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# Association Between Childhood Physical and Emotional Abuse and Disordered Eating Behaviors in Female Undergraduates: An Investigation of the Mediating Role of Alexithymia and Depression

Suzanne E. Mazzeo
Virginia Commonwealth University

Dorothy L. Espelage
University of Illinois at Urbana-Champaign

Although disordered eating behaviors are relatively common among college women, many questions about their etiology remain. In the present study, structural equation modeling was used to investigate potential mediating associations among variables previously found to be associated with the continuum of disordered eating behaviors in a large sample of college women. Results indicated that family conflict, family cohesion, and childhood physical and emotional abuse and neglect were not directly associated with disordered eating. Rather, their association with disordered eating was mediated by alexithymia and depression. These results were cross-validated in a second sample of college women. These data highlight the complexity of disordered eating and provide future directions for the prevention and treatment of the continuum of disordered eating behaviors.

Although estimates of the prevalence of eating disorders in the general population vary, studies have found that as many as 64% of college women engage in disordered eating behaviors (e.g., Mintz & Betz, 1988). Several authors have suggested that disordered eating behaviors occur on a continuum, with asymptomatic, unrestrained eating on one end and clinical eating disorders on the other (e.g., Mintz, O'Halloran, Mulholland, & Schneider, 1997; Nylander, 1971; Rodin, Silberstein, & Striegel-Moore, 1985; Tylka & Subich, 1999). Numerous studies have found support for the validity of the eating disorder continuum (e.g., Dancyger & Garfinkel, 1995; Lowe et al., 1996; Mintz & Betz, 1988; Stice, Ziemba, Margolis, & Flick, 1996; Tylka & Subich, 1999). For example, Lowe and colleagues tested the continuity hypothesis using trend analyses and found that weight concerns increased in a linear fashion across categories of disordered eating (i.e., unrestrained nondieters, restrained nondieters, dieters, and bulimics). In contrast, tests of quadratic and cubic trends were nonsignificant. In addition to finding that problematic eating behaviors exist on a continuum, several investigators of the continuity hypothesis have found that women with subclinical eating disorders experience significant psychological distress, including low self-esteem (Mintz & Betz, 1988), neuroticism (Tylka & Subich, 1999), and depression (Dancyger & Garfinkel, 1995).

Nonetheless, some have argued that women with eating disorders are categorically different from individuals with subclinical forms of eating pathology (e.g., Bruch, 1973; Ruderman & Besbeas, 1992), and researchers have also found support for this discontinuous model. For example, Ruderman and Besbeas (1992) investigated the continuum of disordered eating and psychological distress in an undergraduate sample using the Bulimia Test (BULIT; Smith & Thelen, 1984) and the Revised Restraint Scale (Herman, Polivy, Pliner, Threlkeld, & Munic, 1978). In their study, women classified as bulimic exceeded the BULIT clinical cutoff, women classified as noneating disordered dieters did not exceed the BULIT cutoff but scored above 17 on the Revised Restraint Scale, and nondieting controls scored below the BULIT cutoff and below 13 on the Revised Restraint Scale. Ruderman and Besbeas found that women with bulimia differed from dieters on 16 of the 24 measures of psychological distress included in their study (e.g., Taylor Manifest Anxiety Scale [Taylor, 1953], Beck Depression Inventory [BDI; Beck, 1972], Tennessee Self-Concept Scale [Fitts, 1964]). In contrast, dieters differed from controls on only 1 of the 24 measures (i.e., Marlowe–Crowne Social Desirability Scale; Crowne & Marlowe, 1960). Similarly, Gleaves, Lowe, Snow, Green, and Murphy-Eberenz (2000) conducted a taxometric analysis of disordered eating behaviors in a sample of noneating disordered college women and women with bulimia nervosa and found that the majority of their results supported the discontinuity model.

Suzanne E. Mazzeo, Department of Psychology, Virginia Commonwealth University; Dorothy L. Espelage, Department of Educational Psychology, University of Illinois at Urbana-Champaign.

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Correspondence concerning this article should be addressed to Suzanne E. Mazzeo, Department of Psychology, P.O. Box 842018, Virginia Commonwealth University, Richmond, Virginia 23284-2018. E-mail: semazzeo@vcu.edu

However, other studies provided mixed support for the discontinuity model. Laessle, Tuschl, Waadt, and Pirke (1989) found that restraining (i.e., subclinical) and nonrestraining (i.e., noneating disordered) women did not differ on six of the eight scales of the Eating Disorder Inventory (EDI; Garner, Olmsted, & Polivy, 1983) or on the Hunger subscale of the Three-Factor Eating Questionnaire (Stunkard & Messick, 1985). However, these groups did differ significantly on the Drive for Thinness and Body Dissatisfaction scales of the EDI, the Body Shape Questionnaire (Cooper, Taylor, Cooper, & Fairburn, 1987), and a short form of the BDI; scores on these measures were associated with disordered eating in a linear fashion.

In a recent study, Stice, Killen, Hayward, and Taylor (1998) reviewed several of these disparate findings and suggested that these differences may be accounted for, in part, by variability in the statistical power of the studies. Specifically, these authors noted that cell sizes were larger in studies supporting the continuity perspective. Stice et al. (1998) extended previous research on the continuity hypothesis by using a large community sample ($N$ = 920) as well as structured clinical interviews of disordered eating behavior. They found clear linear associations across eating disorder categories (bulimic, subthreshold bulimic, or noneating disordered) for both weight-related factors (i.e., thin-ideal internalization, dietary restraint, and body dissatisfaction) and psychopathology (i.e., anxiety symptoms, depressive symptoms, and emotionality), providing strong support for the continuum hypothesis.

In sum, investigations of the similarities between subclinical and clinical levels of disordered eating suggest that, although results are somewhat mixed, it seems reasonable to view problematic eating behaviors as existing on a continuum. Moreover, in general, results of previous research suggest that many women whose symptoms do not currently meet criteria for a clinical eating disorder diagnosis nonetheless experience considerable distress. As Tylka and Subich (1999) have noted, investigating distress associated with problematic eating behaviors (both clinical and subclinical) is highly consistent with the mission of counseling psychology, which emphasizes working with individuals at all levels of adjustment. Thus, the present study measured disordered eating behaviors on a continuum for the purpose of adding to the understanding of the full range of problematic eating behaviors manifested by nonclinical undergraduate women.

Although several authors have previously investigated factors associated with the continuum of disordered eating behaviors (e.g., Mintz & Betz, 1988; Tylka & Subich, 1999), many questions about the etiology and maintenance of these behaviors remain (e.g., Marx, 1994; Pike, 1995). Previous research has identified several correlates of disordered eating behavior, including family dysfunction (e.g., Szmukler, 1985), childhood abuse (e.g., Steiger & Zanko, 1990), depression (e.g., Mizes, 1988), and alexithymia (e.g., Laquatra & Clopton, 1994). Yet there are still significant gaps in the literature due, in part, to the fact that most researchers in this area have used univariate statistical methods. This approach is problematic, as univariate analyses are unable to address potential associations among measured (dependent) variables. In a recent review of the literature on risk factors for disordered eating, Mussell, Binford, and Fulkerson (2000) noted that advances in the theoretical and empirical understanding of the etiology of disordered eating have been limited by the fact that hypothesized risk factors are often studied individually, rather than in a multivariate context.

Structural equation modeling (SEM) is a methodology that is particularly useful in empirically testing complex theoretical conceptualizations (e.g., Fassinger, 1987). A few studies of disordered eating behavior have used this statistical approach; however, in general, these studies focused on very specific etiological factors such as family processes (Leung, Schwartzman, & Steiger, 1996), stress (Shatford & Evans, 1986), and mood (Stice, 1998) and did not address associations among these variables. Thus, there appears to be a need for a more comprehensive, empirically validated, theoretical model of disordered eating to increase researchers' understanding of these behaviors. Consequently, in the present study we used SEM to test a mediational model of variables that have been identified as significant correlates of disordered eating behaviors in previous studies, including family functioning, abuse history, alexithymia, and depression. The sections that follow describe these variables in turn and describe their associations with one another on the basis of the results of previous research.

## Family Functioning and Disordered Eating Behaviors

The association between family functioning and disordered eating behaviors has received a great deal of attention. Numerous clinicians and researchers have noted the influence of familial factors in the development and course of both anorexia and bulimia nervosa (e.g., Minuchin, Rosman, & Baker, 1978; Selvini-Palazzoli, 1974). However, an eating disorder can often cause significant changes in familial relationships (Waller & Calam, 1994). Thus, results of studies of family functioning among women with clinical eating disorders are potentially confounded by the fact that families are not assessed until they are in treatment. Consequently, data collected from clinical samples cannot clearly indicate which, if any, family functioning variables may predispose an individual to develop an eating disorder and which develop in response to the stress inherent in the treatment process. The use of nonclinical samples in the present study should add to researchers' understanding of the association between family functioning and disordered eating severity.

