Cogn Ther Res (2008) 32:143–160
DOI 10.1007/s10608-006-9030-0

ORIGINAL ARTICLE

Weight-related Teasing, Dysfunctional Cognitions, and Symptoms of Depression and Eating Disturbances

Jessica S. Benas · Brandon E. Gibb

Published online: 22 June 2006

Abstract Although there is evidence that verbal victimization in childhood may be a nonspecific risk factor for a variety of disorders, there may be mediational pathways that are specific to each disorder. In this study, we hypothesized that disorder-specific cognitions would mediate the relation between reports of childhood verbal victimization and undergraduates' current symptoms of depression and eating disorders. In addition, we hypothesized that a specific type of verbal victimization, weight-related teasing, would be more strongly related to the presence of dysfunctional eating cognitions than depressive cognitions. Both hypotheses were supported, providing preliminary evidence for the hypothesized disorder-specific mediational pathways.

Keywords Verbal victimization · Teasing · Depression · Eating disorders

Introduction

Eating disorders are characterized by behaviors such as extreme restrictive or uninhibited eating and preoccupation with eating, weight, and shape (American Psychiatric Association, 1994; National Institute of Mental Health [NIMH], 2001). Although more common among women than men, eating disorders are debilitating and chronic diseases that present an increased risk for mortality regardless of gender (NIMH, 2001; National Eating Disorders Association, 2002). Research focused on eating disorders has shown that abnormal eating behaviors tend to peak in mid to late adolescence (Steinhausen, Gavez, & Metzke, 2005), with the mean age of onset for an eating disorder between the

An earlier version of this paper was presented at the annual meeting of the Association for Behavioral and Cognitive Therapies, Washington, DC, 2005. This project was supported in part by National Institute of Child Health and Human Development grant HD048664 awarded to the second author.

J. S. Benas (✉) · B. E. Gibb
Department of Psychology, P.O. Box 6000, Binghamton, NY 13902, USA
e-mail: jessica.benas@binghamton.edu

ages of 15 and 17 (Striegel-Moore et al., 2004). In addition, being diagnosed with an eating disorder earlier in life presents an increased risk for experiencing such symptoms as an adult (Kotler, Cohen, Davies, Pine, & Walsh, 2001).

To date, cognitive-behavioral theories of eating disorders (e.g., Cooper, 2005a; Fairburn, Cooper, & Shafran, 2003; Fairburn, Shafran, & Cooper, 1998; Shafran, Fairburn, Nelson, & Robinson, 2003) have generally focused on factors that may maintain the disorder, and have emphasized the role of body dissatisfaction and body image distortion. According to the theories, individuals suffering from an eating disorder tend to have negative beliefs about themselves, and the activation of these negative beliefs increases distress and arousal. Engaging in maladaptive eating behaviors tends to quell these negative cognitions and the subsequent distress that accompanies them (Cooper 2005b). Supporting these theories, studies have found that anorexics and bulimics tend to view their body as larger than it actually is, be obsessed with their body and bodily functions, and perceive minor bodily flaws as large imperfections (Brouwers, 1988; Fairburn et al., 1998). In addition, studies have found that individuals with eating disorders have more dysfunctional eating-related cognitions than do individuals without these disorders (Butow, Beumont, & Touyz, 1993), and these cognitions contribute to the maintenance of disordered eating patterns (Shafran et al., 2003).

Despite the utility of these theories for understanding the *maintenance* of eating disorders, they have not adequately addressed factors that contribute to the *development* of eating disorders. Specifically, these theories do not adequately address how dysfunctional cognitions related to eating, weight, and shape might develop, or their role as a potential vulnerability to eating disorders. It has been suggested that research on cognitive vulnerabilities to eating disorders, along with the integration of other potential risk factors, is lacking (Cooper, 2005b). There is, however, some evidence that negative events in childhood, such as weight-related teasing, may contribute to the development of dysfunctional eating-related cognitions as well as disordered eating itself.

Weight-related teasing is prevalent among adolescents and occurs across ethnic groups (Neumark-Sztainer, Falkner, Story, Hannan, & Mulert, 2002). It has become an increasingly prevalent problem, as weight stigmatization among children has substantially increased over the last 40 years (Latner & Stunkard, 2003). This type of teasing among peers is related to later body image disturbances and a greater likelihood of eating disorder symptoms (Grilo, Wilfley, Brownell, & Rodin, 1994; Gleason, Alexander, & Somers, 2000; Lieberman, Gauvin, Bukowski, & White, 2001; Thompson, Coovert, Richards, Johnson, & Cattarin, 1995). Weight-related teasing is significantly related to higher weight concerns and disordered eating behaviors in both overweight and non-overweight individuals, though overweight individuals are more likely to be teased (Hayden-Wade et al., 2005; Neumark-Sztainer et al., 2002). In addition, the frequency of teasing in childhood significantly predicts poorer body image later in life among both males and females (Gleason et al., 2000). Given this, it is possible that weight-related teasing contributes to the development of dysfunctional eating-related cognitions, which then leaves the individual vulnerable to developing disordered eating behaviors.

There is evidence, however, that teasing and related constructs such as verbal victimization and emotional abuse may contribute risk to a variety of disorders. For example, studies have assessed the relation between childhood teasing and later depressive symptoms and found that reported levels of teasing are significantly and positively related to higher levels of depression in children (Craig, 1998), adolescents (Eisenberg, Neumark-Sztainer, & Story, 2003), and young adults (Lundgren, Anderson, Thompson, Shapiro, & Paulosky, 2004; Roth, Coles, & Heimberg, 2002;

Storch et al., 2004). Rose and Abramson (1992) proposed a developmental extension of the hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989) by suggesting that childhood emotional abuse may contribute to the development of a cognitive vulnerability to depression. Specifically, they hypothesized that when a negative event such as emotional abuse occurs, the child initially makes hopefulness-inducing attributions (e.g., ''it won't happen again'') and will view the event as preventable in the future, thus eliciting an expectation of change. However, when the event recurs repeatedly, the child's hopefulness-inducing attributions are disconfirmed and the child may begin to make hopelessness-inducing attributions (e.g., ''I deserve what happens to me''). Over time these attributions may become less situation specific and may be applied to other negative events in the child's environment and ultimately culminate into a general cognitive vulnerability to depression. Rose and Abramson hypothesized that emotional abuse, which includes teasing and humiliation, is more likely to promote the development of a cognitive vulnerability to depression than are other forms of abuse, because with emotional abuse the depressogenic inferences are directly supplied to the child by the abuser (e.g., ''You are stupid'').

Numerous studies have supported the hopelessness theory's (Abramson, Metalsky, & Alloy 1989) vulnerability hypothesis. Specifically, there is increasing evidence that depressive cognitions contribute to the development of both symptoms and diagnoses of depression (for reviews, see Abramson et al., 2002; Alloy, Abramson, Safford, & Gibb, 2005; Gibb & Coles, 2005). A number of studies have also supported Rose and Abramson's (1992) developmental extension of the hopelessness theory. Specifically, studies have suggested that reports of childhood emotional abuse by parents as well as verbal victimization by peers are more strongly related to the presence of depressive cognitions (Hankin, in press; Gibb, Abramson, & Alloy, 2004; Gibb, Alloy, Abramson, & Marx, 2003; Gibb et al., 2001) as well as symptoms and diagnoses of depression (Gibb et al., 2001; Gibb, Butler, & Beck, 2003; Gibb, Chelminski, & Zimmerman, 2005; Stone, 1993) than are other forms of negative life events. In addition, there is evidence that childhood emotional abuse/verbal victimization actually contributes to the development of negative cognitive styles and that these depressive cognitions mediate the relation between childhood emotional abuse/verbal victimization and symptoms and diagnoses of depression in children and adults (Gibb & Alloy, 2006; Gibb et al., 2001, 2003; Hankin, in press).

