

The Relationship Between Dissociation and Binge Eating

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ABSTRACT. Despite research findings demonstrating a relationship between dissociation and binge eating, the psychological processes that may underlie this association remain unclear. The present study examined 2 potential explanations: (a) that dissociation disinhibits behavioral control over eating and (b) that dissociation interferes with self-awareness and undermines body image. A total of 151 female university students completed measures of dissociation, body dissatisfaction, impulsivity, internalization of the thin ideal, body comparison, and binge eating. Correlations confirmed the presence of a relationship between dissociation and binge eating, and regression analyses revealed that this relationship is limited to body-specific (somatic) symptoms of dissociation. Path analyses identified body dissatisfaction, comparison, and impulsivity as significant mediators of this relationship. However, inclusion of all 3 mediated paths in a full model revealed that only body dissatisfaction is a unique mediator. The relevance of somatic symptoms, and the unique contribution of body dissatisfaction as a mediator, are consistent with an explanation of the relationship between dissociation and binge eating that is based on a vulnerability of body image. The results emphasize the need for future research to consider the relation of dissociation to a broader range of disordered eating symptoms than simply binge eating.

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Dissociative disorders are characterized by deficits of consciousness, memory, and identity (American Psychiatric Association, 2000) and are implicated in diminished behavioral self-control and disturbances of self-awareness (Brown, 2002, 2006). Dissociative symptoms are more commonly found in individuals who have experienced trauma associated with, for example, emotional, sexual, or physical abuse; exposure to combat; witnessing the death of a loved one; and so on (Kihlstrom, Tatarzyn, & Hoyt, 1993; Putnam, Guroff, Silberman, Barban, & Post, 1986; Schachter, Wang, Tulving, & Freedman, 1982). On this basis, it has been argued that dissociation may develop as a psychological defense mechanism that facilitates the escape from awareness of threatening stimuli and emotions (Everill & Waller, 1995; Hallings-Pott, Waller, Watson, & Scragg, 2005; van der Kolk & van der Hart, 1989).

Dissociative symptoms are overrepresented in individuals diagnosed with an eating disorder (Demitrack, Putnam, Brewerton, Brandt, & Gold, 1990; Everill, Waller, & MacDonald, 1995; Goldner, Cockhill, Bakan, & Birmingham, 1991; Groth-Marnat & Michel, 2000; McManus, 1995; Vanderlinden, Vandereycken, van Dyck, & Vertommen, 1993; Waller, Ohanian, Meyer, Everill, & Rouse, 2001). Although this may simply reflect a common origin for dissociation and body dissatisfaction in abuse experienced in childhood (Beato, Cano, & Belmonte, 2003), there are at least two reasons to suspect a *causal* relationship between the two. First, correlations between child abuse and disordered eating are neither strong nor consistent, and they are weaker than the relationship between dissociation and body dissatisfaction (Ball, Kenardy, & Lee, 1999). Second, the need to escape from awareness of abuse would predict widespread symptoms of psychological disturbance consistent with *generalized* dissociation. Contrary to this, although body dissatisfaction is broadly relevant to symptoms of disordered eating (Stice & Agras, 1998), recent findings have suggested that somatic dissociative symptoms, more so than generalized dissociative symptoms, are related to disordered eating symptomatology (Waller et al., 2003). Furthermore, case reports and correlational studies have indicated that dissociative experiences may be relevant to eating psychopathology (Chandarana & Malla, 1989; McCallum, Lock, Kulla, Rorty, & Wetzel, 1992; Torem, 1986) and are related to frequency of binge-eating episodes (Everill et al., 1995; Waller et al., 2001; cf. Covino, Jimerson, Wolfe, Franko, & Frankel, 1994).

Accordingly, the elevated levels of dissociation exhibited by these individuals may reflect the direct effects of dissociation on binge eating.