Previous studies that have used nonclinical samples have found somewhat inconsistent results regarding the association between family functioning and disordered eating behavior. For example, Kent and Clopton (1992) found that emotional expressiveness within the family was the only aspect of family functioning that differentiated undergraduate women who engaged in disordered eating behaviors from those who did not. The disordered eating group reported lower levels of emotional expressiveness in their families compared with the noneating disordered group. However, the two groups did not differ in their self-reports of family cohesiveness, independence, conflict, organization, or control. In contrast, in another sample of female undergraduates, Scalf-McIver and Thompson (1989) found that inconsistent expression of parental affection and family conflict were positively correlated with bulimic symptomatology, whereas family cohesion was negatively correlated with bulimic behaviors.

Leung et al. (1996) tested a structural equation model of family processes in the eating disorders using a nonclinical sample. Although this study exclusively included adolescents, it is important to note here as it is, to date, the only structural equation model of

family processes in the eating disorders. Leung et al. found that adolescent girls who regarded their families as relatively incohesive and inflexible felt more inadequate about themselves and engaged in disordered eating behaviors more frequently.

In addition to the fact that previous research investigating the association between family functioning and disordered eating has yielded conflicting results, family functioning has been found to be significantly associated with other variables of interest in this study. For example, Ray, Jackson, and Townsley (1991) found that low levels of family cohesion were associated with increased rates of both intrafamilial and extrafamilial childhood sexual abuse. Consequently, it seemed important to include family variables in the present investigation to reduce the probability of specification error (i.e., the error of omitting variables that are particularly relevant to the criteria; Kline, 1998).

## Abuse History and Disordered Eating Behaviors

The vast majority of research that has investigated the association between disordered eating and abuse has exclusively measured sexual forms of abuse (Waller, Everill, & Calam, 1994). However, researchers have recently begun to examine associations between nonsexual forms of childhood abuse and disordered eating behaviors (e.g., Rorty, Yager, & Rossotto, 1994). Hence, in the present study we investigated multiple forms of abuse. We first review the research on sexual abuse and then discuss the smaller number of studies that have examined the influence of physical and emotional abuse on disordered eating behaviors.

### *Sexual Abuse*

In a recent review, Wonderlich, Brewerton, Jocic, Dansky, and Abbott (1997) noted that 53 studies examining some aspect of the relationship between childhood sexual abuse and disordered eating were conducted between 1987 and 1994. However, results of these studies were inconsistent, with some reporting no significant association between childhood sexual abuse and disordered eating behaviors (e.g., Pribor & Dinwiddie, 1992; Rorty et al., 1994) and others suggesting that a significant association does exist (e.g., Calam & Slade, 1989; Steiger & Zanko, 1990). One proposed explanation for these conflicting results is that much research to date has failed to consider the influence of potential mediators or moderators of the association between sexual abuse and disordered eating (e.g., Schmidt, Humfress, & Treasure, 1997; Waller et al., 1994). Recent research has begun to examine some of these factors. For example, Kinzl, Traweger, Guenther, and Biebl (1994) found no significant relationship between sexual abuse and disordered eating behaviors in a sample of female undergraduates. However, they did find that higher levels of disordered eating behaviors were related to higher levels of family dysfunction. These findings highlight the need to evaluate further the potential influence of both abuse and family functioning on disordered eating severity.

### *Physical and Emotional Abuse*

Far less research has addressed the contribution of physical or emotional abuse to disordered eating. However, in the few studies that have examined the association between nonsexual forms of abuse and disordered eating behaviors, researchers have generally found that individuals with eating disorders report higher rates of childhood physical and emotional abuse than do noneating disordered individuals. Rorty et al. (1994) found that rates of emotional and physical abuse were significantly higher among women with a current or previous diagnosis of bulimia nervosa than they were among women with no eating disorder history. In addition, they found that women with a current or previous diagnosis of bulimia nervosa were more likely to report experiencing multiple forms of childhood abuse than were noneating disordered women.

Similarly, in a recent study, Kent, Waller, and Dagnan (1999) investigated the relationship between diverse types of childhood abuse experiences and disordered eating behaviors in a nonclinical sample. They found that when multiple forms of abuse (i.e., sexual, physical, emotional, and neglect) were evaluated simultaneously (using regression), only emotional abuse was significantly related to disordered eating behaviors. However, although the association between emotional abuse and disordered eating behaviors remained significant, it was relatively small in magnitude ($t = 1.91$).[1] Thus, it appeared important to examine further the association between emotional abuse and disordered eating.

## Alexithymia and Disordered Eating Behaviors

The term *alexithymia* was first defined by Sifneos (1973), who used it to describe a cluster of characteristics he frequently observed in patients with psychosomatic symptoms, including "a relative constriction in emotional functioning" and "the inability to find appropriate words to describe . . . feelings" (p. 256). He further noted that these characteristics were often associated with a concrete cognitive style.

Clinicians and researchers have long noted that individuals with eating disorders typically have difficulty identifying and describing their emotions (e.g., Bruch, 1973). Indeed, Heatherton and Baumeister (1991) have proposed an escape theory of binge eating in which they argue that this behavior develops as an attempt to avoid self-awareness. Specifically, these authors suggested that binge eating may enable individuals to distract themselves from negative emotions by narrowing their focus of attention and making their cognitive processes more concrete.

Although Heatherton and Baumeister (1991) focused specifically on binge eating, they theorized that difficulties with emotions are an important clinical feature of women with bulimic and restricting symptoms. Some more recent studies have suggested that individuals who engage in the continuum of disordered eating behaviors have higher levels of alexithymia than do noneating disordered individuals (e.g., Cochrane, Brewerton, Wilson, &

[1] Kent et al. (1999) reported only the adjusted $R^2$ for the regression equation yielded when four types of abuse (sexual abuse, physical abuse, emotional abuse, and neglect, as measured by the Child Abuse and Trauma Scale; Sanders & Becker-Lausen, 1995) were used to predict scores on the eating scales of the EDI (Drive for Thinness, Bulimia, and Body Dissatisfaction). They noted that Emotional Abuse was the only abuse subscale that had a significant effect on EDI scores (and the corresponding $t$ was reported). However, these authors did not report a standardized beta coefficient. Kent et al. noted that the zero-order correlation between emotional abuse and eating pathology, as measured by a summary score on the eating scales of the EDI, was .24 ($p < .01$).

Hodges, 1993; Sexton, Sunday, Hurt, & Halmi, 1998). This association has been found in both clinical (e.g., Schmidt, Jiwany, & Treasure, 1993; Troop, Schmidt, & Treasure, 1995) and nonclinical (e.g., Laquatra & Clopton, 1994; G. J. Taylor, Parker, Bagby, & Bourke, 1996) samples. Moreover, Leon, Fulkerson, Perry, and Early-Zadd (1995) found that an inability to discriminate and label emotional states was a significant predictor of eating disorder risk status in their 3-year longitudinal study of adolescent girls.

Furthermore, we included alexithymia in the present study because it has not only been associated with disordered eating, but it has also been found to be correlated with various forms of trauma, including combat (Shipko, Alvarez, & Noviello, 1983) and rape (Zeitlin, McNally, & Cassiday, 1993). However, there have been relatively few empirical studies of the association between alexithymia and childhood abuse. In one study of undergraduates, Berenbaum and James (1994) found that high levels of alexithymia were associated with low levels of family expressiveness and with feeling emotionally unsafe in one's childhood environment.

Camras et al. (1988) found additional support for the association between childhood abuse, family functioning, and the ability to express and identify emotions. These authors examined the ability of abused and nonabused children and their mothers to recognize and produce emotional expressions. They found that abused children and their (abusive) mothers produced less recognizable emotional expressions than did nonabused children and their mothers. In addition, abused children had greater difficulty accurately recognizing emotional expressions.

Berenbaum (1996) found that clients in an outpatient sample (with heterogeneous problems) who reported a history of childhood abuse had more difficulty identifying their emotions than did nonabused clients, even when their scores on a measure of depression were controlled. On the basis of these results, Berenbaum suggested that alexithymia may mediate the association between abuse and psychological distress. Consequently, in the present study, we investigated this potential mediating role of alexithymia.

## Depression and Disordered Eating Behaviors

Previous researchers have found significant positive associations between disordered eating behaviors and depressive symptomatology (e.g., Mizes, 1988). Fairburn and Cooper (1984) found that a clinical sample of women with bulimia had depression scores similar to those of clients diagnosed with major depressive disorder. Depression and eating disorders co-occur so frequently (Herzog, 1982) that some have suggested that eating disorders are a form of affective disorder (e.g., Pope, Hudson, Jonas, & Yurgelun-Todd, 1983).

However, as Leon et al. (1995) have noted, the role of depression in the etiology of disordered eating remains unclear. For example, depressive symptoms may precede disordered eating; however, other explanations for the association between depression and disordered eating appear equally plausible. For example, depression can be a consequence of nutritional abnormalities (e.g., Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950; Laessle, Schweiger, & Pirke, 1988) or of the self-denigration that typically follows disordered eating behaviors (Stice, 1998).

In addition to its potential association with disordered eating, depression has also been found to be significantly associated with other variables included in this study, particularly with alexithymia (e.g., Bagby, Taylor, & Ryan, 1986). Thus, including depression in the present investigation facilitated the examination of the contributions of other variables when depression was taken into account.