Therefore, verbal victimization in childhood may be a nonspecific risk factor for a variety of disorders. Despite this, however, there may be developmental or mediational pathways that are specific to the various disorders. Specifically, it may be that the development of disorder-specific cognitions mediates the relation between verbal victimization and symptoms of depression and eating disorders. The primary goal of this study, therefore, was to test this integrated mediational model. At the outset it should be noted that this was a cross-sectional study and as such no causal conclusions can be drawn. Despite this, however, cross-sectional studies can provide an important initial investigation of developmental hypotheses to determine whether larger-scale longitudinal studies are warranted. In the current study, we hypothesized that current depressive cognitions would mediate the link between reports of childhood verbal victimization and current depressive symptoms and that current dysfunctional cognitions concerning eating, weight, and shape would mediate the link between childhood verbal victimization and current symptoms of eating disorders (see Fig. 1).

A secondary goal of this study was to examine factors that may make the development of one disorder versus the other more likely. Specifically, it may be important to

shift attention from the detrimental effects of verbal victimization as a whole to specific forms of the victimization. Thus, for example, the specific content of the teasing a child experiences may make the development of one disorder more likely than another. As noted above, one form of verbal victimization that may contribute specific risk for the development of disordered eating is weight-related teasing. Given this, we hypothesized that the mediational model described above would also be supported when we focused specifically on weight-related teasing, but that weight-related teasing would be more strongly related to the current dysfunctional eating cognitions than to depressive cognitions (see Fig. 2). Finally, given evidence of gender differences in both eating disorders and depression (NIMH, 2001; Nolen-Hoeksema, 2001), participants' gender was considered as a potential moderator of the relations examined in the mediation models.

Method

Participants

Participants in this study were 203 undergraduates recruited from their introductory psychology classes who participated in exchange for receiving course credit. The

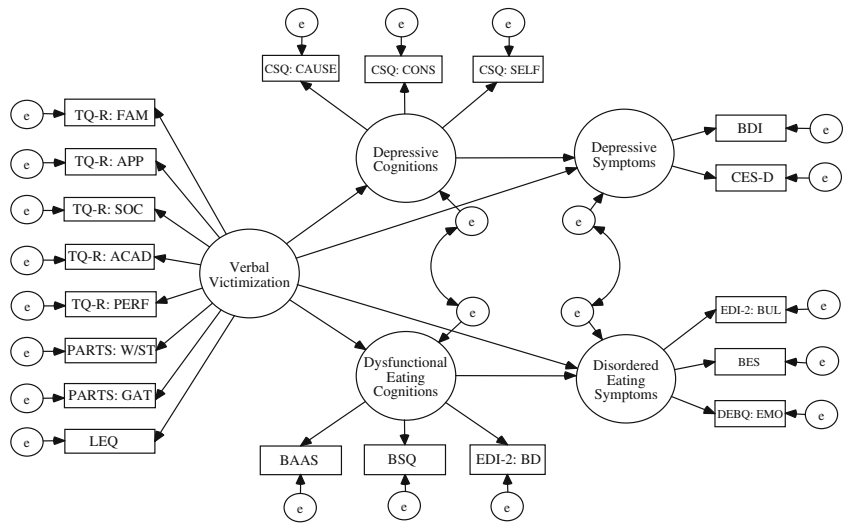

Fig. 1 Hypothesized mediation model. TQ-R: FAM = Teasing Questionnaire-Revised: Family Subscale; TQ-R: APP = Teasing Questionnaire-Revised: Appearance Subscale; TQ-R: SOC = Teasing Questionnaire-Revised: Social Subscale; TQ-R: ACAD = Teasing Questionnaire-Revised: Academic Subscale; TQ-R: PERF = Teasing Questionnaire-Revised: Performance Subscale; PARTS: W/ST = Physical Appearance Related Teasing Scale: Weight/Size Teasing Subscale; PARTS: GAT = Physical Appearance Related Teasing Scale: General Appearance Teasing Subscale; LEQ = Life Experiences Questionnaire: Emotional Abuse by Peers or Romantic Partner Subscale; CSQ: CAUSE = Cognitive Styles Questionnaire: Causes Subscale; CSQ: CON = Cognitive Styles Questionnaire: Consequences Subscale; CSQ: SELF = Cognitive Styles Questionnaire: Self Characteristics Subscale; BAAS = Beliefs About Appearance Scale; BSQ = Body Shape Questionnaire; EDI-2: BD = Eating Disorders Inventory-2: Body Dissatisfaction Subscale; BDI = Beck Depression Inventory-II; CES-D = Center for Epidemiologic Studies-Depression Scale; EDI-2: BUL = Eating Disorders Inventory-2: Bulimia Subscale; BES = Binge Eating Scale; DEBQ: EMO = Dutch Eating Behaviors Questionnaire: Emotional Eating Subscale

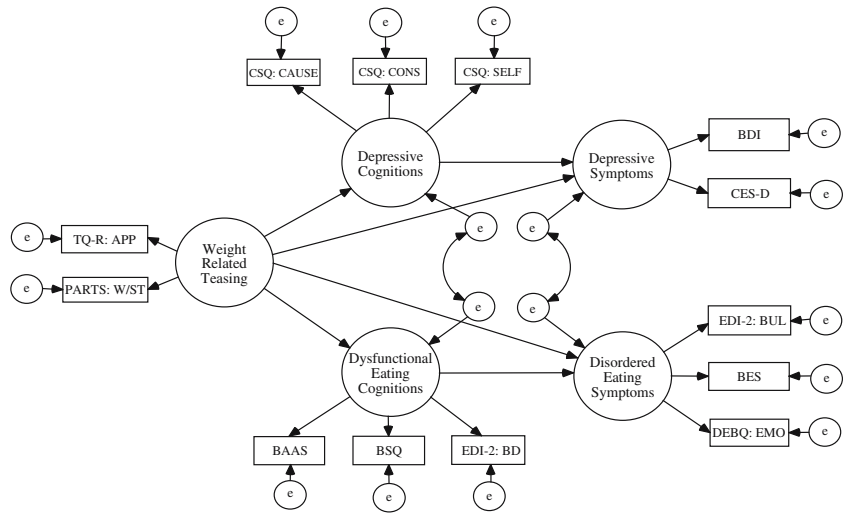

Fig. 2 Hypothesized mediation model testing specificity of weight-related teasing. TQ-R: APP = Teasing Questionnaire-Revised: Appearance Subscale; PARTS: W/ST = Physical Appearance Related Teasing Scale: Weight/Size Teasing Subscale; CSQ: CAUSE = Cognitive Styles Questionnaire: Causes Subscale; CSQ: CON = Cognitive Styles Questionnaire: Consequences Subscale; CSQ: SELF = Cognitive Styles Questionnaire: Self Characteristics Subscale; BAAS = Beliefs About Appearance Scale; BSQ = Body Shape Questionnaire; EDI-2: BD = Eating Disorders Inventory-2: Body Dissatisfaction Subscale; BDI = Beck Depression Inventory-II; CES-D = Center for Epidemiologic Studies-Depression Scale; EDI-2: BUL = Eating Disorders Inventory-2: Bulimia Subscale; BES = Binge Eating Scale; DEBQ: EMO = Dutch Eating Behaviors Questionnaire: Emotional Eating Subscale

majority of participants were female (64%) and the mean age was 19.07 years (*SD* = 1.32). The racial/ethnic background of the participants was as follows: 53.7% Caucasian, 3.9% African American, 27.6% Asian, 8.9% Hispanic, and 5.4% other.

Measures

Verbal victimization/teasing

The Physical Appearance Related Teasing Scale (PARTS; Thompson, Fabian, Moulton, Dunn, & Altabe, 1991) is an 18-item scale used to assess appearance related teasing and is composed of two factors: general appearance teasing (GAT) and weight/size teasing (W/ST). Subjects respond on a 5-point Likert-type scale, with responses ranging from ''never'' to ''frequently.'' Previous studies have suggested that both subscales exhibit good internal consistency and retest reliability (e.g., Thompson et al., 1991). In this study, both the W/ST and the GAT subscales exhibited good internal consistency (αs = .92 and .82, respectively).