One causal explanation for this link focuses on disinhibition of behavioral symptoms of disordered eating. According to this behavioral disinhibition explanation, escape-from-awareness strategies characteristic of dissociation predispose individuals to ignore, or at least to deemphasize, feedback that normally serves to suppress unhealthy behavioral responses to food, particularly overeating (Everill & Waller, 1995; Heatherton & Baumeister, 1991). This feedback can include awareness of what one has recently eaten and awareness of what is an appropriate amount to eat, as well as physiological cues for satiation, including sensitivity to blood glucose levels, stomach distension, and so on. Thus, overutilization of escape-from-awareness strategies in response to threatening stimuli and/or painful emotions can indirectly facilitate binge eating. In support of the behavioral disinhibition explanation, measures of trait impulsivity have been found to correlate with disordered eating symptomatology (Loxton & Dawe, 2001; Wonderlich, Connolly, & Stice, 2004), and experimentally introduced threat cues have been shown to elicit dissociative symptoms as well as overeating in individuals with an eating disorder (Hallings-Pott et al., 2005; Meyer & Waller, 1999; Waller & Mijatovich, 1998). However, the behavioral disinhibition explanation cannot account for the relationship observed between dissociation and disturbances of self-awareness (Beato et al., 2003; Valdiserri & Kihlstrom, 1995a, 1995b).

It is also possible that the relationship between dissociation and disordered eating is due additionally—or primarily—to dissociative disruptions to body image. Mussap and Salton (2006) proposed that body distortions reported by women with eating disorders (Bruch, 1962; Farrell, Lee, & Shafran, 2005; although Hsu & Sobkiewicz, 1991, argued against the relevance of body size overestimation for disordered eating), and the changes in body size evaluations made by these women over different testing sessions (Brinded, Bushnell, McKenzie, & Wells, 1990; Gardner & Bokenkamp, 1996) and/or following exposure to idealized bodies depicted in fashion magazines (e.g., Waller & Barnes, 2002), reflect an underlying instability of body image that is related to somatoform dissociation. Mussap and Salton proposed that dissociation undermines normal integration of appearance-relevant information and might, in turn, contribute to body image vulnerability. They argued further that in a society that reinforces unrealistic standards of thinness, this vulnerability could manifest as internalization of the thin ideal, elevated body comparison attitudes and behaviors, and/or body dissatisfaction. This body image vulnerability explanation is consistent with previous findings that

demonstrate a link between dissociation and body dissatisfaction and unhealthy body change attitudes, such as drive for thinness, dietary restraint, and endorsement of weight-loss techniques (Beato et al., 2003; Kent, Waller, & Dagnan, 1999; Lyubomirsky, Casper, & Sousa, 2001; McManus, 1995; Valdiserri & Kihlstrom, 1995a).

To date, no research has been conducted to evaluate the relevance of behavioral disinhibition and body image vulnerability to the relationship between dissociation and binge eating. In the present study, the behavioral disinhibition explanation was tested by assessing the extent to which impulsivity mediates the relationship between dissociation and binge eating. Impulsivity is a multidimensional construct comprising impulsive urgency, lack of premeditation, lack of perseverance, and sensation seeking (Whiteside & Lynam, 2001). Because previous research has suggested that binge eating is most strongly related to the urgency component of impulsivity (Fischer, Anderson, & Smith, 2004; Fischer, Smith, & Anderson, 2003; Fischer, Smith, Anderson, & Flory, 2003), impulsive urgency should constitute the primary mediator of the effects of dissociation.

The body image vulnerability explanation was tested by measuring the extent to which the relationship between dissociation and binge eating is limited to the somatic symptoms of dissociation (as measured using the Somatoform Dissociation Questionnaire [SDQ-20]; Nijenhuis, Spinhoven, van Dyck, van der Hart, & Vanderlinden, 1996). To test whether these somatic effects are due to vulnerability of body image, vulnerability to sociocultural pressures on appearance, and/or vulnerability to “upward” comparisons, we tested body dissatisfaction, internalization of the thin ideal, and body comparison, respectively, as mediators of the relationship between somatoform dissociation and binge eating.

Finally, to test the independence of the behavioral disinhibition and body image vulnerability paths to binge eating, we used path analyses to test a mediational model in which all putative mediators were included.