## Summary and Hypotheses

Despite the fact that the number of studies investigating correlates of disordered eating has increased rapidly over the past several decades, there remains a need to extend this research to examine potential mediating relationships among family functioning, childhood abuse, and disordered eating behaviors. In addition, research on correlates of the continuum of disordered eating behaviors is particularly needed, as previous studies have consistently demonstrated that subclinical forms of eating disorders are not only far more prevalent than clinical anorexia and bulimia nervosa but are also associated with significant psychological distress (e.g., Dancyger & Garfinkel, 1995; Mintz & Betz, 1988). Consequently, in the present investigation, we tested models of the correlates of disordered eating (using SEM) in two samples of undergraduate women. To provide a more rigorous test of the hypothesized associations among these variables and limit specification error, we tested alternative models (as recommended by Quintana & Maxwell, 1999).

The first model we evaluated was the full model. In this model, we hypothesized that low levels of family cohesion and high levels of family conflict would be associated with higher levels of childhood abuse. In addition, we hypothesized that family cohesion would be negatively associated with alexithymia. We also hypothesized that childhood abuse would be positively associated with disordered eating behaviors. Further, we hypothesized that depression would be positively associated with alexithymia. Finally, we hypothesized that depression and alexithymia would mediate the association between abuse and disordered eating.

We also hypothesized two alternative, nested models on the basis of the results of previous research. In the first nested model, we deleted the path from family cohesion to alexithymia. We chose this path for initial deletion because, although previous research has indicated that family environment can influence the emotional development of young children (Camras et al., 1988), there was, to our knowledge, relatively limited support for this association in adults (compared with the support for other hypothesized associations in the model) and no research on the association between family cohesion, alexithymia, and disordered eating. Consequently, this hypothesis was more exploratory in nature than were other associations tested in these models. We also hypothesized a second nested model in which we deleted the direct path from abuse to disordered eating. We tested this nested model to expand on previous research, which has yielded conflicting results regarding the association between abuse and disordered eating. In this model, we hypothesized that the associations between abuse and disordered eating would be fully mediated by depression and alexithymia. Given that these three hypothesized models were relatively exploratory in nature, we did not identify one a priori as most likely to provide the best fit (in both a theoretical and empirical sense). However, on the basis of the results yielded in the validation sample, the best-fitting model was cross-validated. We hypothesized that this best-fitting model would provide a good fit to the data of both the validation and cross-validation samples (i.e., the entire root-mean-square error of approximation [RMSEA]

confidence interval would be below .08, as recommended by Quintana & Maxwell, 1999).

## Method

### *Participants*

Participants were 820 undergraduate female volunteers from educational and introductory psychology classes and sororities at a large midwestern university. Participants in psychology classes received course credit for their involvement and were entered in a raffle for a cash prize in exchange for their participation. Sorority members were offered the opportunity to complete the questionnaires at the beginning of chapter meetings that had an educational component and were also eligible for the raffle. Women were studied exclusively because eating disorders are far more prevalent in women than they are in men (Carlat & Camargo, 1991; Garfinkel & Garner, 1982; Mintz & Betz, 1988; Striegel-Moore, Garvin, Dohm, & Rosenheck, 1999). In addition, sorority members were specifically targeted for inclusion, as several authors have suggested that disordered eating behaviors are more prevalent in the sorority setting (e.g., Crandall, 1989; Schulken, Pinciaro, Sawyer, Jensen, & Hoban, 1997).

In addition to the measures described below, participants completed a demographic questionnaire. They represented the following ethnic–racial groups: 81.9% Caucasian, 6.2% Latina, 5.5% Asian American or Asian, 3.8% African American, and 1.2% Native American. Five participants (0.6%) reported that they were biracial or belonged to another ethnic or racial group, and six (0.7%) did not report their ethnicity. With respect to year in school, 35.0% were first-year students, 36.1% were second-year students, 16.5% were third-year students, and 12.3% were fourth-year students. One participant did not report her year in school (0.1%). Approximately half of the sample indicated that they were members of a sorority (50.5%). Participants' mean age was 19.1 years ($SD = 1.1$, range = 17 to 22 years). Their mean height was 65.3 in. ($SD = 2.7$, range = 58 to 74 in.), and their mean weight was 134.7 lbs (61.1 kg; $SD = 21.2$, range = 90 to 285 lbs [40.8 to 129.3 kg]). Participants' self-report of their height and weight was used to calculate body mass index (BMI; [weight in kilograms/height in meters]$^2$). According to the World Health Organization (WHO, 1998, as cited in Stevens, Cai, Thun, & Wood, 2000), BMI can be interpreted using the following categories: underweight (BMI $<$ 18.5), normal weight (BMI = 18.5 to 24.9), preobese or overweight (BMI = 25.0 to 29.9), and obese (BMI = 30.0 or above). Results of a meta-analysis indicated that self-report is a valid method of assessing weight in nonclinical samples (Bowman & DeLucia, 1993). Mean BMI was 22.2 ($SD = 3.2$); BMI ranged from 15.7 to 46.0, indicating that the sample included both underweight and severely overweight participants.

The overall sample was randomly divided into validation and cross-validation subsamples following examination of missing data (discussed below in the *Procedure* section) and prior to conducting the modeling analyses. Each subsample included 406 participants. Results of chi-square tests of independence and *t* tests indicated that there were no significant differences between subsamples in age, $t(809) = -0.59$, $p > .10$; BMI, $t(803) = -1.30$, $p > .10$; year in school, $\chi^2(3, N = 811) = 0.53$, $p > .10$; or sorority membership, $\chi^2(1, N = 812) = 0.32$, $p > .10$. To assess potential ethnic–racial differences across subsamples, we first conducted a chi-square test of independence using the six ethnic groups represented in this study. This analysis indicated that there were no significant ethnic–racial differences across subsamples, $\chi^2(5, N = 806) = 3.92$, $p > .10$. However, because of the small cell sizes for some ethnic groups, these results were considered somewhat tentative, as one of the assumptions of the Pearson chi-square test is that expected cell frequencies are of reasonable size (Weinberg & Goldberg, 1990). Consequently, given that the vast majority of the sample was Caucasian, and our primary concern was that members of ethnic minority groups were equally represented in both subsamples, we conducted a second chi-square test of independence, in which ethnicity–race was dichotomized (i.e., Caucasian, non-Caucasian). Results again indicated that there were no differences in ethnicity across subsamples, $\chi^2(1, N = 796) = 0.02$, $p > .10$, providing evidence that the validation and cross-validation samples shared similar demographic characteristics.

### *Measures*

*Family Environment Scale (FES, Form R).* The Cohesion and Conflict subscales of the FES (Moos, 1974; Moos & Moos, 1994) were used to measure family functioning. The FES is a 90-item true–false measure of perceived family environment that contains 10 subscales. The Cohesion and Conflict subscales were selected for use in this study on the basis of both on their demonstrated psychometric properties (discussed below) and the fact that they have been used frequently in previous eating disorders research.

The Cohesion subscale assesses "the extent to which family members are concerned and committed to the family and the degree to which they are helpful and supportive to each other" (Moos & Moos, 1976, p. 360). The Conflict subscale measures "the extent to which open expression of anger and aggression and generally conflictual interactions are characteristic of the family" (Moos & Moos, 1976, p. 360). Each subscale contains nine items.

Normative data for Form R were developed using samples of distressed and nondistressed families. The Cohesion and Conflict subscales were found to yield reliable scores. Estimates of internal consistency were .75 and .78 for Conflict and Cohesion, respectively. Two-month test–retest reliabilities were .85 for Conflict and .86 for Cohesion (Moos & Moos, 1994). Moreover, distressed families scored higher on Conflict and lower on Cohesion than nondistressed families did, even when family background attributes such as socioeconomic status and number of children were controlled (Moos, 1974), thus providing evidence of the measure's discriminative validity. In addition, high Conflict and low Cohesion scores have been found to be predictive of drug use among adolescents (Andrews, Hops, Ary, Lichtenstein, & Tildesley, 1991). In the present study, internal consistency estimates (Kuder Richardson-20) for the Cohesion subscale were .79 in the validation sample and .78 in the cross-validation sample. Internal consistency estimates (Kuder Richardson-20) were similar for the Conflict subscale in both the validation (.77) and cross-validation (.76) samples.

*Childhood Trauma Questionnaire (CTQ).* Abuse history was measured by the CTQ (Bernstein et al., 1994), a 28-item, self-report measure that assesses a range of traumatic childhood experiences. The CTQ was designed to describe childhood trauma in an objective manner; therefore terms such as *abuse* are kept to a minimum (Bernstein et al., 1994). CTQ items begin with the stem, "When I was growing up" and are rated on a 5-point scale, ranging from 1 (*never true*) to 5 (*very often true*). The measure was initially developed in a sample of adults receiving substance abuse treatment (Bernstein et al., 1994). Its psychometric properties have subsequently been evaluated in samples of nonclinical undergraduates, adult psychiatric outpatients, and female members of a health maintenance organization (Bernstein & Fink, 1998).

The 28-item CTQ, a shortened version of the original 70-item CTQ (Bernstein et al., 1994), is composed of six subscales: Emotional Abuse, Physical Abuse, Sexual Abuse, Emotional Neglect, Physical Neglect, and Minimization/Denial. The Minimization/Denial subscale was designed to identify individuals with a tendency to respond in a socially desirable manner. Each abuse subscale is composed of five items; the Minimization/Denial subscale contains three items.