In addition, the Teasing Questionnaire-Revised (TQ-R; Storch et al., 2004) was used, which is a 35-item scale measuring memories of being teased during childhood. Responses are made on a 5-point Likert-type scale, from ''I was never teased about this'' to ''I was always teased about this.'' The TQ-R includes five subscales assessing teasing in different domains: performance, academic abilities, social behaviors, family, and appearance. The TQ-R subscales exhibit adequate internal consistencies and good concurrent validity with measures of depressive symptoms, anxiety, fear of negative

evaluation, and loneliness (Storch et al., 2004). In this study, the five TQ-R subscales exhibited adequate internal consistencies (αs = .59, .89, .76, .56, and .84, respectively).

Lastly, the Life Experiences Questionnaire (LEQ; Gibb et al., 2001) was included, which is a 92 item self-report measure that assesses history of emotional, physical, and sexual maltreatment as well as emotional and physical neglect from a variety of sources. For the purpose of this study, only the emotional abuse subscale was included, which assesses humiliation, rejection, extortion, and teasing. To be consistent with the other measures of verbal victimization included in this study, we focused specifically on victimization from peers and romantic partners (LEQ Peer). Scores were calculated by summing the number of verbal victimization experiences endorsed as having occurred before age 15. The LEQ emotional abuse subscale has demonstrated good internal consistency and is related to depressive cognitions, symptoms, and diagnoses (Gibb et al., 2001, 2003). In this study, the subscale assessing victimization from peers and romantic partners demonstrated good internal consistency (α = .83).

Depressive cognitions

The Cognitive Style Questionnaire (CSQ; Alloy et al., 2000) was used to assess cognitive vulnerability to depression as defined by the hopelessness theory (Abramson et al., 1989). The CSQ consists of 24 hypothetical positive and negative events used to assess an individual's tendency to make stable, global attributions regarding the causes of negative events and to infer negative consequences and self-characteristics following the occurrence of such negative events. The CSQ has shown good internal consistency, retest reliability, and predictive validity for episodes of depression (Alloy et al., 2000; Alloy, Abramson, Whitehouse, Hogan, Panzarella, & Rose, 2006; Hankin, Abramson, Miller, & Haeffel, 2004). In this study, the Depressive Cognitions latent variable was identified by the Generality Composite (average of stability and globality ratings) as well as the composite scores for Consequences and Self-Characteristics. Each of these three subscales exhibited excellent internal consistency (αs = .91, .89, and .90 for generality, consequences, and self-characteristics, respectively).

Dysfunctional eating cognitions

The Beliefs About Appearance Scale (BAAS; Spangler & Stice, 2001) measures dysfunctional attitudes about one's bodily appearance, specifically the perceived consequences of appearance for relationships, achievement, feelings, and self-view. The specific dysfunctional attitudes measured are hypothesized to be common in eating disorders and contribute to restrictive eating patterns, a critical view of the body, and an over-evaluation of body related stimuli. Each of the 20 items is rated on 5-point Likert-type scale with responses ranging from ''not at all'' to ''extremely.'' The BAAS has demonstrated high internal consistency and retest reliability in previous research (Spangler & Stice, 2001). The BAAS is also related to other measures of body dissatisfaction, dieting, and eating disordered symptoms (Spangler & Stice, 2001). In the current study, the BAAS exhibited excellent internal consistency (α = .96).

The Body Shape Questionnaire (BSQ; Cooper, Taylor, Cooper, & Fairburn, 1987) is a 34-item self-report questionnaire used to assess concerns about weight and body shape, specifically how the subject has been feeling about his/her appearance over the past 4 weeks. Responses are on a 6-point Likert scale ranging from ''never'' to ''always.'' The BSQ has high retest reliability and strong concurrent validity with

measures of self-evaluation, body checking, and avoidance behavior (Rosen, Jones, Ramirez, & Waxman, 1995). The BSQ demonstrated excellent internal consistency in the current study (α = .98).

Depressive symptoms

The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) is a 21-item self-report measure that was used to assess the severity of depression in the subjects. The BDI-II exhibits good reliability and validity (Beck et al., 1996) and had good internal consistency in this study (α = .90).

The Center for Epidemiologic Studies-Depression Scale (CES-D; Radloff, 1977), a 20-item self-report inventory, was used to assess participants' levels of depressive symptoms. Total scores on the CES-D range from 0 to 60, with higher scores indicating more severe levels of depressive symptoms. Numerous studies have supported the reliability and validity of the CES-D (e.g., Radloff, 1977; Santor, Zuroof, Ramsay, Cervantes, & Palacios, 1995). In this study, the CES-D exhibited good reliability (α = .89).

Disordered eating symptoms

The Eating Disorders Inventory-2 (EDI-2; Garner, 1991) is a self-report measure, which was used to assess level of symptoms associated with anorexia nervosa and bulimia nervosa. It consists of 91-items comprising 11 subscales: Drive for Thinness, Bulimia, Body Dissatisfaction, Ineffectiveness, Perfection, Maturity Fears, Interpersonal Distrust, Interoceptive Awareness, Asceticism, Impulse Regulation, and Social Insecurity. The items are presented in a six-choice format requiring subjects to indicate whether the item applies to them ''always,'' ''usually,'' ''often,'' ''sometimes,'' ''rarely,'' or ''never.'' The EDI-2 has demonstrated good reliability and validity (Garner, 1991). In this study, we focused on the Drive for Thinness (α = .91), Bulimia (α = .80), and Body Dissatisfaction (α = .90) subscales of the EDI-2.

The Binge Eating Scale (BES; Gormally, Black, Daston, & Rardin, 1982) was also used, which consists of 16 items assessing behavioral manifestations (e.g., eating in secret) and cognitions and emotions surrounding a binge episode (e.g., guilt after a binge). Each item contains up to four statements that are assigned different weights (0–3; ''0'' indicates no binge eating problem; ''3'' indicates a severe binge eating problem). The scale successfully discriminates between individuals with no, moderate, and severe binge eating problems (Gormally et al., 1982). The BES exhibited excellent internal consistency in this study (α = .91).

Lastly, the Dutch Eating Behaviors Questionnaire (DEBQ; van Strien, Frijters, Bergers, & Defares, 1986), was used to assess restrained eating (e.g., ''do you try to eat less at mealtimes than you would like to eat?''), emotional eating (e.g., ''do you have a desire to eat when you are lonely?''), and external eating (e.g., ''if you see others eating, do you also have the desire to eat?''). The questionnaire consists of 33 items on a 5-point Likert-type scale with responses ranging from ''seldom'' to ''very often.'' The DEBQ has high internal consistency and high factorial validity as demonstrated in previous research (van Strien et al., 1986) and the current study (Restrained eating α = .96; Emotional eating α = .96; External eating α = .88).

Procedure

Participants completed the questionnaires in large groups. The questionnaire packet was administered in counterbalanced order to control for any order effects, and preliminary analyses revealed that there were no significant order effects for the variables included in the study. Participants received course credit for their participation.

Results

Preliminary analyses

Preliminary analyses revealed significant skew among many of the variables. These variables were transformed (e.g., square root, log 10, inverse) to satisfy assumptions of normality prior to further analysis. Next, given the presence of some missing data, the pattern of missingness was examined to determine whether the use of data imputation methods for estimating missing values was justified (cf. Shafer & Graham, 2002). Little's Missing Completely at Random test (MCAR; Little & Rubin, 1987), for which the null hypothesis is that the data are missing at random, was nonsignificant, χ^2 (34) = 27.42, P = .78, supporting the estimation of missing values. Therefore, maximum likelihood estimates of missing data were created and used in all subsequent analyses (see Shafer & Graham, 2002). Correlations among the study variables, as well as their means and standard deviations, are presented in Table 1. To facilitate comparisons with other studies, the means and standard deviations presented are from the untransformed variables.

