dummy coded so that 0 = no friend with an eating disorder, and 1 = the presence of a friend with an eating disorder. Adj. = adjusted; OBC = objectified body consciousness.

^a $df = 372$.

* $p < .05$.

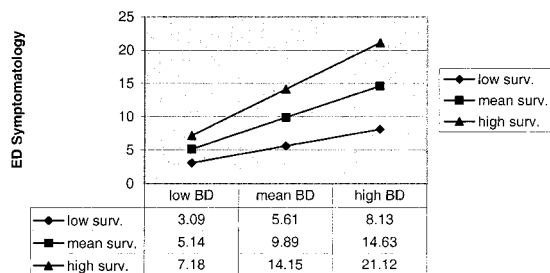


Figure 2. Study 2: Plot of significant Body Dissatisfaction \times Body Surveillance interaction. ED = eating disorder; BD = body dissatisfaction; surv. = body surveillance; low = predicted value for women 1 standard deviation below the mean; mean = predicted value for women at the mean; high = predicted value for women 1 standard deviation above the mean.

dicted values are calculated was presented in detail in Study 1). Testing the significance of the simple slopes (Aiken & West, 1991) indicated that body dissatisfaction did not predict eating disorder symptomatology for women 1 standard deviation below the mean on body surveillance ($\beta = .09$), $t(372) = 1.74$, *ns*; however, body dissatisfaction strongly predicted eating disorder symptomatology for women 1 standard deviation above the mean on body surveillance ($\beta = .52$), $t(372) = 8.17$, $p < .001$.

Objectified body consciousness-appearance control beliefs. Contrary to expectations, appearance control beliefs did not interact with body dissatisfaction to predict eating disorder symptomatology ($\beta = -.04$), $t(372) = -0.79$, *ns* ($\Delta R^2 = .00$).

Neuroticism. Neuroticism was found to strengthen the relation between body dissatisfaction and eating disorder symptomatology ($\beta = .18$), $t(372) = 4.03$, $p < .001$, as expected. This interaction contributed 3% of the unique variance in eating disorder symptomatology ($\Delta R^2 = .03$; a small effect). Figure 3 presents a graph of the regression slopes for predicted values of eating disorder symptomatology calculated from representative groups at the mean, 1 standard deviation above the mean, and 1 standard deviation below the mean on body dissatisfaction and neuroticism. For women 1 standard deviation below the mean on neuroticism, body dissatisfaction slightly predicted eating disorder symptomatology (i.e., producing a small to moderate effect; $\beta = .19$), $t(372) =$

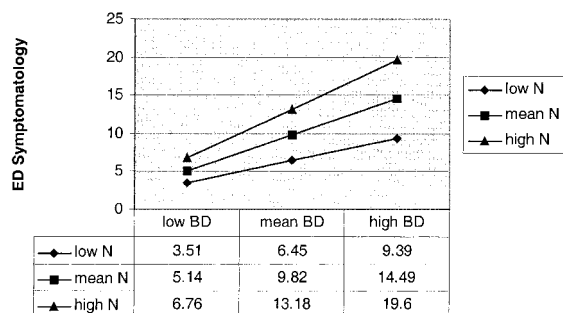


Figure 3. Study 2: Plot of significant Body Dissatisfaction \times Neuroticism interaction. ED = eating disorder; BD = body dissatisfaction; N = neuroticism; low = predicted value for women 1 standard deviation below the mean; mean = predicted value for women at the mean; high = predicted value for women 1 standard deviation above the mean.

3.50, $p < .001$; whereas for women 1 standard deviation above the mean on neuroticism, body dissatisfaction strongly predicted eating disorder symptomatology (i.e., producing a large effect; $\beta = .55$), $t(372) = 7.85$, $p < .001$.

Presence of a family member with an eating disorder. Having a family member who has or has had an eating disorder, as predicted, intensified the relation between body dissatisfaction and eating disorder symptomatology ($\beta = .12$), $t(372) = 2.61$, $p < .01$. This interaction added 2% of the unique variance in eating disorder symptomatology ($\Delta R^2 = .02$), which is considered a small effect. Figure 4 illustrates the plotted eating disorder symptomatology scores for women who do not have a family member with a current or previous eating disorder and women who have a family member with a current or previous eating disorder at the mean, 1 standard deviation above the mean, and 1 standard deviation below the mean on body dissatisfaction. For women who do not have a family member who has or has had an eating disorder, body dissatisfaction moderately predicted eating disorder symptomatology ($\beta = .31$), $t(372) = 4.86$, $p < .001$; whereas for women who have a family member who has or has had an eating disorder, body dissatisfaction strongly predicted eating disorder symptomatology ($\beta = .54$), $t(372) = 8.62$, $p < .001$.