In an undergraduate sample, internal consistency estimates (alpha coefficients) were .60 for Physical Neglect, .72 for Sexual Abuse, .78 for Physical Abuse, .89 for Emotional Abuse, and .92 for Emotional Neglect (Bernstein & Fink, 1998). The CTQ has also demonstrated adequate test–retest reliability. The stability of CTQ scores was assessed in a clinical sample (Bernstein et al., 1994). Test–retest coefficients (obtained at a mean

interval of 3.6 months, $SD = 1.0$) for the abuse subscales ranged from .79 (Physical Neglect) to .81 (Emotional Neglect and Sexual Abuse).

The construct validity of the 28-item CTQ was evaluated using confirmatory factor analyses (Bernstein & Fink, 1998) of the items from the five abuse subscales. Results indicated that a five-factor model provided a good fit for the data of three distinct samples. The CTQ was also significantly correlated with an interview measure of childhood abuse, the Childhood Trauma Interview (Bernstein et al., 1994), providing evidence of the measure's convergent validity. In addition, in an undergraduate sample, scores on the abuse subscales were only modestly associated with a measure of social desirability (Bernstein & Fink, 1998). In the present study, internal consistency estimates (alpha coefficients) for the CTQ subscales in the validation sample were as follows: .53 for Physical Neglect, .73 for Physical Abuse, .83 for Emotional Abuse, .89 for Emotional Neglect, and .92 for Sexual Abuse. Similarly, in the cross-validation sample, Cronbach's alpha coefficients were .58 for Physical Neglect, .77 for Physical Abuse, .81 for Emotional Abuse, .89 for Emotional Neglect, and .93 for Sexual Abuse.

*Toronto Alexithymia Scale (TAS–20).* Alexithymia was measured by the TAS–20 (Bagby, Parker, & Taylor, 1994; Bagby, Taylor, & Parker, 1994), a 20-item, self-report measure composed of three subscales: Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF), and Externally Oriented Thinking (EOT). Items are rated on a 5-point scale, ranging from 1 (*strongly disagree*) to 5 (*strongly agree*). Sample items include the following: "I am often confused about what emotion I am feeling" (DIF), "People tell me to describe my feelings more" (DDF), and "I prefer talking to people about their daily activities rather than their feelings" (EOT).

The TAS–20 was developed in a sample of 965 undergraduate students (Bagby, Parker, et al., 1994). Both exploratory and confirmatory factor analyses supported the construct validity of the three subscales (Bagby, Parker, et al., 1994). In addition, the internal consistency of each subscale was adequate (alpha coefficients were .78 for DIF, .75 for DDF, and .66 for EOT), as was the 3-week test–retest reliability of the overall TAS–20 ($r = .77$). These psychometric properties were further supported in two validation samples (Bagby, Taylor, et al., 1994). In addition, the TAS–20 exhibited discriminant validity (Bagby, Taylor, et al., 1994). Specifically, TAS–20 scores were negatively correlated with measures of psychological mindedness and openness to experience and uncorrelated with agreeableness and conscientiousness. In the present study, coefficient alpha for the DIF subscale was .83 in the validation sample and .86 in the cross-validation sample. For the DDF subscale, coefficient alpha was .81 in the validation sample and .80 in the cross-validation sample, and for the EOT subscale, coefficient alpha was .70 in the validation sample and .63 in the cross-validation sample.

*Center for Epidemiological Studies Depression Scale (CES–D).* The CES–D (Radloff, 1977) is a 20-item scale designed to measure current levels of depressive symptomatology in the general population. Respondents are asked to rate the frequency of each symptom over the past week on a 4-point scale, ranging from 1 (*rarely*) to 4 (*all of the time*). Higher scores indicate more severe depressive symptoms.

Previous research has found that the CES–D yields internally consistent scores (e.g., Espelage, Quittner, Sherman, & Thompson, 2000; Radloff, 1977). Factor analysis of the CES–D in the derivation sample yielded four factors: Depressed Affect, Positive Affect, Somatic and Vegetative Activity, and Interpersonal Symptoms (Radloff, 1977). Sheehan, Fifield, Reisine, and Tennen (1995) used a confirmatory factor analytic approach and found that the four-factor structure was stable over a 2-year period.

The CES–D has been found to discriminate effectively between depressed and nondepressed individuals (e.g., Radloff, 1977). It also exhibits convergent validity with other measures of depression (Radloff, 1977). A recent investigation found the CES–D to be a better predictor of depressive symptoms than the BDI in a college sample (Santor, Zuroff, Ramsay, Cervantes, & Palacios, 1995). In the present study, the four subscales of the CES–D yielded internally consistent scores. In the validation sample, Cronbach's alpha coefficients were .69 for the Somatic and Vegetative Activity subscale, .66 for the Interpersonal Symptoms subscale, .80 for the Positive Affect subscale, and .87 for the Depressed Affect subscale. Similarly, in the cross-validation sample, Cronbach's alpha coefficients were .72 for the Somatic and Vegetative Activity subscale, .74 for the Interpersonal Symptoms subscale, .81 for the Positive Affect subscale, and .86 for the Depressed Affect subscale.

*Eating Attitudes Test—26 (EAT–26).* Disordered eating behaviors were assessed using the EAT–26, an abbreviated version of the original 40-item EAT. This self-report measure consists of 26 items designed to assess eating disorder symptomatology (Garner & Garfinkel, 1979). The EAT–26 can be used as a continuous measure of eating disturbances in a nonclinical population (Koslowsky et al., 1992; Mintz & O'Halloran, 2000). Items are rated on a 6-point frequency scale. Responses range from 1 (*never*) to 6 (*always*). The higher the score, the more symptomatic the respondent. However, Garner, Olmsted, Bohr, and Garfinkel (1982) recommended that the responses *never*, *rarely*, and *sometimes* receive a score of 0 and that the responses *often*, *very often*, and *always* receive scores of 1, 2, and 3, respectively.

Factor analysis of the EAT–26 indicated that the measure is composed of three factors: Dieting, Bulimia and Food Preoccupation, and Oral Control (Garner et al., 1982). Garner et al. reported Cronbach's alpha coefficients of .86 for Dieting, .61 for Bulimia and Food Preoccupation, and .46 for Oral Control in a sample of noneating disordered women and .90, .84, and .83, respectively, in a sample of women with anorexia. The factor structure of the measure has also been replicated (Koslowsky et al., 1992). In the present study, coefficient alpha for the Dieting subscale was .88 in the validation sample and .89 in the cross-validation sample. For the Bulimia and Food Preoccupation subscale, coefficient alpha was .82 in the validation sample and .76 in the cross-validation sample. Coefficient alpha for the Oral Control subscale was .38 in the validation sample and .53 in the cross-validation sample.

*Bulimia Test—Revised (BULIT–R).* Disordered eating was also assessed using the BULIT–R, a 28-item self-report questionnaire designed to assess bulimic behaviors (Thelen, Farmer, Wonderlich, & Smith, 1991). The BULIT–R is a revision of the original BULIT (Smith & Thelen, 1984) and has been found to be a valid instrument for identifying individuals who meet *DSM–IV* criteria for bulimia nervosa (Thelen, Mintz, & Vander Wal, 1996). The BULIT–R's developers (Thelen et al., 1991) have noted that this measure is a particularly useful and cost-effective means of investigating the frequency of bulimic behaviors in nonclinical populations where the relative prevalence of the disorder is low (Thelen et al., 1991). The BULIT–R was validated in five stages. In total, the measure was evaluated on the basis of the scores of 93 women with bulimia nervosa and 2,477 nonbulimic females. Items are rated on a scale ranging from 1 to 5, with the most symptomatic response receiving a score of 5. Thelen et al. (1991) found that the 2-month test–retest reliability of the measure was .95. Furthermore, a significant difference was found between scores of participants with bulimia and those of noneating disordered participants (Thelen et al., 1991). Subsequent research has provided additional evidence of the reliability and validity of BULIT–R scores (e.g., Welch, Thompson, & Hall, 1993). In the present study, coefficient alpha for the BULIT–R was .95 in both the validation and cross-validation samples.

## *Procedure*

Participants in psychology classes signed up for time periods of 30 min. Sorority members completed the questionnaires at the beginning of chapter meetings with an educational component. After consent was obtained, measures were administered in a randomized sequence to control for order effects. Participants were told that the purpose of the study was to examine the associations among mood, relationships, and behavior. Following completion of the measures, participants were given a written debriefing form,

which explained study objectives and included information about campus resources that could assist them with any concerns they may have regarding their own eating habits, body image, mood, or relationships.

*Missing-data imputation.* Data were discarded for all respondents who completed less than 50% of the items on any given scale or subscale. For participants with modest amounts of missing data, item means (rounded to their integer value) were substituted for missing responses if a respondent omitted one item on a short scale (10 items or fewer) and up to two items on longer scales (more than 10 items). No imputation was used when more than two items were missing on short scales or three or more items were missing on longer scales; these participants were dropped from the analyses. This data imputation method has been found to be quite effective for factor analysis (Finkbeiner, 1979). Using this approach, we deleted 4 participants from the analyses because of missing data. We dropped 3 additional participants from the analyses because of response inconsistencies (e.g., reporting gender as male). We deleted another participant from the analyses because she was 34-years-old and married and, thus, differed from the traditional-age college students who were the primary focus of this study. Consequently, the initial sample of 820 was reduced to 812.