Confirmatory factor analyses of measurement models

Analyses were then conducted to examine the various measurement models to be included in the tests of mediation. The variables used as indicators for the Verbal Victimization latent variable were the five TQ-R subscales, the two PARTS subscales, and the LEQ. This measurement model provided an adequate fit to the data, χ^2 (20, N = 203) = 62.03, P < .001, CFI = .93, RMSEA = .10, SRMR = .05 (cf. Hu & Bentler, 1999).[1]

Next, analyses were conducted to confirm that the Depressive Cognitions (indicated by the three CSQ subscales) and Depressive Symptoms (indicated by the BDI-II and the CES-D) constructs were indeed distinct. Two models were compared: one that defined the two as a single construct, and one that defined them as separate constructs. The separate constructs model provided a good fit to the data χ^2 (4, N = 203) = 7.17, P = .13, CFI = 1.0, RMSEA = .06, SRMR = .03, and fit significantly better than the single constructs model, χ^2 (1, N = 203) = 170.41, P < .001.

Next, a confirmatory factor analysis was performed to determine if all of the eating disordered symptom measures loaded on a single latent variable. The Disordered Eating Symptoms construct included the Drive For Thinness and Bulimia subscales of the EDI-2, the Binge Eating Scale, and the three subscales of the DEBQ: Restrained,

[1] Although the chi-square was significant, this is not uncommon for well-fitting models with large sample sizes (Kline, 2005).

Table 1 Correlations and descriptive statistics for study variables

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
1.	PARTS: GAT	–																					
2.	PARTS: W/ST	.28**	–																				
3.	TQ-R: PERF	.48**	.38**	–																			
4.	TQ-R: ACAD	.42**	.33**	.47**	–																		
5.	TQ-R: SOC	.53**	.25**	.60**	.47**	–																	
6.	TQ-R: FAM	.30**	.15*	.34**	.31**	.45**	–																
7.	TQ-R: APP	.61**	.57**	.54**	.58**	.59**	.40**	–															
8.	LEQ PEER	.38**	.32**	.29**	.28**	.29**	.24**	.43**	–														
9.	CSQ CAUSE	.29**	.25**	.29**	.25**	.22**	.16*	.36**	.25**	–													
10.	CSQ CONS	.26**	.18*	.27**	.23**	.22**	.20**	.24**	.22**	.80**	–												
11.	CSQ SELF	.31**	.32**	.42**	.29**	.31**	.19**	.34**	.32**	.67**	.74**	–											
12.	BAAS	.18*	.41**	.23**	.24**	.24**	.14*	.33**	.21**	.29**	.37**	.45**	–										
13.	BSQ	.05	.49**	.23**	.27**	.14*	.09	.33**	.18*	.19**	.19**	.35**	.64**	–									
14.	BDI-II	.24**	.27**	.26**	.25**	.29**	.07	.30**	.27**	.28**	.29**	.34**	.30**	.42**	–								
15.	CES-D	.24**	.27**	.31**	.28**	.22**	.16*	.36**	.29**	.36**	.33**	.37**	.33**	.38**	.79**	–							
16.	EDI-2: THIN	.05	.43**	.11	.23**	.07	.04	.31**	.12	.21**	.18**	.24**	.64**	.83**	.35**	.28**	–						
17.	EDI-2: BUL	.03	.17*	.06	.08	.07	.01	.17*	.12	.20**	.14*	.16*	.32**	.41**	.22**	.21**	.46**	–					
18.	EDI-2: BD	.00	.41**	.14	.15*	.06	–.04	.20**	.08	.21**	.18*	.27**	.53**	.79**	.40**	.40**	.70**	.32**	–				
19.	BES	.48**	.39**	.20**	.17*	.14*	.02	.29**	.20**	.28**	.27**	.34**	.57**	.73**	.46**	.45**	.67**	.55**	.59**	–			
20.	DEBQ:REST	.00	.45**	.13	.21**	.04	.00	.26**	.08	.07	.00	.18*	.54**	.85**	.26**	.25**	.84**	.36**	.67**	.66**	–		
21.	DEBQ: EMO	.08	.21**	.09	.12	.15*	.07	.13	.08	.18*	.16*	.20**	.41**	.46**	.19**	.24**	.42**	.54**	.38**	.63**	.48**	–	
22.	DEBQ: EXT	.08	.22**	.16*	.05	.22**	.20**	.12	.15*	.13	.13	.29**	.22**	.28**	.24**	.23**	.13	.44**	.22**	.53**	.16*	.60**	–
	Mean	9.25	16.81	1.82	5.05	3.51	1.75	7.20	2.57	3.77	3.34	3.36	25.96	78.34	10.05	16.24	4.43	1.48	8.30	9.13	24.13	28.61	30.38
	SD	3.40	7.47	2.05	5.19	3.84	2.16	6.79	3.27	1.01	1.15	1.29	16.31	37.29	8.15	9.80	5.69	2.98	7.32	8.19	10.80	13.15	7.59

Note: PARTS: W/ST = Physical Appearance Related Teasing Scale: Weight/Size Teasing Subscale; PARTS: GAT = Physical Appearance Related Teasing Scale: General Appearance Teasing Subscale; TQ-R: PERF = Teasing Questionnaire-Revised: Performance Subscale; TQ-R: ACAD = Teasing Questionnaire-Revised: Academic Subscale; TQ-R: SOC = Teasing Questionnaire-Revised: Social Subscale; TQ-R: FAM = Teasing Questionnaire-Revised: Family Subscale; TQ-R: APP = Teasing Questionnaire-Revised: Appearance Subscale; LEQ PEER = Life Experiences Questionnaire: Emotional Abuse by Peers or Romantic Partner Subscale; CSQ: CAUSE = Cognitive Styles Questionnaire: Causes Subscale; CSQ: CON = Cognitive Styles Questionnaire: Consequences Subscale; CSQ: SELF = Cognitive Styles Questionnaire: Self Characteristics Subscale; BAAS = Beliefs About Appearance Scale; BSQ = Body Shape Questionnaire; BDI-II = Beck Depression Inventory-II; CES-D = Center for Epidemiologic Studies-Depression Scale; EDI-2: THIN = Eating Disorders Inventory-2: Drive For Thinness Subscale; EDI-2: BUL = Eating Disorders Inventory-2: Bulimia Subscale; EDI-2: BD = Eating Disorders Inventory-2: Body Dissatisfaction Subscale; BES = Binge Eating Scale; DEBQ: REST = Dutch Eating Behaviors Questionnaire: Restrained Eating Subscale; DEBQ: EMO = Dutch Eating Behaviors Questionnaire: Emotional Eating Subscale; DEBQ: EXT = Dutch Eating Behaviors Questionnaire: External Eating Subscale

*$P < .05$

**$P < .01$

Emotional, and External Eating. This model provided a poor fit to the data, χ^2 (9, $N = 203$) = 223.02, $P < .001$, CFI = .70, RMSEA = .34, SRMR = .14. It was hypothesized that the poor data fit was a result of the heterogeneous nature of the symptoms included, specifically overeating and undereating. Therefore, the two measures assessing undereating (EDI-2: Drive For Thinness and DEBQ: Restrained Eating) were omitted from the model.[2] This revised overeating model provided a good fit to the data, χ^2 (2, $N = 203$) = 2.42, $P = .30$, CFI = 1.00, RMSEA = .03, SRMR = .02. Finally, a separate vs. single constructs model was analyzed to assess if the Disordered Eating Symptoms and Dysfunctional Eating Cognitions (indicated by BAAS, BSQ, and EDI-2: Body Dissatisfaction) constructs were distinct in the sample. Although the separate constructs model did provide a better fit than the single construct model, χ^2 (1, $N = 203$) = 96.86, $P < .001$, the separate constructs model itself provided a relatively poor fit to the data, χ^2 (13, $N = 203$) = 63.96, $P < .001$, CFI = .93, RMSEA = .14, SRMR = .07. An examination of modification indices revealed that the External Eating subscale of the DEBQ, specified as an indicator for the Disordered Eating Symptoms construct, was also significantly related to the Dysfunctional Eating Cognitions construct. Omitting this variable from the model, the separate constructs model provided an adequate fit to the data, χ^2 (8, $N = 203$) = 20.91, $P = .007$, CFI = .98, RMSEA = .09, SRMR = .04, and continued to fit better than the single construct model, χ^2 (1, $N = 203$) = 66.24, $P < .001$.