METHOD

Participants

A convenience sample of 151 adult women, aged between 18 and 40 years ($M = 22.65$, $SD = 4.37$), participated in the study. Invitations to participate were announced at the beginning of lessons in various undergraduate classes offered in the Faculty of Health, Medicine, Nursing, and Behavioural

Sciences at Deakin University's Melbourne campus in Semester One of 2006. No demographic information (other than age) was sought from participants, although it should be noted that students in the classes sampled were predominantly unmarried women in their 20s, and almost all were of European descent. Copies of the materials were left in lecture hall foyers for students interested in the study to collect at the end of the class. Participation was voluntary, and no financial or academic inducements were offered. A total of 450 questionnaires were distributed in this way, and 151 (34%) were returned. National Institutes of Health (1998) guidelines were applied to interpret body mass index (BMI) scores: 8% of this sample was "underweight" (BMI < 18.5), 75% was "normal weight" (BMI = 18.5–24.9), 16% was "overweight" (BMI = 25.0–29.9), and 1% was "obese" (BMI ≥ 30.0). For the overall sample, average BMI was 22.03 ($SD = 2.97$).

Materials

Eating disorders examination-questionnaire (EDE-Q). The EDE-Q is a self-report questionnaire (Fairburn & Beglin, 1994), derived from the Eating Disorders Examination (Cooper & Fairburn, 1987), that uses 7-point Likert scales that range from 0 = *no days* to 6 = *every day* to assess the number of days out of the past 28 that particular attitudinal, emotional, and behavioral symptoms of disordered eating were present. There are four subscales of the EDE-Q: dietary restraint (REST; $M = 1.37$, $SD = 1.32$), concern with eating (EAT; $M = 0.87$, $SD = 1.03$), concern with body weight (WEIGHT; $M = 1.82$, $SD = 1.43$), and concern with body shape (SHAPE; $M = 2.21$, $SD = 1.51$). Because we were interested in exploring the effects of overall body dissatisfaction (rather than the specific aspects of dissatisfaction), and because the measures of shape and weight concerns were multicollinear ($r = .91$), the two measures were averaged together to form a single measure of body dissatisfaction (DISS; $M = 2.06$, $SD = 1.45$).

The EDE-Q subscales have good internal consistency and test–retest reliability, and concurrent and discriminant validity support the use of the EDE-Q as a screening tool in clinical practice (Binford, LeGrange, & Jellar, 2005; Cooper, Cooper, & Fairburn, 1989; Mond, Hay, Rodgers, Owen, & Beumont, 2004).

The EDE-Q also assesses the symptoms of purging and binge eating over the past 28 days. The symptoms of purging include vomiting, laxative use, use of diuretics, and exercising hard (the EDE-Q does not

define the term *hard* for respondents). For the statistical analyses, the presence of vomiting, laxative use, use of diuretics, and exercising hard were summed to get a variable that represented a tally of symptoms of purging (i.e., between 0 and all 4 symptoms), and the frequency of binge-eating episodes over the previous 28 days was taken to get a variable that represented the frequency of binge eating. In the present study, the mean number of purge symptoms (PURGE) was 0.43 ($SD = 0.53$), and the mean frequency of binge eating (fBINGE) was 1.04 ($SD = 1.32$).

Given our focus on binge eating, we sought to assess the diverse symptoms (including the evaluative, emotional, and behavioral symptoms) of binge eating. To this end, we included Question 4 of the Questionnaire of Eating and Weight Patterns–Revised (QEWP; Spitzer et al., 1992). This question assesses the presence of six symptoms of bingeing: rapid consumption of food, eating until uncomfortably full, eating despite not being hungry, eating large quantities of food throughout the day, eating alone, and feeling guilt and shame about a binge episode. Responses to these six subquestions were summed to yield a tally of symptoms of binge eating (BINGE) ranging from 0 to all 6 symptoms ($M = 1.54$, $SD = 1.88$). Although the global QEWP scale is a validated measure of binge eating (Nangle, Johnson, Carr-Nangle, & Engler, 1994), it was important for our purposes to validate Question 4 of the QEWP in isolation. We did this by correlating this question with the fBINGE subscale of the EDE-Q (note that although fBINGE was a validated measure, it was a single item that did not differentiate between diverse symptoms of binge eating and thus relied on participants' own understandings of the concept of overeating). A large and significant correlation ($r = .85$, $p < .01$) between QEWP Question 4 (BINGE) and the EDE-Q measure of binge eating (fBINGE) was in fact obtained. Thus, the BINGE measure derived from Question 4 of the QEWP was used in subsequent inferential analyses.