## Results

### *Descriptive Statistics*

Means and standard deviations of scores on each of the measures used in the modeling analyses are presented in Table 1. Prior to conducting SEM, we analyzed the frequency and type of abusive childhood experiences reported on the CTQ because previous research has suggested that the prevalence of childhood sexual abuse ranges from approximately 10% to 30% (e.g., Finkelhor, 1984); thus, we anticipated that CTQ scores may be skewed. Although the CTQ is scored on a 5-point scale and we used these polytomous scores in all modeling analyses, we calculated the number of participants endorsing at least one item on each CTQ subscale to evaluate the prevalence of childhood abuse in these samples. These data (displayed in Table 2) indicated that, in both the validation and cross-validation samples, emotional abuse was the most frequently reported type of childhood abuse experience. Nearly two thirds of both samples endorsed at least one item on this subscale. Emotional neglect was the second most frequently endorsed type of childhood trauma, with more than 60% of respondents in both samples endorsing at least one of the experiences included on this subscale. In contrast, less than 10% of participants in either sample reported that they had experienced childhood sexual abuse. Participants' mean score on the Minimization/Denial subscale was .50 ($SD$ = .89) in the validation sample and .63 in the cross-validation sample ($SD$ = .95). This score is comparable with that obtained in the undergraduate sample used in the validation of the CTQ (Bernstein & Fink, 1998) and suggests

Table 1
*Means, Standard Deviations, and Bivariate Correlations of Indicators*

| Indicator | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Cohesion–odd | — | .66 | −.53 | −.33 | −.51 | −.25 | −.68 | −.29 | −.23 | −.22 | −.08 | −.27 | −.33 | −.21 | −.25 | −.15 | .06 | .01 | −.07 |
| 2. Cohesion–even | .64 | — | −.55 | −.38 | −.52 | −.23 | −.66 | −.28 | −.17 | −.16 | −.07 | −.25 | −.27 | −.18 | −.20 | −.11 | .05 | .05 | −.04 |
| 3. Conflict–odd | −.53 | −.49 | — | .60 | .57 | .26 | .53 | .25 | .20 | .12 | .02 | .30 | .30 | .23 | .24 | .21 | −.00 | .04 | .12 |
| 4. Conflict–even | −.41 | −.38 | .67 | — | .46 | .25 | .39 | .21 | .16 | .12 | .11 | .26 | .20 | .15 | .16 | .13 | −.01 | .04 | .11 |
| 5. Emot. Abuse | −.48 | −.40 | .56 | .51 | — | .51 | .69 | .37 | .31 | .21 | .04 | .38 | .31 | .32 | .41 | .25 | −.02 | .06 | .14 |
| 6. Phys. Abuse | −.24 | −.19 | .26 | .28 | .51 | — | .41 | .37 | .14 | .12 | .07 | .22 | .19 | .21 | .31 | .09 | .00 | .05 | .02 |
| 7. Emot. Neglect | −.71 | −.60 | .57 | .43 | .60 | .32 | — | .48 | .30 | .26 | .09 | .32 | .40 | .29 | .31 | .18 | −.04 | −.00 | .08 |
| 8. Phys. Neglect | −.36 | −.28 | .26 | .20 | .36 | .33 | .47 | — | .22 | .20 | .11 | .24 | .28 | .22 | .26 | .21 | −.05 | .06 | .10 |
| 9. DIF | −.25 | −.24 | .22 | .25 | .25 | .10 | .31 | .24 | — | .62 | .24 | .40 | .43 | .38 | .43 | .31 | −.04 | .15 | .22 |
| 10. DDF | −.22 | −.18 | .14 | .09 | .21 | .06 | .34 | .17 | .65 | — | .37 | .27 | .34 | .22 | .27 | .16 | −.00 | .05 | .04 |
| 11. EOT | −.20 | −.12 | .07 | .03 | .10 | .06 | .21 | .12 | .27 | .44 | — | .10 | .14 | −.01 | .06 | .01 | −.05 | −.04 | −.01 |
| 12. Somatic | −.23 | −.25 | .23 | .23 | .28 | .18 | .29 | .17 | .51 | .34 | .12 | — | .55 | .68 | .55 | .27 | .03 | .16 | .21 |
| 13. Positive Affect[a] | −.26 | −.21 | .23 | .20 | .28 | .19 | .32 | .22 | .41 | .30 | .18 | .58 | — | .67 | .51 | .23 | −.03 | .15 | .12 |
| 14. Depress. Affect | −.21 | −.22 | .25 | .22 | .26 | .22 | .27 | .20 | .52 | .29 | .06 | .67 | .65 | — | .66 | .30 | .05 | .27 | .23 |
| 15. Interpersonal | −.27 | −.28 | .32 | .32 | .37 | .27 | .36 | .28 | .47 | .31 | .11 | .51 | .53 | .61 | — | .29 | .01 | .19 | .19 |
| 16. BULIT–R | −.17 | −.13 | .17 | .20 | .25 | .13 | .20 | .20 | .37 | .22 | .20 | .33 | .31 | .35 | .39 | — | −.03 | .73 | .79 |
| 17. EAT–Oral | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | — | .07 | .05 |
| 18. EAT–Diet | −.09 | −.03 | .09 | .12 | .12 | .03 | .04 | .09 | .20 | .06 | .07 | .19 | .21 | .21 | .20 | .71 | — | — | .71 |
| 19. EAT–Bulimia | −.15 | −.11 | .12 | .16 | .19 | .07 | .10 | .13 | .23 | .10 | .08 | .21 | .25 | .25 | .28 | .73 | — | .72 | — |
| $M$[b] | 3.83 | 3.15 | 1.80 | 1.45 | 7.48 | 5.86 | 7.61 | 6.02 | 15.00 | 12.11 | 17.00 | 6.75 | 2.83 | 4.05 | 2.11 | 50.37 | 1.66 | 6.09 | 1.14 |
| $SD$[b] | 1.35 | 1.10 | 1.41 | 1.12 | 3.14 | 1.98 | 3.29 | 1.75 | 5.72 | 4.71 | 4.71 | 3.65 | 2.56 | 3.52 | 2.11 | 18.91 | 1.96 | 6.96 | 2.58 |
| $M$[c] | 3.91 | 3.24 | 1.63 | 1.38 | 7.56 | 5.89 | 7.66 | 5.92 | 14.97 | 11.83 | 16.57 | 7.02 | 2.78 | 4.31 | 2.08 | 52.66 | — | 6.81 | 1.41 |
| $SD$[c] | 1.35 | 1.03 | 1.31 | 1.12 | 3.24 | 2.27 | 3.39 | 1.87 | 6.15 | 4.60 | 4.24 | 3.77 | 2.60 | 3.47 | 2.24 | 19.26 | — | 7.47 | 2.59 |

*Note.* Validation sample ($n$ = 406) appears above the diagonal; cross-validation sample ($n$ = 406) appears below the diagonal. Cohesion–odd = sum of odd items of the Cohesion subscale of the Family Environment Scale (FES); Cohesion–even = sum of even items of the Cohesion subscale of the FES; Conflict–odd = sum of odd items of the Conflict subscale of the FES; Conflict–even = sum of even items of the Conflict subscale of the FES; Emot. Abuse = Emotional Abuse subscale of the Childhood Trauma Questionnaire (CTQ); Phys. Abuse = Physical Abuse subscale of the CTQ; Emot. Neglect = Emotional Neglect subscale of the CTQ; Phys. Neglect = Physical Neglect subscale of the CTQ; DIF = Difficulty Identifying Feelings subscale of the Toronto Alexithymia Scale—20 (TAS–20); DDF = Difficulty Describing Feelings subscale of the TAS–20; EOT = Externally Oriented Thinking subscale of the TAS–20; Somatic = Somatic and Vegetative subscale of the Center for Epidemiological Studies Depression Scale (CES–D); Positive Affect = Positive Affect subscale of the CES–D; Depress. Affect = Depressed Affect subscale of the CES–D; Interpersonal = Interpersonal subscale of the CES–D; BULIT–R = Bulimia Test—Revised; EAT–Oral = Oral Control subscale of the Eating Attitudes Test—26 (EAT–26); EAT–Diet = Dieting subscale of the EAT–26; EAT–Bulimia = Bulimia and Food Preoccupation subscale of the EAT–26.

[a] Higher scores on the Positive Affect subscale of the CES–D indicate lower levels of positive affect. [b] Descriptives for validation sample ($n$ = 406). [c] Descriptives for cross-validation sample ($n$ = 406).

Table 2
*Frequency of Childhood Abuse Experiences*

| | Sample (%) | |
|---|---|---|
| CTQ scale | Validation *f* | Cross-validation *f* |
| Emotional Abuse | 65.8 | 66.3 |
| Physical Abuse | 31.8 | 30.8 |
| Sexual Abuse | 8.1 | 9.6 |
| Emotional Neglect | 61.1 | 61.8 |
| Physical Neglect | 40.1 | 34.0 |

*Note.* Percentages reported indicate the proportion of respondents who endorsed at least one item on the respective Childhood Trauma Questionnaire (CTQ) subscale.

that, in the present study, CTQ responses were not unduly influenced by social desirability.