Mediation models

The mediation models were tested using structural equation modeling in AMOS 5 (Arbuckle, 2003). Following the suggestions of Shrout and Bolger (2002), nonparametric bootstrapping was used to provide parameter estimates for the mediation models. Consistent with the recommendations of Efron and Tibshirani (1986), 1,000 bootstrap samples were used in this study.

In testing our primary hypothesis, we hypothesized that the association between Verbal Victimization and Depressive Symptoms would be mediated by Depressive Cognitions and the association between Verbal Victimization and Eating Disordered Symptoms would be mediated by Dysfunctional Eating Cognitions (see Fig. 1). Using nested model comparisons, a full mediation version of this model was compared to a partial mediation model, with the latter including direct pathways from Verbal Victimization to symptoms of Depression and Eating disorders.

First, the full mediation model was tested. This model provided poor fit to the data, χ^2 (146, $N = 203$) = 352.65, $P < .001$, CFI = .90, RMSEA = .08, SRMR = .10. Next, the partial mediation model was tested. The partial mediation model provided an adequate fit to the data, χ^2 (144, $N = 203$) = 335.37, $P < .001$, CFI = .91, RMSEA = .08, SRMR = .09 and fit significantly better than the full mediation model, χ^2 (2, $N = 203$) = 17.29, $P < .001$. Examination of the partial mediation results revealed that the path linking Verbal Victimization to Eating Disordered Symptoms was nonsignificant ($\beta = .05$, $P = .44$). Therefore, a final model was tested, with this path removed. This model provided an adequate fit to the data, χ^2 (145, $N = 203$) = 336.16, $P < .001$, CFI = .91, RMSEA = .08, SRMR = .09, and removing this path did not significantly

[2] The hypothesized mediation models were also tested with the undereating variables (EDI-2: Drive For Thinness and DEBQ: Restrained Eating) comprising the Disordered Eating Symptoms construct. The results of these analyses were virtually identical to the results presented in the paper.

reduce model fit compared to the original partial mediation model, χ^2 (1, $N = 203$) = .79, $P = .37$. Each of the paths included in this partial mediation model (Fig. 3) were significant. In addition, the indirect path linking Verbal Victimization to Depressive Symptoms was significant ($\beta = .12$, $P = .001$) as was the path from Verbal Victimization to Eating Disordered Symptoms ($\beta = .28$, $P = .002$). In this model, there was no significant difference in the magnitudes of the paths linking Verbal Victimization to Depressive Cognitions versus Dysfunctional Eating Cognitions ($z = .54$, $P = .29$).

The specificity of this model was then tested by including direct pathways from Depressive Cognitions to Eating Disordered Symptoms, as well as direct pathways from Dysfunctional Eating Cognitions to Depressive Symptoms. Adding these paths significantly improved model fit compared to the partial mediation model, χ^2 (2, $N = 203$) = 27.32, $P < .01$, and provided a good fit to the data, χ^2 (143, $N = 203$) = 308.84, $P < .01$, CFI = .92, RMSEA = .08, SRMR = .08. In this model, Depressive Cognitions were related to Depressive Symptoms ($\beta = .22$, $P < .01$) as well as Eating Disordered Symptoms ($\beta = .13$, $P = .02$). The difference in the magnitude of these relations was significant ($z = 1.97$, $P = .02$), indicating that Depressive Cognitions were more strongly related to Depressive Symptoms than to Eating Disordered Symptoms. Dysfunctional Eating Cognitions were also related to Depressive Symptoms

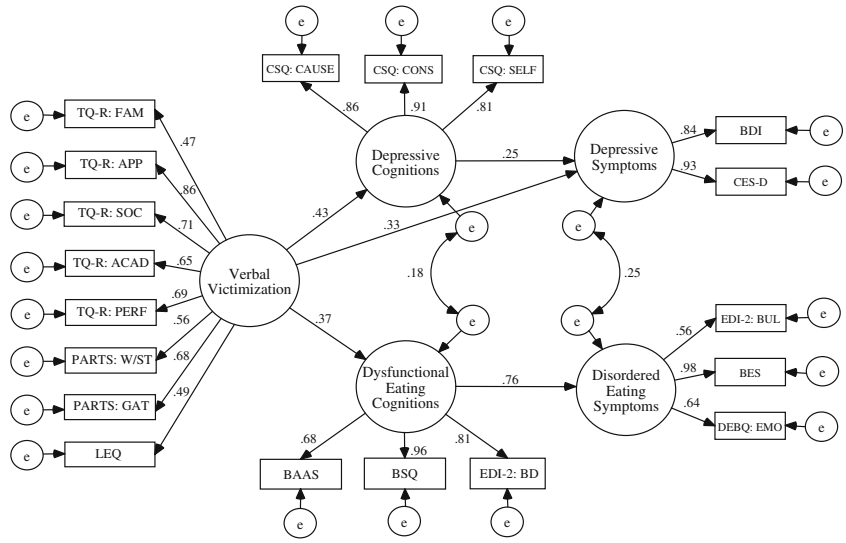

Fig. 3 Supported mediation model with estimates. TQ-R: FAM = Teasing Questionnaire-Revised: Family Subscale; TQ-R: APP = Teasing Questionnaire-Revised: Appearance Subscale; TQ-R: SOC = Teasing Questionnaire-Revised: Social Subscale; TQ-R: ACAD = Teasing Questionnaire-Revised: Academic Subscale; TQ-R: PERF = Teasing Questionnaire-Revised: Performance Subscale; PARTS: W/ST = Physical Appearance Related Teasing Scale: Weight/Size Teasing Subscale; PARTS: GAT = Physical Appearance Related Teasing Scale: General Appearance Teasing Subscale; LEQ = Life Experiences Questionnaire: Emotional Abuse by Peers or Romantic Partner Subscale; CSQ: CAUSE = Cognitive Styles Questionnaire: Causes Subscale; CSQ: CON = Cognitive Styles Questionnaire: Consequences Subscale; CSQ: SELF = Cognitive Styles Questionnaire: Self Characteristics Subscale; BAAS = Beliefs About Appearance Scale; BSQ = Body Shape Questionnaire; EDI-2: BD = Eating Disorders Inventory-2: Body Dissatisfaction Subscale; BDI = Beck Depression Inventory-II; CES-D = Center for Epidemiologic Studies-Depression Scale; EDI-2: BUL = Eating Disorders Inventory-2: Bulimia Subscale; BES = Binge Eating Scale; DEBQ: EMO = Dutch Eating Behaviors Questionnaire: Emotional Eating Subscale. $P < .01$ for all estimates

(β = .33, P < .01) as well as Eating Disordered Symptoms (β = .73, P < .01). However, the magnitude of these two relations did not differ significantly (z = 0.99, P = .16). Therefore, in this model, there was some evidence for the specificity of depressive cognitions to depressive symptoms, but not for eating cognitions to eating symptoms.

A secondary aim of this study was to test the integrated mediation model for weight-related teasing, specifically. As for the previous hypothesis, structural equation modeling was used to test the mediation model (see Fig. 2), and a full mediation model was compared to a partial mediation model, with direct pathways from Weight-Related Teasing to symptoms of Depression and Eating Disorders, using nested model comparisons. Paralleling the previous analyses, we hypothesized that the association between Weight-Related Teasing and Depressive Symptoms would be mediated by Depressive Cognitions and the association between Weight-Related Teasing and Eating Disordered Symptoms would be mediated by Dysfunctional Eating Cognitions. In addition, we hypothesized that Weight-Related Teasing would be more strongly related to Dysfunctional Eating Cognitions than Depressive Cognitions. Consistent with the procedure for testing the first hypothesis, a full mediation model was tested. This model provided a poor fit to the data, χ^2 (59, N = 203) = 165.49, P < .001, CFI = .93, RMSEA = .10, SRMR = .11. Next, a partial mediation model was tested, which provided an adequate fit to the data, χ^2 (57, N = 203) = 147.51, P < .001, CFI = .94, RMSEA = .09, SRMR = .08 and fit significantly better than the full mediation model, χ^2 (2, N = 203) = 17.98, P < .001.