Sociocultural attitudes towards appearance questionnaire (SATAQ-3). The Internalization–General subscale of the SATAQ-3 (Thompson, van den Berg, Roehrig, Guarda, & Heinberg, 2004) uses 5-point Likert scales that range from 0 = *never* to 4 = *always* to assess internalization (INTERN; $M = 1.90$, $SD = 0.96$) of the thin ideal. The SATAQ-3 has demonstrated content and convergent validity with regard to measures of body image and disordered eating, and it possesses adequate internal consistency (Thompson et al., 2004).

Physical appearance comparisons scale (PACS). The PACS (Thompson, Heinberg, & Tantleff-Dunn, 1991) uses 5-point Likert scales that range from 0 = *never* to 4 = *always* to assess frequency of engagement in

body comparisons. The PACS measures frequency of comparison of one's whole body and one's body parts (e.g., stomach, hips, buttocks). The scale also measures frequency of comparison of one's body against the bodies of others (e.g., at parties, in magazines). An example item is "In social situations, I sometimes compare my figure to the figures of other people." The PACS ($M = 2.08$, $SD = 0.78$) has demonstrated internal consistency and test-retest reliability (Thompson et al., 1991).

Dissociative experiences scale (DES). The DES (Carlson & Putnam, 1993) uses 5-point Likert scales that range from 0 = *never* to 4 = *always* to assess the presence of general dissociative experiences, such as absorption and imaginative involvement, amnesic dissociation, and depersonalization/derealization. The DES ($M = 0.75$, $SD = 0.26$) has good psychometric properties (Bernstein & Putnam, 1986; Frischholz et al., 1991; Gleaves, Williams, Harrison, & Cororve, 2000).

SDQ-20. The SDQ-20 (Nijenhuis et al., 1996) uses 5-point Likert scales that range from 0 = *never* to 4 = *always* to assess the presence and frequency of dissociative symptoms related to bodily sensations and functions. The SDQ-20 ($M = 0.35$, $SD = 0.32$) has good psychometric properties (Nijenhuis et al., 1996; Nijenhuis, Spinhoven, van Dyck, van der Hart, & Vanderlinden, 1998).

UPPS impulsivity scale (Whiteside & Lynam, 2001). This scale uses 4-point Likert scales that range from 1 = *agree strongly* to 4 = *disagree strongly* to assess four dimensions of trait impulsivity: impulsive urgency (URGENCY; $M = 2.54$, $SD = 0.48$), lack of premeditation (PREMED; $M = 2.07$, $SD = 0.46$), lack of perseverance (PERSEV; $M = 1.92$, $SD = 0.43$), and sensation seeking (SEN-SEEK; $M = 2.70$, $SD = 0.58$). The UPPS Impulsivity Scale has good test-retest reliability and validity (Whiteside, Lynam, Miller, & Reynolds, 2005).

Procedure

Participants completed the scales in private and returned the questionnaire using the postage-paid reply envelope provided with the questionnaire.

Data Screening, Variable Construction, and Analytic Strategy

Preliminary data screening revealed missing values distributed randomly across the items and participants. When participants failed to provide a response to diagnostic symptoms, it was assumed that these symptoms were absent. Accordingly, missing values for items that composed the EDE-Q subscales were replaced with scores of 0 (*no days*); missing values

for BINGE, PURGE, DES, and SDQ items were replaced with a “no” response (for the presence of a symptom) or a 0 response (for the frequency of occurrence of the symptom). For age and for each of the impulsivity subscales, missing values were replaced with median values. For all other variables, missing values were replaced with the value midway between these extremes (i.e., the middle value of the scale). Fewer than 2% of cases for each variable had missing values.

Scales were created from the averaged responses to the internally consistent items (Table 1 lists Cronbach’s alphas for each scale). All scales were screened for violations from normality (Cohen & Cohen, 1983), and, as a result, square root transformations of SDQ, BINGE, EAT, PURGE, and DES were conducted. Univariate outliers (greater than ± 3.29 standard deviations from the mean) were identified and replaced with the value that corresponded to 3.29 standard deviations. One multivariate outlier was identified and deleted. Because multiple exploratory analyses were conducted, a more conservative statistical significance criterion of $p < .01$ was used to correct for the increased risk of Type I errors.