On the basis of the infrequency of childhood sexual abuse reported, this type of abuse was not included in the remaining analyses. This decision was based on the fact that, because of the low base rate for these items, scores on this scale would be too skewed to provide stable results.

### *SEM*

We conducted SEM using LISREL 8.30 (Jöreskog & Sörbom, 1999a). We used the data of the validation sample ($n = 406$) to test the relative fit of measurement model. Because of the skewness of several of the indicators (e.g., EAT–26, Physical Abuse, Depressed Affect), we normalized all indicators using the normal scores transformation function included in PRELIS 2.3 (Jöreskog & Sörbom, 1999b). We used the maximum-likelihood estimation method in all modeling analyses. Correlations among the indicators for the validation sample are reported in Table 1.

*Measurement Model 1.* Anderson and Gerbing (1988) recommended that the adequacy of a proposed measurement model be evaluated prior to the simultaneous analysis of the measurement and structural components of a model. Thus, in the first modeling analysis we evaluated the fit of the measurement model using the data of the validation sample. We used scales (as opposed to individual items) as indicators in all analyses. Estimating a measurement model using subscales rather than items greatly reduces the number of parameters that must be estimated. In addition, the measurement properties of subscales are superior to those of single item indicators (Drasgow & Dorans, 1982). For those constructs with a single scale indicator (i.e., Cohesion and Conflict), we divided items into two parcels (composed of odd- and even-numbered items) to aid in model identification (as recommended by Kishton & Widaman, 1994).

Results indicated that this model provided a good fit for the data. Fit indices are presented in Table 3. Using Quintana and Maxwell's (1999) criteria, we rejected the null hypothesis of not a fair fit (i.e., the entire RMSEA confidence interval was below .08). In addition, less than 10% of the standardized residuals were greater than an absolute value of 3.0. Examination of the factor loadings revealed that the indicators all loaded significantly on their respective factors, with the exception of the Oral Control subscale of the EAT–26. It should be noted that this factor loading was not only nonsignificant, but it was also negative. This result is problematic, as the theory underlying this subscale suggests that this factor loading should be large and positive. In addition, as noted in the *Measures* section, this subscale also demonstrated low internal consistency. Consequently, on the basis of these results and given the number of indicators for the disordered eating construct, we dropped this indicator from the measurement model. We then evaluated this revised model in the next analysis.

*Measurement Model 2.* We tested Model 2, presented in Figure 1, using the procedures described above. We deleted the EAT–26 Oral Control subscale from this analysis. Measurement Model 2 is identical to Measurement Model 1 in all other respects. Results of this analysis indicated that this model provided a good fit for the data (see Table 3). We rejected the null hypothesis of not a fair fit. All factor loadings were significant and in the predicted direction, and less than 10% of the standardized residuals were greater than an absolute value of 3.0. Consequently, we used this measurement model in all subsequent analyses.

*Structural Model 1.* The first structural model analyzed was the full model (presented in Figure 2). Results indicated that this model fit the data well (see Table 3), and we rejected the null hypothesis of not a fair fit examination of the standardized residual matrix indicated that less than 8% were greater than an absolute value of 3.0, further supporting the model's adequacy. However, we had hypothesized that low levels of family cohesion would be associated with high levels of alexithymia. Yet, the path from cohesion to alexithymia was very small and not in the expected direction; indeed, these variables were virtually unrelated ($\beta = .06$, $t = 0.38$, $p > .05$). As was noted previously, there was relatively less empirical and theoretical support for this association in the literature. We had included this path in the model for exploratory purposes only, and we deleted it in the subsequent, nested model discussed below. In addition, the direct path from abuse to disordered eating behaviors was very small and not in the expected direction ($\beta = –.04$, $t = -0.57$, $p > .05$). However, because this path could have important theoretical implications for the model, we used an iterative approach to modification. Specifically, we deleted the less theoretically relevant path (i.e., cohesion to alexithymia) first, and we reexamined the association between abuse and disordered eating in the subsequent model (Structural Model 2). All other paths in this model were significant ($p < .05$), with the exceptions of the path from conflict to abuse ($t = 1.50$) and abuse to alexithymia ($t = 1.18$). Given that these paths had important theoretical and clinical relevance to the model (and examination of residuals and modification indices suggested that deleting them would not enhance the model's explanatory power), we retained them pending the results of further analyses.

*Structural Model 2.* This model, depicted in Figure 2, is identical to Structural Model 1, except that we deleted the path from cohesion to alexithymia. This model fit the data well, and we rejected the null hypothesis of not a fair fit. Less than 8% of the standardized residuals were greater than an absolute value of 3.0, further supporting the adequacy of the model. We compared the fit of this nested model with the fit of the full model (Structural Model 1) using a chi-square test, as outlined by Quintana and Maxwell (1999, p. 506). Results indicated that the full model did not provide a significantly better fit than the nested model, $\chi^2_{\text{comparison}}$ $(1, n = 406) = .15$, $p > .05$. Because the path from abuse to disordered eating behaviors remained very small and was not in the expected direction ($\beta = –.04$, $t = –0.57$, $p > .05$), we

Table 3
*Fit Indices for Measurement and Structural Models*

| Model | $\chi^2$ | *df* | CFI | GFI | NFI | NNFI | SRMSR | RMSEA | 90% CI for RMSEA |
|---|---|---|---|---|---|---|---|---|---|
| Measurement Model 1 | 308.24 | 137 | .95 | .93 | .90 | .93 | .05 | .056 | (.047, .064) |
| Measurement Model 2 (deleting EAT–Oral) | 290.83 | 120 | .94 | .93 | .91 | .93 | .05 | .059 | (.051, .068) |
| Structural Model 1 (Full model) | 300.53 | 125 | .94 | .92 | .91 | .93 | .05 | .059 | (.050, .067) |
| Structural Model 2 (deleting path from cohesion to alexithymia) | 300.68 | 126 | .94 | .92 | .91 | .93 | .05 | .059 | (.050, .067) |
| Structural Model 3 (deleting path from abuse to disordered eating) | 302.06 | 127 | .94 | .92 | .91 | .93 | .05 | .058 | (.050, .067) |
| Structural Model 4 (Cross-validation) | 345.32 | 127 | .94 | .91 | .90 | .93 | .05 | .065 | (.057, .073) |
| Structural Model 5 (Invariant) | 671.78 | 291 | .94 | .91 | .90 | .94 | .06 | .057 | (.051, .062) |

*Note.* Measurement Models 1 and 2 and Structural Models 1–3 analyzed the data of the validation sample ($n$ = 406). Structural Model 4 analyzed the data of the cross-validation sample ($n$ = 406). Structural Model 5 analyzed data from both samples ($N$ = 812). CFI = comparative fit index; GFI = goodness-of-fit index; NFI = normed fit index; NNFI = non-normed fit index; SRMSR = standardized root-mean-square residual; RMSEA = root-mean-square measure error of approximation; CI = confidence interval.

deleted it in the subsequent model. All other paths in the model were significant ($p < .05$), with the exception of the path from conflict to abuse ($t = 1.55$). Again, given this path's theoretical and clinical relevance, we retained it in the model.

*Structural Model 3.* This model, depicted in Figure 2, is identical to Structural Model 2, except that we deleted the direct path from abuse to disordered eating. Thus, this model tested whether the association between abuse and disordered eating was fully mediated by depression and alexithymia. This model provided a good fit to the data, as indicated by both the fit indices and the standardized residuals. We rejected the null hypothesis of not a fair fit. Less than 6% of the standardized residuals were greater than an absolute value of 3.0, further supporting the adequacy of the model. All paths were in the expected direction. In addition, all were significant, with the exceptions of the paths from conflict to abuse ($t = 1.58$) and from alexithymia to disordered eating ($t = 1.63$). We compared the fit of this nested model to the fit of the previous nested model (Structural Model 2), and results indicated that Model 2 did not provide a significantly better fit than did Model 3, $\chi^2_{\text{comparison}}$ (1, $n$ = 406) = 1.38, $p > .05$. Model 3 was thus considered the most parsimonious fit.[2]

*Structural Model 4—cross-validation.* On the basis of the strong results obtained for Structural Model 3, we evaluated the fit of this model in the cross-validation sample ($n$ = 406). Correlations among the indicators in the cross-validation sample are reported in Table 1. We used maximum-likelihood estimation to test the same pattern of fixed and free elements specified in Structural Model 3. As in the validation sample, we used normalized scores for each indicator. Results suggested that this model provided a good fit for the data of the cross-validation sample (see Table 3), and again we rejected the null hypothesis of not a fair fit.