Consistent with what we found before, examination of the partial mediation results revealed that the path linking Weight-Related Teasing to Disordered Eating Symptoms was nonsignificant (β = .08, P = .34). Therefore, this path was removed, and a model in which Depressive Cognitions partially mediated the link between Weight-Related Teasing and Depressive Symptoms and Dysfunctional Eating Cognitions fully mediated the link between Weight-Related Teasing and Disordered Eating Symptoms provided an adequate fit to the data, χ^2 (58, N = 203) = 148.38, P < .001, CFI = .94, RMSEA = .09, SRMR = .08. In addition, this model fit significantly better than the full mediation model, χ^2 (1, N = 203) = 17.11, P < .001 and did not fit significantly worse than a partial mediation model including a direct path from Weight-Related Teasing to Disordered Eating Symptoms, χ^2 (1, N = 203) = .87, P = .35. All of the paths in this model were also significant (Fig. 4). In addition, the indirect path linking Weight-Related Teasing to Depressive Symptoms was significant (β = .10, P = .01) as was the indirect path from Weight-Related Teasing to Disordered Eating Symptoms (β = .48, P = .002). As hypothesized, however, Weight-Related Teasing was significantly more strongly related to Dysfunctional Eating Cognitions than Depressive Cognitions (z = 3.28, P < .001).

The specificity of this model was also tested. Adding crossover effects from Depressive Cognitions to Disordered Eating Symptoms and from Disordered Eating Cognitions to Depressive Symptoms significantly improved model fit compared to the partial mediation model, χ^2 (2, N = 203) = 19.58, P < .01, and provided a good fit to the data, χ^2 (56, N = 203) = 128.80, P < .01, CFI = .95, RMSEA = .08, SRMR = .06. In this model, Depressive Cognitions were related to Depressive Symptoms (β = .28, P < .01) as well as Disordered Eating Symptoms (β = .13, P = .02). However, the difference in the magnitude of these relations was not significant (z = 1.59, P = .06). In contrast, Dysfunctional Eating Cognitions were more strongly related to Disordered Eating Symptoms (β = .73, P < .01) than to Depressive Symptoms (β = .34, P < .01), z = 4.7, P < .01, indicating some specificity of eating cognitions to eating symptoms in this model.

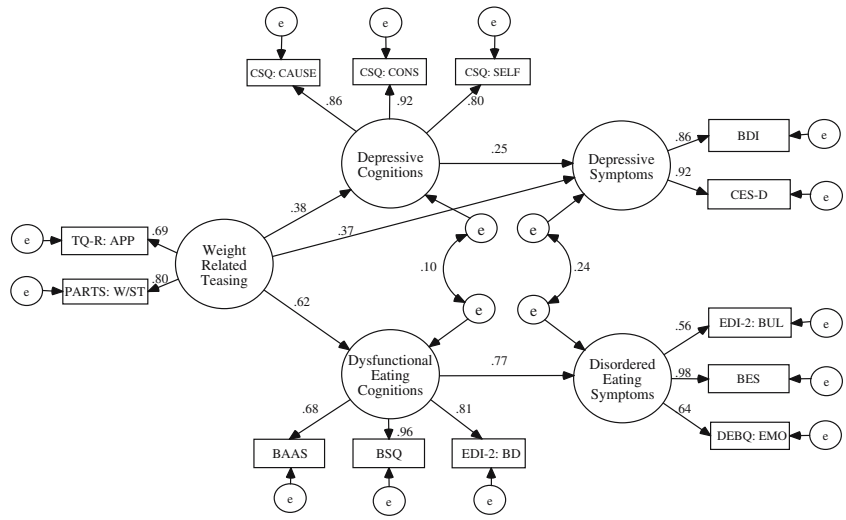

Fig. 4 Supported mediation model testing specificity of weight-related teasing, with estimates. TQ-R: APP = Teasing Questionnaire-Revised: Appearance Subscale; PARTS: W/ST = Physical Appearance Related Teasing Scale: Weight/Size Teasing Subscale; CSQ: CAUSE = Cognitive Styles Questionnaire: Causes Subscale; CSQ: CON = Cognitive Styles Questionnaire: Consequences Subscale; CSQ: SELF = Cognitive Styles Questionnaire: Self Characteristics Subscale; BAAS = Beliefs About Appearance Scale; BSQ = Body Shape Questionnaire; EDI-2: BD = Eating Disorders Inventory-2: Body Dissatisfaction Subscale; BDI = Beck Depression Inventory-II; CES-D = Center for Epidemiologic Studies-Depression Scale; EDI-2: BUL = Eating Disorders Inventory-2: Bulimia Subscale; BES = Binge Eating Scale; DEBQ: EMO = Dutch Eating Behaviors Questionnaire: Emotional Eating Subscale. $P < .05$ for all estimates

Finally, participants' gender was evaluated as a potential moderator of the relations in the models. Specifically, it was examined whether the magnitudes of the relations specified between latent variables in the mediation models differ significantly based upon participants' gender. Results revealed that gender did not moderate model fit for either the general Verbal Victimization model or the more specific Weight-Related Teasing model.

Discussion

The purpose of this study was to examine the relations between reports of verbal victimization in childhood and cognitions and symptoms of depression and eating disorders in adulthood. Supporting our primary hypothesis, depressive cognitions partially mediated the relation between verbal victimization and depressive symptoms, and dysfunctional eating cognitions fully mediated the relation between verbal victimization and eating disorder symptoms. Supporting our secondary hypothesis, this mediation model was also supported when we focused specifically on weight-related teasing, and weight-related teasing was more strongly related to dysfunctional eating cognitions than depressive cognitions. Providing further support for our specificity hypothesis, dysfunctional eating cognitions were more strongly related to disordered eating symptoms than depressive symptoms when focusing specifically on weight-related teasing, but not when examining verbal victimization more generally.

The current results provide additional support for Rose and Abramson's (1992) developmental extension of the hopelessness theory (Abramson et al., 1989) and extend this model to examine a potential vulnerability to eating disorders. These results are also consistent with previous findings that verbal victimization in childhood, and weight-related teasing specifically, is related to a higher incidence of eating disordered symptoms later in life (Jackson, Grilo, & Masheb, 2002; Lunner et al., 2000; Neumark-Sztainer et al., 2002; Thompson et al., 1995) and suggest a potential mechanism through which this may occur. Specifically, although based on cross-sectional data, the current results are consistent with the hypothesis that verbal victimization may contribute to the development of depression and eating disorders through the development of disorder-specific dysfunctional cognitions.

This study is the first of which we are aware to demonstrate that the specific content of verbal victimization experienced may make an individual more likely to exhibit one form of negative cognitions over another. Although conclusions must remain tentative pending replication, the results may have potentially important implications for refining models of developmental psychopathology. Specifically, despite evidence that a given factor may contribute risk for the development of a variety of disorders (i.e., multifinality), the current results suggest that specific characteristics of a given type of negative event (e.g., content of teasing) may contribute increased risk for one disorder over another. Future longitudinal research would benefit from a continued examination of the specific content of verbal victimization (e.g., weight-related teasing) and particular disorder-specific developmental pathways. In addition, although our mediation models were supported independently for both overeating and undereating symptoms, future studies should seek to determine whether certain aspects of weight-related teasing would be more strongly related to the development of restrictive versus excessive eating patterns. Finally, it would be beneficial to examine whether different targets of teasing (e.g., rejection from a social group) would contribute specific risk for other forms of dysfunctional cognitions (e.g., those related to social anxiety). If supported, the results could help shift the focus from multifinality to more specific developmental models of psychopathology.