Zero-order correlations were undertaken to explore relationships between both forms of dissociation, components of impulsivity, and disordered eating symptomatology. A hierarchical multiple regression was conducted in which the measure of binge eating (BINGE) was regressed on the measure of generalized dissociation in Step 1 and the measure of somatoform dissociation in Step 2. This allowed the determination of (a) the relative importance of somatic symptoms relative to generalized dissociative symptoms and (b) the extent to which somatic symptoms accounted for variance in binge eating that was additional to that accounted for by symptoms of generalized dissociation.

Path analyses, using maximum likelihood estimation with AMOS™, were then carried out to test the proposed behavioral disinhibition and body image vulnerability explanations for the relationship between somatoform dissociation and binge eating, both individually and then collectively. The significance of indirect effects tested in AMOS™ was used to determine whether the following variables mediated the relationship between dissociation and binge eating: (a) impulsive urgency (URGENCY), (b) body dissatisfaction (DISS), (c) internalization of the thin ideal (INTERN), and (d) body comparisons (COMP). Finally, a combined path model with each of the individually significant mediators (see Figure 1) was conducted to evaluate the independent predictive value of these behavioral disinhibition (i.e., impulsive urgency) and body image vulnerability (i.e., DISS, INTERN, and COMP) explanations. To test

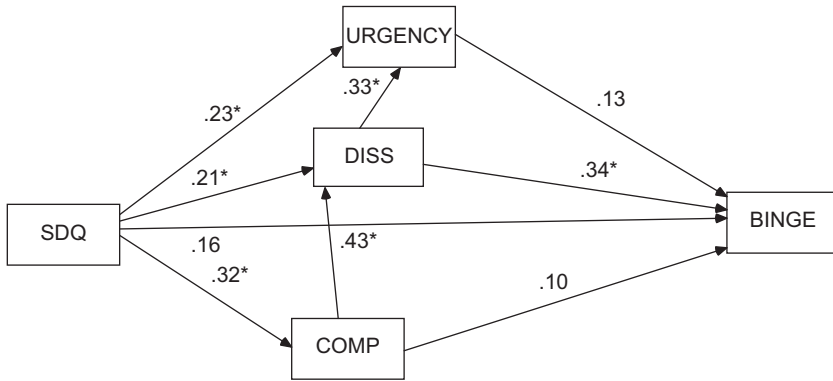
TABLE 1. Bivariate correlations (Pearson's *r*).

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1. SDQ	.91												
2. DES	.68*	.91											
3. BINGE	.39*	.32*	NA										
4. PURGE	.23*	.15	.24*	NA									
5. DISS	.35*	.30*	.50*	.31*	.94								
6. EAT	.42*	.37*	.57*	.34*	.80*	.78							
7. REST	.27*	.20*	.20*	.39*	.70*	.61*	.78						
8. COMP	.33*	.34*	.38*	.09	.49*	.45*	.31*	.82					
9. INTERN	.20*	.18	.23*	.09	.43*	.34*	.34*	.49*	.90				
10. PREMEDI	.19	.11	.06	.19	-.09	.01	-.16	-.05	-.12	.82			
11. URGENCY	.37*	.38*	.36*	.18	.41*	.34*	.21*	.39*	.28*	.16	.83		
12. PERSEV	.05	.11	.13	.15	.15	.16	.02	.08	-.04	.43*	.34*	.79	
13. SEN-SEEK	.10	.15	.00	.08	-.05	-.03	-.13	-.20*	.04	.23*	.04*	-.14	.87

Notes: The numbers (in bold) along the diagonal are Cronbach's alphas for the respective variables. SDQ = somatoform dissociative experiences; DES = generalized dissociative experiences; BINGE = number of binge-eating symptoms; PURGE = number of purge symptoms; DISS = body dissatisfaction; EAT = eating concerns; REST = dietary restraint; COMP = body comparison practices; INTERN = internalization of the thin ideal; PREMEDI = lack of premeditation; URGENCY = impulsive urgency; PERSEV = lack of perseverance; SEN-SEEK = sensation seeking; NA = not applicable.

* $p < .01$.

FIGURE 1. Full Model, Including All Significant Mediation Pathways



URGENCY = impulsive urgency; DISS = body dissatisfaction; SDQ = somatoform dissociative experiences; BINGE = number of binge-eating symptoms; COMP = body comparison practices. * $p < .01$ for standardized coefficients.

model fit, we used the chi-square goodness-of-fit statistic, an index of the overall proportion of explained variance (good fit ≥ 0.95), an index of overall proportion of explained variance that adjusted for model complexity (good fit ≥ 0.95), and an index of the standardized residuals of estimation (standardized root mean square residual; good fit < 0.08 ; Kline, 1998).