*Structural Model 5—invariant model.* Given that the model had a similar form in both the validation and cross-validation samples, we subsequently conducted a test of model invariance. This invariant model provides a much more stringent test of model fit than does independent estimation (Bollen, 1989). In independent estimations, the same pattern of fixed and freed elements can be specified (as in Structural Models 3 and 4). However, in an invariant model, both samples are analyzed simultaneously, and all paths not only follow the same pattern but also are constrained to be equivalent. In the present model, we specified measurement (i.e., factor loadings), structural, and error paths as invariant across the two samples. The invariant model provided a good fit to the data; all factor loadings and path coefficients were in the expected direction, and we rejected the null hypothesis of not a fair fit (see Figure 2). In addition, each sample contributed relatively equally to the chi-square of the invariant model (validation sample contribution = 306.02, cross-validation sample contribution = 328.21). Taken together, the invariant measurement and structural results indicated that this model is equivalent across samples.

[2] In addition to the SEM analyses reported here, we tested whether alexithymia and depression fully mediated the association between abuse and disordered eating in the validation sample using the criteria outlined by Holmbeck (1997), as suggested by an anonymous reviewer. Results indicated that, although the direct association between abuse and disordered eating was significant when we included only these two variables in the model, this association became nonsignificant when we added alexithymia to the model. We found similar results when we evaluated the mediating role of depression. Overall, both alexithymia and depression met all of Holmbeck's (1997) criteria for mediation in the present sample. On the basis of these results and using the terms defined by Holmbeck, it seems appropriate to view alexithymia and depression as mediators of disordered eating, rather than simply indirect correlates. Complete results of these analyses are available from Suzanne E. Mazzeo on request.

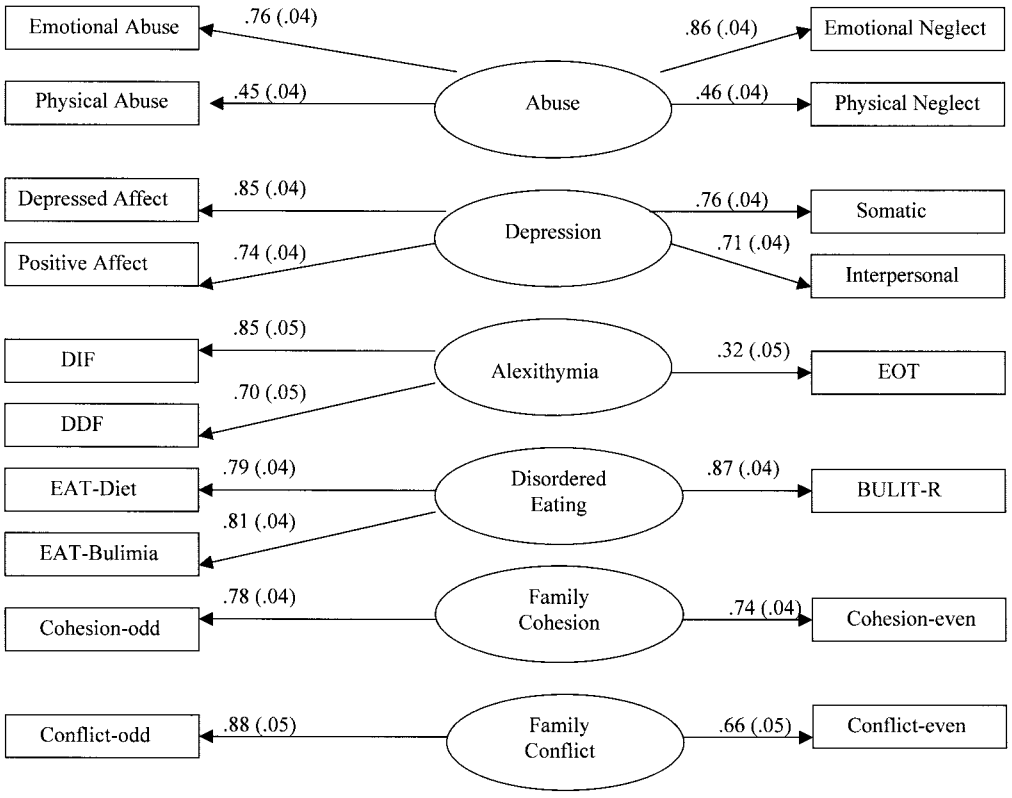


*Figure 1.* Measurement Model 2. Standard errors are in parentheses. Emotional Abuse = Emotional Abuse subscale of the Childhood Trauma Questionnaire (CTQ); Physical Abuse = Physical Abuse subscale of the CTQ; Emotional Neglect = Emotional Neglect subscale of the CTQ; Physical Neglect = Physical Neglect subscale of the CTQ; Depressed Affect = Depressed Affect subscale of the Center for Epidemiological Studies Depression Scale (CES–D); Positive Affect = Positive Affect subscale of the CES–D; Somatic = Somatic and Vegetative subscale of the CES–D; Interpersonal = Interpersonal subscale of the CES–D; DIF = Difficulty Identifying Feelings subscale of the Toronto Alexithymia Scale—20 (TAS–20); DDF = Difficulty Describing Feelings subscale of the TAS–20; EOT = Externally Oriented Thinking subscale of the TAS–20; EAT–Diet = Dieting subscale of the Eating Attitudes Test—26 (EAT–26); EAT–Bulimia = Bulimia and Food Preoccupation subscale of the EAT–26; BULIT–R = Bulimia Test—Revised; Cohesion–odd = sum of odd items of the Cohesion subscale of the Family Environment Scale (FES); Cohesion–even = sum of even items of the Cohesion subscale of the FES; Conflict–odd = sum of odd items of the Conflict subscale of the FES; Conflict–even = sum of even items of the Conflict subscale of the FES.

## Discussion

Over the past 2 decades, research on the correlates of disordered eating behaviors has increased exponentially. However, in spite of the quantity of literature addressing disordered eating, many questions about its etiology remain. In particular, several variables have been found to be significantly related to disordered eating behaviors. Yet the specific processes through which these variables potentially influence disordered eating have not been fully articulated, in part because researchers in this area have tended to rely heavily on univariate statistical approaches. In the present study, we used SEM to address some of the potential indirect relationships among variables previously found to be associated with disordered eating. The major finding of this study is that the associations among family conflict, family cohesion, childhood physical and emotional abuse and neglect, and college women's disordered eating behaviors were mediated by depression and alexithymia. These results both integrate and expand the results of previous studies. In particular, they contribute to researchers' understanding of the continuum of disordered eating behaviors as they occur in nonclinical college women.

Previous research on the relevance of family cohesion and conflict to disordered eating behavior has found that these variables are related to disordered eating behaviors in some nonclinical samples (e.g., Leung et al., 1996; Scalf-McIver & Thompson, 1989) and not in others (e.g., Kent & Clopton, 1992). The present results suggest that low levels of family cohesion and high levels of family conflict were indirectly associated with disordered eating behaviors. Low levels of family cohesion and high levels of family conflict were directly related to higher levels of childhood physical and emotional abuse and neglect. It should be noted, however, that (in both the validation and cross-validation samples), the influence of family cohesion on childhood abuse was much stronger than

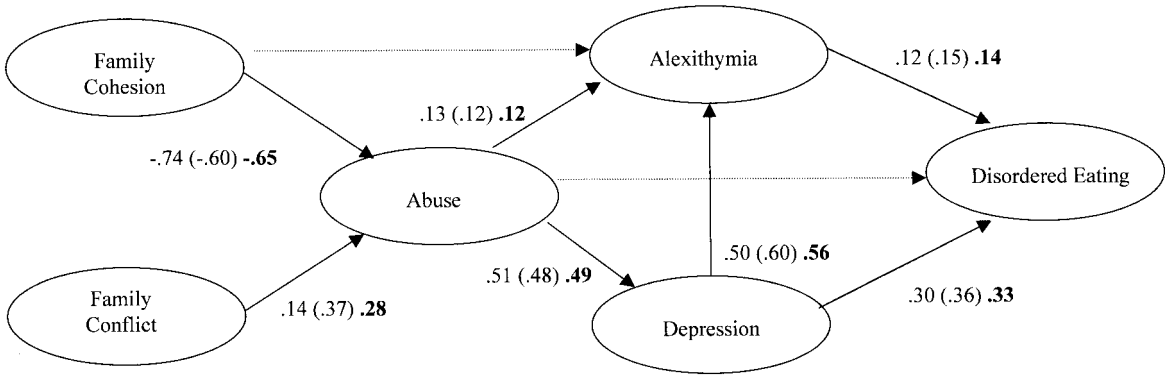


*Figure 2.* Structural Models 3, 4, and 5 are the final validation sample model, cross-validation model, and invariant model. Path coefficients for the validation sample appear in regular type. Coefficients for the cross-validation sample are in parentheses, and invariant model coefficients are in bold. Dotted lines represent paths tested in Structural Models 1 and 2 that we subsequently deleted.

that of family conflict. This suggests that family cohesion may be a particularly important aspect of family functioning to investigate further in future studies of the development of disordered eating. In addition, future research should evaluate the potential influence of other familial characteristics on disordered eating behaviors, including parental attachment and family constellation.

The present results differ from those of Kent and Clopton (1992), who found that family cohesion and conflict were not significantly associated with disordered eating. One reason for this difference may be because the present study included a much larger number of participants ($n$ = 406 participants per group vs. 72 participants divided into three groups); thus, there was much more power to detect differences in the present investigation. In addition, although Kent and Clopton studied a nonclinical sample, they used the BULIT (Smith & Thelen, 1984) to classify participants into three distinct groups: bulimic, subclinical bulimic, and symptom-free. In contrast, in the present study we examined disordered eating behaviors on a continuum and assessed a wider range of disordered eating behaviors, including dieting and restricting.