This study exhibited a number of strengths including its large sample size as well as the inclusion of multiple assessments of each construct. There were several limitations as well. First, due to the cross-sectional nature of the study, no causal conclusions can be drawn. Prospective longitudinal studies are needed to determine whether weight-related teasing is more likely to contribute to the actual development of dysfunctional eating cognitions and symptoms than depressive cognitions and symptoms. Given evidence that dysfunctional cognitive styles appear to develop throughout childhood and stabilize by early adolescence (see Cooper, 2005b; Garber & Flynn, 1998; Haines, Metalksy, Cardamone, & Joiner, 1999) as well as evidence for the dramatic rise in the prevalence of both depression (Hankin et al., 1998) and eating disorders (Steinhausen et al., 2005; Striegel-Moore et al., 2004) in adolescence, the time period of late childhood and adolescence seems a particularly important area of focus.

A second limitation was that all of the measures were based upon participants' self-report, which may be subject to recall or response bias. That is, those who were currently depressed, or suffering from an eating disorder, may remember more instances of teasing than those who were not currently suffering from the disorder. This said, however, the current results are consistent with those of previous studies demonstrating that experiences of childhood emotional abuse/verbal victimization prospectively predict the development of depressive cognitions, which then contribute to the development of

depressive symptoms in children and young adults (e.g., Gibb & Alloy, 2006; Hankin, in press). The results are also consistent with previous studies suggesting that weight-related teasing predicts the development of both dysfunctional eating cognitions and symptoms (Gardner, Stark, Friedman, & Jackson, 2000; Gleason et al., 2000; Wertheim, Koerner, & Paxton, 2001). Despite this, future studies would benefit from the use of multi-method assessments of these constructs. Specifically, these studies could utilize both self-report and interview-based measures of symptoms, multiple informants regarding verbal victimization (e.g., self-reports and peer reports), and multi-method assessments of dysfunctional cognitions (e.g., self-report and computer-based tasks).

Third, the focus on university undergraduates may limit the generalizability of our findings. Specifically, it is unclear whether the current results will generalize to more impaired samples, or those with more severe developmental histories or diagnosed episodes of depression and eating disorders. Future studies, therefore, should seek to replicate these findings in more severely impaired samples (e.g., individuals receiving outpatient treatment).

In conclusion, although previous studies have suggested that teasing may contribute risk to future symptoms of eating disorders as well as depression (i.e., multifinality), the current results suggest that there may be mediational pathways (i.e., disorder-specific dysfunctional cognitions) specific to each disorder. In addition, the specific content of childhood victimization may make the development of one disorder more likely than another. Specifically, weight-related teasing was more strongly related to dysfunctional eating cognitions than depressive cognitions. These findings may have important implications for developmental psychopathology models, and future research should continue to explore factors that may make the development of one disorder versus another more likely.

References

Abramson, L. Y., Alloy, L. B., Hankin, B. L., Haeffel, G. J., MacCoon, D. G., & Gibb, B. E. (2002). Cognitive vulnerability-stress models of depression in a self-regulatory and psychobiological context. In I. H. Gotlib, C. L. Hammen (Eds.), *Handbook of depression* (pp. 268–294). New York: Guilford.

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review, 96*, 358–372.

Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., Kim, R. S., & Lapkin, J. B. (2000). The Temple-Wisconsin cognitive vulnerability to depression project: Lifetime history of Axis I psychopathology in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology*, *109*, 403–418.

Alloy, L. B., Abramson, L. Y., Safford, S. M., & Gibb, B. E. (2005). The Cognitive Vulnerability to Depression Project: Current findings and future directions. In L. B. Alloy, J. H. Riskind (Eds.), *Cognitive vulnerability to emotional disorders* (pp. 33–61). Hillsdale, NJ: Erlbaum.

Alloy, L. B., Abramson, L. Y., Whitehouse, W. G., Hogan, M. E., Panzarella, C., & Rose, D. T. (2006). Prospective incidence of first onsets and recurrences of depression in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology, 115*, 145–156.

American Psychiatric Association. (1994). *Diagnostic and statistical manual for mental disorders* (4th ed.). Washington, DC: Author.

Arbuckle, J. L. (2003). Amos 5 [Computer software]. Chicago: Smallwaters.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory Manual* (2nd ed.). San Antonio: The Psychological Corporation.

Brouwers, M. (1988). Depressive thought content among female college students with bulimia. *Journal of Counseling an (2003) Development*, *66*, 425–428.

Butow, P., Beumont, P., & Touyz, S. (1993). Cognitive processes in dieting disorders. *International Journal of Eating Disorders*, *14*, 319–329.

Cooper, M. J. (2005a). Cognitive theory in anorexia nervosa and bulimia nervosa: Progress, development and future directions. *Clinical Psychology Review*, *25*, 511–531.
Cooper, M. J. (2005b). A developmental vulnerability-stress model of eating disorders: A cognitive approach. In B. L. Hankin, J. R. Z. Abela, (Eds.), *Development of psychopathology: A vulnerability-stress perspective* (pp. 328–354). Thousand Oaks CA: Sage.
Cooper, P. J., Taylor, M. J., Cooper, Z., & Fairburn, C. G. (1987). The development and validation of the body shape questionnaire. *International Journal of Eating Disorders*, *6*, 485–494.
Craig, W. M. (1998). The relationship among bullying, victimization, depression, anxiety, and aggression in elementary school children. *Personality and Individual Differences*, *24*, 123–130.
Efron, B., & Tibshirani, R. (1986). Bootstrap methods for standard errors, confidence intervals, and other measures of statistical accuracy. *Statistical Science*, *1*, 54–77.
Eisenberg, M. E., Neumark-Sztainer, D., & Story, M. (2003). Associations of weight-based teasing and emotional well-being among adolescents. *Archives of Pediatrics & Adolescent Medicine*, *157*, 733–738.
Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A ''transdiagnostic'' theory and treatment. *Behaviour Therapy and Research*, *41*, 509–528.
Fairburn, C. G., Shafran, R., & Cooper, Z. (1998). A cognitive behavioural theory of anorexia nervosa. *Behaviour Therapy and Research*, *37*, 1–13.
Garber, J., & Flynn, C. (1998). Origins of the depressive cognitive style. In D. K. Routh, R. J. DeRubeis (Eds.), *The science of clinical* psychology*: Accomplishments and future directions* (pp. 53–93). Washington, DC: American Psychological Association.
Gardner, R. M., Stark, K., Friedman, B. N., & Jackson, N. A. (2000). Predictors of eating disorder scores in children ages 6 through 14: A longitudinal study. *Journal of Psychosomatic Research*, *49*, 199–205.
Garner, D. M. (1991). *Eating Disorder Inventory—2 professional manual.* Odessa, Florida: Psychological Assessment Resources.
Gibb, B. E., Abramson, L. Y., & Alloy, L. B. (2004). Emotional maltreatment from parents, verbal peer victimization, and cognitive vulnerability to depression. *Cognitive Therapy and Research*, *28*, 1–21.
Gibb, B. E., & Alloy, L. B. (2006). A prospective test of the hopelessness theory of depression in children. *Journal of Clinical Child and Adolescent Psychology, 35*, 264–274.
Gibb, B. E., Alloy, L. B., Abramson, L. Y., & Marx, B. P. (2003). Childhood maltreatment and maltreatment-specific inferences: A test of Rose and Abramson's (1992) extension of the hopelessness theory. *Cognition and Emotion*, *17*, 917–931.
Gibb, B. E., Alloy, L. B., Abramson, L. Y., Rose, D. T., Whitehouse, W. G., Donovan, P., Hogan, M. E. et al. (2001). History of childhood maltreatment, negative cognitive styles, and episodes of depression in adulthood. *Cognitive Therapy and Research*, *25*, 425–446.
Gibb, B. E., Butler, A. C., & Beck, J. S. (2003). Childhood abuse, depression, and anxiety in adult psychiatric outpatients. *Depression and Anxiety*, *17*, 226–228.
Gibb, B. E., Chelminski, I., & Zimmerman, M. (2005). Childhood emotional, physical, and sexual abuse and diagnoses of depressive and anxiety disorders in adult psychiatric outpatients. Manuscript submitted for publication.
Gibb, B. E., & Coles, M. E. (2005). Cognitive vulnerability-stress models of psychopathology: A developmental perspective. In B. L. Hankin, J. R. Z. Abela (Eds.), *Development of psychopathology: A vulnerability-stress perspective* (pp. 104–135). Thousand Oaks, CA: Sage.
Gleason, J. H., Alexander, A. M., & Somers, C. L. (2000). Later adolescents' reactions to three types of childhood teasing: Relations with self-esteem and body image. *Social Behaviors and Personality*, *28*, 471–480.
Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors*, *7*, 47–55.
Grilo, C. M., Wilfley, D. E., Brownell, K. D., & Rodin, J. (1994). Teasing, body image, and self-esteem in a clinical sample of obese women. *Addictive Behaviors*, *19*, 443–450.
Haines, B. A., Metalksy, G. I., Cardamone, A. L., & Joiner, T. (1999). Interpersonal and cognitive pathways into the origins of attributional style: A developmental perspective. In T. Joiner, J. C. Coyne (Eds.), *The interactional nature of depression*. (pp. 65–92). Washington, DC: American Psychological Association.
Hankin, B. L. (in press). Childhood maltreatment and psychopathology: Prospective tests of attachment, cognitive vulnerability, and stress as mediating processes. Cognitive Therapy and Research.
Hankin, B. L., Abramson, L. Y., Miller, N., & Haeffel, G. J. (2004). Cognitive vulnerability-stress theories of depression: Examining affective specificity in the prediction of depression versus anxiety in three prospective studies. *Cognitive Therapy and Research*, *28*, 309–345.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology*, *107*, 128–140.