Although AMOSTM calculated the significance of the combined indirect effects involving somatoform dissociation (SDQ) in the full model, it did not allow determination of the significance of each indirect effect independently. Accordingly, unstandardized coefficients and standard errors were obtained from AMOSTM for the relationships between the putative mediator and both SDQ and BINGE. These statistics were then input into Sobel's (1982) product of coefficients test to calculate z scores corresponding to the independent effects of these mediators, holding constant the influence of the other mediators.

RESULTS

Inspection of bivariate correlations in Table 1 shows that generalized (DES) and somatoform (SDQ) dissociation correlated significantly but were not multicollinear ($r = .68$, $p < .01$). Both also correlated significantly with binge eating, body comparison, body dissatisfaction, eating

concerns, and dietary restraint; and somatoform dissociation also correlated with internalization of the thin ideal and purging. We tested the extent to which the relationship between dissociation and binge eating was unique to somatic symptoms of dissociation by conducting a hierarchical regression in which BINGE was regressed on DES in Step 1 and on both DES and SDQ in Step 2. Although generalized dissociation accounted for a significant proportion of the variance in BINGE at Step 1 ($R^2 = .10, p < .01; \beta = .32, p < .01$), its contribution reduced to nonsignificance ($\beta = .12, p > .01$) after inclusion of somatoform dissociation ($\beta = .31, p < .01$). In fact, SDQ accounted for an additional 6% variance in binge eating ($\Delta R^2 = .06, p < .01$). Despite the strong correlation between the two measures of dissociation, collinearity diagnostics indicated that the two independent variables were not multicollinear (tolerance = .57, variance inflation factor = 1.76). This regression analysis supports the hypothesis that binge eating is related to somatic, more so than generalized, dissociative symptoms.

Table 1 also shows that of the dimensions of impulsivity assessed in the present study, only impulsive urgency (URGENCY) correlated with both DES and SDQ. URGENCY was also positively correlated with binge eating, body dissatisfaction, body comparison, and internalization. That these relationships involved urgency, rather than the other aspects of impulsivity, is consistent with the proposition that one pathway from dissociation to binge eating is via behavioral disinhibition. However, to test this and the alternative explanation (namely, body image vulnerability) explicitly, we undertook path analyses.

When the paths were measured separately, analyses provided support for both the behavioral disinhibition and body image vulnerability explanations. The results of these path analyses revealed that the indirect (i.e., mediated) effects involving body dissatisfaction (DISS, $\beta = .15, p < .01$), body comparison (COMP, $\beta = .09, p < .01$), and impulsive urgency (IMPULSE, $\beta = .09, p < .01$), but not through internalization of the thin ideal (INTERN, $\beta = .03, p > .01$), were significant, indicating that these variables mediate the relationship between SDQ and binge eating. However, when all significant mediators were included together in a full model, only body dissatisfaction (DISS) remained significant (Sobel's $z = 3.26, p < .01$). This finding highlights the interrelations between the proposed mediators (see Table 1) while also indicating that body dissatisfaction contributes independently to the relationship between dissociation and binge eating.

Of interest is that the direct (unmediated) path between SDQ and binge eating was reduced to nonsignificance in the full model, indicating that the

combination of these mediators *fully* mediates the relationship between somatoform dissociation and binge eating. The fit indices suggest that the model was relatively good, $\chi^2(1) = 6.26$, $p > .01$, $N = 150$, goodness-of-fit index = .98, standardized root mean square residual = .02, although the proportion of variance explained was relatively poor when model complexity was taken into consideration (adjusted goodness-of-fit index = .76). This indicates that one or several of the (nonsignificant) pathways could be trimmed or constrained to zero without loss of explanatory power. Given the anticipated correlation between several of the mediators (particularly the body image vulnerability variables), this finding is not unexpected.