Presence of a friend with an eating disorder. Although having a friend with an eating disorder strengthened the body dissatisfaction and eating disorder symptomatology relation ($\beta = .10$), $t(372) = 2.21$, $p < .05$, the interaction accounted for a minimal amount of incremental variance (i.e., 1%) in eating disorder symptomatology ($\Delta R^2 = .01$). Plotted in Figure 5 are the eating disorder symptomatology scores for women who do not have a friend with a current or previous eating disorder and women who have a friend with a current or previous eating disorder at each level of body dissatisfaction (i.e., mean, 1 standard deviation above the mean, and 1 standard deviation below the mean). Body dissatisfaction moderately predicted eating disorder symptomatology for women who do not have a friend with a current or previous eating disorder ($\beta = .37$), $t(372) = 6.33$, $p < .001$; whereas body dissatisfaction strongly predicted eating disorder symptomatology among women who have a friend with a current or previous eating disorder ($\beta = .57$), $t(372) = 8.31$, $p < .001$.

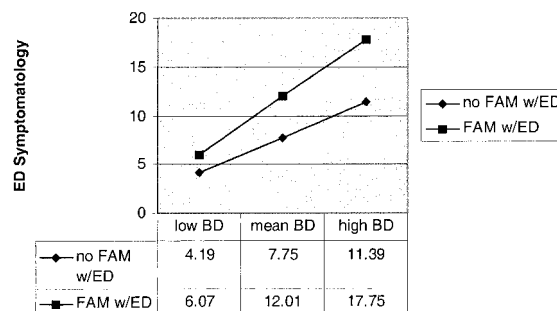


Figure 4. Study 2: Plot of significant Body Dissatisfaction \times Family Member With an Eating Disorder interaction. ED = eating disorder; BD = body dissatisfaction; FAM = family member; low = predicted value for women 1 standard deviation below the mean; mean = predicted value for women at the mean; high = predicted value for women 1 standard deviation above the mean.

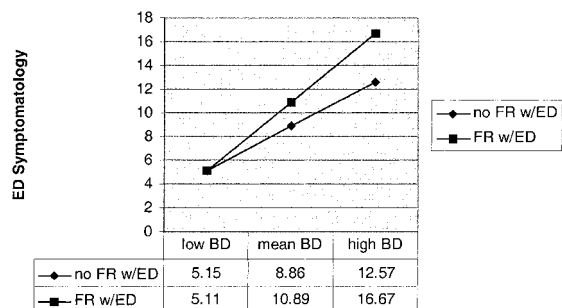


Figure 5. Study 2: Plot of significant Body Dissatisfaction \times Friend With an Eating Disorder interaction. ED = eating disorder; BD = body dissatisfaction; FR = friend; low = predicted value for women 1 standard deviation below the mean; mean = predicted value for women at the mean; high = predicted value for women 1 standard deviation above the mean.

Combined HMR Analysis

When all predictors were entered into Step 2 and all interactions were entered into Step 3 of an HMR analysis, the only interaction that was found to predict unique variance in eating disorder symptomatology was body surveillance ($\beta = .17$), $t(372) = 3.61$, $p < .001$. That is, appearance control beliefs ($\beta = .02$), $t(372) = 0.36$, ns ; neuroticism ($\beta = .08$), $t(372) = 1.64$, ns ; and the presence of a family member ($\beta = .07$), $t(372) = 1.61$, ns , and friend ($\beta = .06$), $t(372) = 1.55$, ns , with an eating disorder did not moderate the relation between body dissatisfaction and eating disorder symptomatology when all predictors and interactions were analyzed.

General Discussion

The present study investigated a perplexing question raised in the theoretical literature: Which variables moderate the relation between body dissatisfaction and eating disorder symptomatology? Identifying such moderating variables is essential, as this knowledge would help professionals understand which women with high levels of body dissatisfaction have an increased (or decreased) tendency to display clinical eating disorder symptomatology. Within two studies, a total of seven theoretically relevant variables were explored as to whether they intensified the primary body dissatisfaction–eating disorder symptomatology relation. Three variables (body surveillance, neuroticism, presence of family member with an eating disorder) received solid support as moderators of this relation, as they interacted with body dissatisfaction to account for 6%, 3%, and 2% of the criterion variance, respectively. As expected, these variables strengthened this relation; body dissatisfaction either did not predict or predicted a small to moderate amount of variance in eating disorder symptomatology for women 1 standard deviation below the mean on these moderating variables, but body dissatisfaction predicted a large amount of variance in eating disorder symptomatology for women 1 standard deviation above the mean on these moderating variables. One additional variable (the presence of a friend with an eating disorder) strengthened this relation, but this interaction contributed a minimal amount of the variance (i.e., 1%) in eating disorder symptomatology. Interestingly, poor impulse regulation

weakened the relation between body dissatisfaction and one component of eating disorder symptomatology (i.e., drive for thinness); however, this interaction accounted for only 1% of the criterion variance. Contrary to predictions, social insecurity and appearance control beliefs did not interact with body dissatisfaction to predict eating disorder symptomatology, and poor impulse regulation did not moderate the relation between body dissatisfaction and bulimia.