Present results regarding the association between childhood physical and emotional abuse and neglect and disordered eating also add to the literature, as few previous studies have examined the association between nonsexual forms of childhood abuse and disordered eating severity. Moreover, to our knowledge, no studies have evaluated the impact of these forms of childhood trauma in combination with the other variables included in this model. Results suggest that childhood physical and emotional abuse and neglect were not directly associated with disordered eating. Rather, these childhood experiences were indirectly related to disordered eating by means of alexithymia and depression.

These results complement those found in previous studies. For example, Ray et al. (1991) found that low levels of family cohesion were associated with increased risk of childhood sexual abuse both within and outside of the family environment. Present results suggest that low levels of cohesion were also associated with high rates of physical and emotional abuse and neglect. One potential explanation for this association may be that, if an individual from an incohesive family environment becomes a victim of abuse (whether inside or outside the home), she may be less likely to receive the support she needs from family members to prevent negative psychological outcomes. Future research could examine this hypothesis by expanding the present model to include social support as a moderator of the association between family cohesion and abuse.

Present results also extend researchers' knowledge of the role of alexithymia in disordered eating. Previous research has investigated the association between alexithymia and disordered eating (e.g., Laquatra & Clopton, 1994) and alexithymia and childhood abuse (e.g., Camras et al., 1988). This study's results suggest that alexithymia mediates the association between abuse and disordered eating, implying that individuals who have difficulty identifying and describing their emotions may be more likely to engage in disordered eating behaviors.

Further, these results may indicate that the intense focus on appearance (i.e., "turning outside") evident among women who engage in disordered eating behavior could be an attempt to cope with the difficulties they tend to have identifying and describing their emotions (i.e., "turning inside"). This result provides support for Heatherton and Baumeister's (1991) escape theory of disordered eating and suggests that emotion skills training may be an important addition to eating disorder prevention and treatment programs. Future research (particularly longitudinal research) is needed to further clarify the role of alexithymia in the development of disordered eating. Present results regarding the mediating role of alexithymia should not be overstated, given that, although this path contributed to the overall model, its coefficient was not particularly large in either subsample or in the invariant model. Nonetheless, as indicated in footnote 2, a test of mediation as outlined by Holmbeck (1997) suggested that alexithymia fully mediated the association between childhood abuse and disordered eating.

The present results also expand the understanding of the role of depression as a mediator of the association between childhood physical and emotional abuse and neglect and disordered eating behaviors. These results differ from those of Kent et al., 1999, who found that depression did not mediate the relationship between emotional abuse and disordered eating behaviors. However, these authors used multiple regression analyses to test for mediation. Unlike SEM, multiple regression does not identify measurement

problems that may influence results. The use of the more powerful SEM approach in the present study provided a more rigorous test of the role of depression in disordered eating behaviors.

An alternative explanation for why the present results differ from those of Kent et al. (1999) is that these authors also used measures of anxiety and dissociation in their study and found that depression was not a significant mediator of the association between childhood emotional abuse and disordered eating when anxiety and dissociation were taken into account. Unfortunately, we did not include measures of anxiety and dissociation in the present study; it is important to combine them with the variables used in the present model in future research.

Nonetheless, present results suggest that individuals who engage in disordered eating behaviors, as well as individuals at risk for developing these behaviors, may benefit from interventions that address adaptive ways to cope with depression. Taken together, results regarding the associations among alexithymia, depression, and disordered eating suggest that it is not the mere presence or absence of childhood emotional and physical abuse and neglect that is associated with disordered eating. Rather, the development of alexithymia and depressive symptomatology in response to these childhood experiences seems to be most strongly associated with disordered eating severity.

Overall, these results indicate that several factors simultaneously influence the severity of disordered eating. This suggests that there is a need for a holistic approach to the assessment of disordered eating behaviors. For example, these data indicate that low levels of family cohesiveness and, to a lesser degree, high levels of family conflict are associated with a greater incidence of emotional and physical abuse and neglect. Thus, practitioners working with college women with disordered eating behaviors should evaluate not only the presence or absence of cohesiveness and conflict in the student's family of origin but also the consequences of an incohesive or conflictual family environment (i.e., did this student's family environment include abuse or increase her vulnerability to abuse outside the home?). Furthermore, when asking women about their experiences of abuse and neglect, practitioners should attempt to determine whether these individuals may have developed alexithymic or depressive symptomatology in response to experiences of abuse or neglect, as the present results highlight the influence of these factors on disordered eating.

The major strength of this study is its test of alternative multivariate models of the correlates of disordered eating. Previous research has not examined the simultaneous influence of the variables included in these models. Additional strengths of this study include the cross-validation of the final model (including a test of model invariance), large sample size, oversampling of sororities, the assessment of multiple types of childhood abuse, and the use of measures with strong psychometric support. Finally, the use of SEM in this study facilitated the identification of measurement problems in the models tested.

Nonetheless, we should note several methodological limitations of this study. First, the models evaluated in this investigation did not include a measure of sexual abuse. Because of the relative infrequency of childhood sexual abuse in these samples, this type of abuse could not be included in the modeling analyses. Thus, the potential role of childhood sexual abuse within a structural model of disordered eating behavior needs to be evaluated in future studies.

Second, because this study assessed abuse and family environment retrospectively, participants' recollections were subject to recall bias (Briere, 1992; Kinzl et al., 1994). However, Brewin, Andrews, and Gotlib (1993) have noted that the use of a structured assessment tool that asks about a range of specific abusive events enhances the reliability of participants' recollections. The measure of childhood abuse used in the present study met these criteria.

An additional limitation is that all measures used in this study were self-reports. Thus, results may be affected by monomethod bias (Cook & Campbell, 1979). In particular, participants' self-reports of their family-of-origin environments may differ from results yielded by direct observation measures. Also, given that a core feature of eating disorders is secrecy about the symptoms themselves, some participants may not have been completely honest in reporting their eating and weight-related behaviors.

Another possible limitation of this study is that internal consistency estimates were low for some of the measures, particularly for the Physical Neglect subscale of the CTQ. However, this measure demonstrated convergent validity with the Emotional Neglect subscale of the CTQ, and results of the measurement model provided evidence of its factorial validity. In addition, as noted in the *Measures* section, previous research has found support for the stability of the Physical Neglect subscale (Bernstein et al., 1994). These indices of reliability and validity appear to be more critical than internal consistency for the purposes of the present study.

In addition, these samples were not necessarily representative, as participants were recruited from their classes or sororities. However, this method of sample selection is, arguably, preferable to recruiting women with eating disorders from treatment centers. Shaw and Garfinkel (1990) have noted that recruiting participants from treatment centers may bias results, as the most severe cases would likely be overrepresented. Furthermore, we were interested in the scores of noneating disordered women as well, to evaluate a continuum of scores. Fairburn and Beglin (1990) argued that it is important to investigate the full continuum of disordered eating behaviors that exist in the general population. The present study makes a contribution in this respect. Nonetheless, before these results could be generalized to women who meet diagnostic criteria for anorexia or bulimia nervosa, the present study would need to be replicated in a clinical sample.

Future research is also needed to clarify the issue of whether disordered eating behaviors occur on a continuum or whether clinical eating disorders are categorically different from subclinical variants. Previous research has found mixed results regarding the continuum hypothesis (e.g., Gleaves et al., 2000; Stice et al., 1998). Research using the model validated in the present study could address the continuum issue by testing the fit independently in groups that have been proposed along the continuum (i.e., nonclinical, nonrestrained dieters, restrained dieters, bulimics) and by subsequently testing an invariant, multigroup model.

The generalizability of this study is also limited by the fact that participants were predominantly Caucasian. Thus, it is unclear whether similar results would be obtained in ethnically diverse samples. It is important to examine the influence of sociocultural factors on the continuum of disordered eating in future research.

In addition, the cross-sectional design of this study is a significant limitation, as it precludes definitive conclusions about the causality of disordered eating behaviors. In particular, because all variables used in this study were assessed at a single time point,

their potential roles as concomitants, consequences, or predictors of disordered eating cannot be determined (Kraemer et al., 1997). However, we hope that the results yielded by this study provide important hypotheses for future longitudinal research, which is sorely needed to clarify causal risk factors that could be targeted in prevention programs (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997; Kraemer et al., 1997; Striegel-Moore & Cachelin, in press). Finally, although the model developed and cross-validated in this study provided a good fit for the data, these results do not imply that this model has been "proven" (Kline, 1998). Rather, it has not been rejected in the present study. Another model could provide an equally valid explanation of the associations among these variables. In particular, the variables included in the final model presented here could be influenced by unmeasured variables. As Quintana and Maxwell (1999) have noted, specification error is perhaps the most difficult type of error to detect when using SEM procedures. On the basis of the present results and previous research, it appears that important additional variables to consider in future studies to avoid specification error include anxiety, dissociation, and social support. Nonetheless, the present study attempted to provide a relatively parsimonious integration of variables that have been identified by previous researchers as particularly salient to the severity of disordered eating behaviors.

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