DISCUSSION

The present findings provide insight into the psychological basis for the relationship between dissociation and binge eating. A behavioral disinhibition explanation of this relationship, in which impulsive urgency mediates the effects of dissociation, received only limited support. However, the importance of *somatic* symptoms of dissociation, and the involvement of body dissatisfaction and body comparison as mediators of the relationship between these somatic symptoms and binge eating, supports an alternative explanation—that dissociation is relevant to uncontrolled eating because it serves to undermine normal processes of self-awareness.

Somatoform dissociation not only explained significant variance in binge eating in addition to variance explained by generalized dissociation (the magnitude of these effects was comparable to previous research; e.g., Waller et al., 2003), it also reduced to nonsignificance the relationship between generalized dissociation and binge eating. Together, these results suggest that the relationship between dissociation and binge eating is primarily related to the *somatic* symptoms of dissociation that reflect disruptions to body-based awareness and bodily functions (Nijenhuis et al., 1996).

As to the psychological bases of this somatic effect, impulsive urgency partially mediated the relationship between somatoform dissociation and binge eating. The observation that lack of premeditation/planning (a component of impulsivity) was unrelated to binge eating or somatoform dissociation further suggests that the effects of dissociation on binge eating do not reflect a general impairment of executive functioning. Heatherton and Baumeister (1991) posited that, in many instances, individuals who utilize

escape-from-awareness strategies do so to avoid negative self-appraisals such as may arise from internalization of the (unrealistic) thin ideal, body comparison practices, and body dissatisfaction. Thus, the observation that these body image variables correlate with impulsive urgency is consistent with the view that negative emotions and stimuli can promote disinhibited behaviors. The specificity of the link between body-based negative self-appraisals and binge eating may result from the preoccupation with, and overimportance attributed to, appearance for individuals with symptoms of disordered eating.

Consistent with the body image vulnerability explanation, body dissatisfaction and frequency of body comparison were also found to partially mediate the relationship between somatoform dissociation and binge eating. These factors are known to predispose individuals to adopt a range of unhealthy body change attitudes and behaviors (Fairburn et al., 2003; Stice, 2001; Stice & Agras, 1998), and their mediating role in the context of dissociation is consistent with the proposition that somatic symptoms of dissociation undermine body image. Previous research has shown that experimentally measured instability of body image is associated both with vulnerability to sociocultural pressures to be thin and with symptoms of disordered eating (Mussap & Salton, 2006).

It is important to note that (a) only body dissatisfaction remained a significant mediator when tested as part of a full model that included all other putative mediators (including impulsive urgency, which did not remain significant), and (b) the full model's mediation effects were not trivial—inclusion of the mediators reduced to nonsignificance the direct path between somatoform dissociation and binge eating, suggesting the presence of *full* mediation. These results call into question the behavioral disinhibition explanation. Previous research (e.g., Everill et al., 1995; Waller et al., 2001) cited in support of the behavioral disinhibition explanation has shown that dissociation (particularly generalized dissociation) tends to be related to short-term body change strategies and disturbances (e.g., bingeing). However, the present finding that somatoform dissociation is relevant to body image variables (independent of impulsivity) implicates dissociation in a broader range of disturbances.

Before concluding, it is worth considering the limitations of the present study. Interpretations of causality (including the *direction* of mediated effects in path analysis) were limited by the cross-sectional design employed (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). Although beyond the intended scope of the present study, structural equation

modeling (of which path analysis is a subtype) permits computation of mathematically equivalent models with different configurations of paths among these same variables, thus allowing for model fit comparisons (Kline, 1998). Accordingly, it is possible that causal relationships exist between the variables in the present study but that we have misspecified the model. We are currently engaged in an experimental confirmation of the behavioral disinhibition model that trait dissociation contributes to attentional shifts consistent with an escape from awareness, and that the tendency to escape from awareness predisposes individuals to overeat. Finally, the low response rate (34%) and use of a nonclinical sample in the present study limit generalizability of the findings to the broader community and to individuals with diagnosed dissociative disorders and/or diagnosed eating disorders, respectively. Lack of a clinical sample also makes it possible that any null results obtained reflect lack of variance in dissociative and/or disordered eating symptomatology.

The present findings indicate that dissociation is relevant to a broad range of disordered eating symptoms in a nonclinical sample. Future research could examine the applicability of behavioral disinhibition and body image vulnerability explanations for clinical samples. Furthermore, longitudinal studies are necessary to determine the influence of trait dissociation on the development and maintenance of disordered eating symptomatology.

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