Therefore, of the variables examined, it appears that body surveillance received the strongest support for intensifying the body dissatisfaction–eating disorder symptomatology relation. The influence of body surveillance as a stable moderator of this relation is further highlighted by the fact that only body surveillance interacted with body dissatisfaction to predict unique variance in eating disorder symptomatology when all predictors and interactions in the second study were examined within the same analysis. This finding is consistent with feminist theory (e.g., Fallon et al., 1994; Maine, 2000; Striegel-Moore et al., 1986), suggesting that women who think of their bodies as objects by focusing on their outer appearance rather than internal experiences (e.g., feelings, functionality of the body), when dissatisfied with their bodies, are more likely to use drastic and harmful weight control techniques. Because women in Western culture are encouraged to be dissatisfied with their bodies and to monitor their appearance rather than attend to their internal qualities (Fredrickson & Roberts, 1997), it is not surprising that most individuals (90%) with clinical eating disorders are women (Striegel-Moore & Cachelin, 2001). Self-objectification, of which body surveillance is one form, is a result of exposure to social institutions and individuals that sexually objectify women's bodies (Fredrickson & Roberts, 1997). Efforts, then, must be made to reduce sexual objectification within culture as well as attend to women who have internalized these messages and help them reduce their body surveillance behaviors.

This study also garnered empirical support for theoretical propositions that several other variables strengthen the body dissatisfaction–eating disorder symptomatology relation. The significant neuroticism–body dissatisfaction interaction is consistent with assertions that high levels of neuroticism exaggerate the negative elements in other personality characteristics (e.g., body dissatisfaction) and increase maladaptive behaviors associated with psychological disorders (e.g., eating disorder symptomatology; Claridge & Davis, 2001; Striegel-Moore et al., 1986). The finding that the presence of a family member and friend with an eating disorder each slightly strengthened the body dissatisfaction–eating disorder symptomatology relation offers some support for assertions that women with high levels of body dissatisfaction who are exposed to individuals engaging in eating disordered behaviors internalize these behaviors and use them in an attempt to change their body size or shape (Mann, 1995; Striegel-Moore et al., 1986). These findings clarify to clinicians the importance of assessing clients' body surveillance behaviors, levels of neuroticism, and the presence of disordered eating within their family and friendship networks to identify those women, out of the many with high levels of body dissatisfaction, who are more likely to engage in eating disorder symptomatology.

Interestingly, contrary to extant theory (e.g., Garner, 1991; Striegel-Moore et al., 1986), poor impulse regulation slightly reduced rather than accentuated the body dissatisfaction–drive for thinness relation. This finding is particularly surprising given that

poor impulse regulation, when entered as an independent predictor, contributed to higher levels of drive for thinness. An inspection of the regression slopes reveals insight into this antagonistic interaction. It appears that, for women low in body dissatisfaction, having high rather than low levels of poor impulse regulation results in higher drive for thinness scores. In contrast, for women high in body dissatisfaction, women with high poor impulse regulation and low impulse regulation have similar drive for thinness scores. Therefore, poor impulse regulation has little impact on drive for thinness for women with high levels of body dissatisfaction, whereas it does appear to be connected with higher drive for thinness scores for women with low body dissatisfaction.

The present study did not support theorists' predictions that social insecurity (Berndt & Hestenes, 1996; Striegel-Moore et al., 1986) and appearance control beliefs (McKinley & Hyde, 1996) worsen eating disorder symptomatology among women with high levels of body dissatisfaction. Previous research has upheld social support as a moderator of the relation between stress and many psychological disorders (e.g., Krause, 1986). Perceptions of social insecurity and actual social support, however, may not be opposite ends of the same construct. Perhaps actual levels of social support rather than perceptions of social insecurity would moderate this relation. Whereas one component of objectified body consciousness (i.e., body surveillance) moderated the body dissatisfaction–eating disorder symptomatology relation and was related to both body dissatisfaction and eating disorder symptomatology, another component of objectified body consciousness (i.e., appearance control beliefs) did not moderate this relation and was not related to either body dissatisfaction or eating disorder symptomatology. In fact, body surveillance and appearance control beliefs were not related. These findings support the distinctiveness of these components of objectified body consciousness. In addition, it is possible that beliefs that weight specifically can be controlled, rather than general appearance control beliefs, could intensify this relation (Stotland & Zuroff, 1990). Weight is only one dimension assessed by the Appearance Control Beliefs subscale of the OBC (McKinley & Hyde, 1996).