Hayden-Wade, H. A., Stein, R. I., Saelens, B. E., Zabinski, M., Ghaderi, A., & Wilfley, D. E. (2005). Prevalence, characteristics, and correlates of teasing experiences among overweight children versus non-overweight peers. *Obesity Research*, *13*, 1381–1392.

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*, *6*, 1–55.

Jackson, T. D., Grilo, C. M., & Masheb, R. M. (2002). Teasing history and eating disorder features: An age- and body mass index- matched comparison of bulimia nervosa and binge-eating disorder. *Comprehensive Psychiatry*, *43*, 108–113.

Kline, R. B. (2005). *Principles and practice of structural equation modeling* (2nd ed.). New York: Guilford Press.

Kotler, L. A., Cohen, P., Davies, M., Pine, D. S., & Walsh, B. T. (2001). Longitudinal relationships between childhood, adolescent, and adult eating disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *40*, 1434–1440.

Latner, J. D., & Stunkard, A. J. (2003). Getting worse: The stigmatization of obese children. *Obesity Research*, *11*, 452–456.

Lieberman, M., Gauvin, L., Bukowski, W. M., & White, D. R. (2001). Interpersonal influence & disordered eating behaviors in adolescent girls: The role of peer modeling, social reinforcement & body-related teasing. *Eating Behaviors*, *3*, 215–236.

Little, R. J. A., & Rubin, D. B. (1987). *Statistical analysis with missing data*. New York: Wiley.

Lundgren, J. D., Anderson, D. A., Thompson, J. K., Shapiro, J. R., & Paulosky, C. A. (2004). Perception of teasing in underweight persons: A modification of the perception of teasing scale. *Eating and Weight Disorders*, *9*, 139–146.

Lunner, K., Werthem, E. H., Thompson, J. K., Paxton, S. J., McDonald, F., & Halvaarson, K. S. (2000). A cross-cultural examination of weight-related teasing, body image, and eating disturbance in Swedish and Australian samples. *International Journal of Eating Disorders*, *28*, 430–435.

National Eating Disorders Association. (2002). Health consequences of eating disorders. Retrieved July 11, 2005 from http://www.nationaleatingdisorders.org/p.asp?WebPage_ID = 320&Profile_ID = 41143.

National Institute of Mental Health. (2001). Eating disorders: Facts about eating disorders and the search for solutions. Retrieved November 4, 2004 from http://www.nimh.nih.gov/publicat/eatingdisorders.cfm.

Neumark-Sztainer, D., Falkner, N., Story, M., Perry, C., Hannan, P. J., & Mulert, S. (2002). Weight-teasing among adolescents: Correlations with weight status and disordered eating behaviors. *International Journal of Obesity*, *26*, 123–131.

Nolen-Hoeksema, S. (2001). Gender differences in depression. *Current Directions in Psychological Science*, *10*, 173–176.

Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, *1*, 385–401.

Rose, D. T., & Abramson, L. Y. (1992). Developmental predictors of depressive cognitive style: Research and theory. In D. Cicchetti, S. L. Toth (Eds.), *Rochester symposium on developmental psychopathology.* (Vol .IV, pp. 323–349). Rochester, NY: University of Rochester Press.

Rosen, J. C., Jones, A., Ramirez, E., & Waxman, S. (1995). Body Shape Questionnaire: Studies of validity and reliability. *International Journal of Eating Disorders*, *20*, 315–319.

Roth, D. A., Coles, M. E., & Heimberg, R. G. (2002). The relationship between memories for childhood teasing and anxiety and depression in adulthood. *Anxiety Disorders*, *16*, 149–164.

Santor, D. A., Zuroff, D. C., Ramsay, J. O., Cervantes, P., & Palacios, J. (1995). Examining scale discriminability in the BDI and CES-D as a function of depressive severity. *Psychological Assessment*, *7*, 131–139.

Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods*, *7*, 147–177.

Shafran, R., Fairburn, C. G., Nelson, L., & Robinson, P. H. (2003). The interpretation of symptoms of severe dietary restraint. *Behaviour Research and Therapy*, *41*, 887–894.

Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods*, *7*, 422–445.

Spangler, D. L., & Stice, E. (2001). Validation of the beliefs about appearance scale. *Cognitive Therapy and Research*, *25*, 813–827.

Steinhausen, H., Gavez, S., & Metzke, C. W. (2005). Psychosocial correlates, outcome, and stability of abnormal adolescent eating behavior in community samples of young people. *International Journal of Eating Disorders*, *37*, 119–126.

Stone, N. (1993). Parental abuse as a precursor to childhood onset depression and suicidality. *Child Psychiatry and Human Development*, *24*, 13–24.

Storch, E. A., Roth, D. A., Coles, M. E., Heimberg, R. G., Bravata, E. A., & Moser, J. (2004). The measurement and impact of childhood teasing in a sample of young adults. *Anxiety Disorders*, *18*, 681–694.

Striegel-Moore, R. H., Franko, D. L., Thompson, D., Barton, B., Schreiber, G. B., & Daniels, S. R. (2004). Changes in weight and body image over time in women with eating disorders. *International Journal of Eating Disorders*, *36*, 315–327.

Thompson, J. K., Coovert, M. D., Richards, K. J., Johnson, S., & Cattarin, J. (1995). Development of body image, eating disturbance, and general psychological functioning in female adolescents: Covariance structure modeling and longitudinal investigations. *International Journal of Eating Disorders*, *18*, 221–236.

Thompson, J. K., Fabian, L. J., Moulton, D. O., Dunn, M. E., & Altabe, M. N. (1991). Development and validation of the physical appearance related teasing scale. *Journal of Personality Assessment*, *56*, 513–521.

van Strien, T., Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders*, *5*, 295–315.

Wertheim, E. H., Koerner, J., & Paxton, S. J. (2001). Longitudinal predictors of restrictive eating and bulimic tendencies in three different age groups of adolescent girls. *Journal of Youth and Adolescence*, *30*, 69–81.