The findings of the present study could greatly inform practice efforts. Although body dissatisfaction repeatedly has been shown to be a strong predictor of disordered eating across studies (e.g., Phelps et al., 1999; Polivy & Herman, 2002), many women experience body dissatisfaction but do not engage in severe levels of disordered eating (Striegel-Moore & Cachelin, 1999). To better understand the link between body dissatisfaction and eating disorder symptomatology, counselors need to be aware of the particular variables that moderate this relation. The results of this study, then, suggest that it may behoove counselors to explore their female clients' levels of body surveillance and neuroticism and the eating patterns of family and friendship networks in conjunction with body dissatisfaction when exploring their clients' eating practices. Counselors could work with their female clients and their clients' social networks to decrease these moderating variables and body dissatisfaction to reduce their clients' eating disorder symptomatology. Many extant eating disorder prevention efforts mainly focus on lowering levels of body dissatisfaction (Mann, 1995). These programs also could concentrate on preventing high levels of body surveillance, neuroticism, and disordered eating among their social networks.

As the present study is among the first to examine whether variables moderate the relation between body dissatisfaction and eating disorder symptomatology, additional research clearly is needed within this area. In particular, it is essential to investigate whether certain variables (e.g., instrumentality, social support of friends and family, active coping strategies) weaken this relation, as knowledge of factors that decrease this relation could further inform practice (e.g., counselors could work to increase levels of these protective variables among their clientele). Twamley and Davis (1999) found that self-esteem reduced this relation. Identification of additional variables that enhance well-being by buffering the body dissatisfaction–eating disorder symptomatology link is particularly congruent with counseling psychology's emphasis on individuals' strengths (Striegel-Moore & Cachelin, 2001). Research in this area also would address calls in the eating disorders literature (e.g., Kashubeck-West & Mintz, 2001) to move beyond identifying risk factors of eating disorder symptomatology to articulating variables that could protect women from engaging in harmful eating behaviors. In addition, investigations are needed to determine whether the present study's findings, which are based largely on samples of White women, would generalize to samples of women of color, older and younger women, women who are not in college, and men. Although these groups clearly are not immune from body dissatisfaction and eating disorder symptomatology (e.g., Tylka & Subich, 2002b), they are greatly underrepresented in research within this domain.

Limitations in the present study need to be addressed. First, as with any study using self-report measures exclusively, its findings may be susceptible to selective or erroneous reporting. Second, it is not clear whether the lack of practical significance in the first study is attributable to the inadequacy of the moderator hypotheses or to the questionable validity of the measures. The measures used in the first study were subscales of the same scale (i.e., EDI-2; Garner, 1991). Even though they are discussed as relatively distinct constructs (Garner, 1991), measures designed as subscales reflecting a common higher order construct (i.e., eating disorder symptomatology) may contain overlapping variance that would not be present in separate scales measuring the primary constructs of interest. The Impulse Regulation and Social Insecurity subscales also have not been studied thoroughly in terms of their validity and test–retest reliability among nonclinical samples of women. Furthermore, the two subscales used to assess eating disorder symptomatology (i.e., Drive for Thinness and Bulimia) were not strongly related and represented somewhat divergent characteristics of eating disorder symptomatology. The second study controlled for these limitations by using a more general measure of eating disorder symptomatology and separate instruments with adequate psychometric evidence among college women. Third, the use of categorical items to assess the presence of a family member or a friend with an eating disorder was not ideal. Participants may not have known specifically what constitutes eating disorder symptomatology and therefore responded to these items with their perceptions of what constitutes disordered eating. Other activities also could be salient in determining whether having a friend or family member predicts women's levels of eating disturbance, such as the level of closeness, the extent and depth of conversations, and the modeling of attitudes and behaviors shared with the person with an eating disorder.

In conclusion, several variables (i.e., body surveillance, neuroticism, presence of family member and friend with an eating disorder) were supported as moderators of the relation between body dissatisfaction and eating disorder symptomatology; in particular, these variables strengthened this relation. Because this study is among the first to explore the complexity of the body dissatisfaction–eating disorder symptomatology relation in this fashion, there clearly is more that we can learn about this area. Future examinations within this domain would greatly inform the field of counseling psychology, as well as other disciplines, by articulating which variables intensify and weaken this relation (so interventions can be directed toward ameliorating these variables) and furthering our understanding of how multiple factors interact to predict eating disorder symptomatology.

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