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# Risk and Maintenance Factors for Eating Pathology: A Meta-Analytic Review

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This meta-analytic review of prospective and experimental studies reveals that several accepted risk factors for eating pathology have not received empirical support (e.g., sexual abuse) or have received contradictory support (e.g., dieting). There was consistent support for less-accepted risk factors(e.g., thin-ideal internalization) as well as emerging evidence for variables that potentiate and mitigate the effects of risk factors(e.g., social support) and factors that predict eating pathology maintenance(e.g., negative affect). In addition, certain multivariate etiologic and maintenance models received preliminary support. However, the predictive power of individual risk and maintenance factors was limited, suggesting it will be important to search for additional risk and maintenance factors, develop more comprehensive multivariate models, and address methodological limitations that attenuate effects.

Eating disorders are one of the most common psychiatric problems faced by women and girls and are characterized by chronicity and relapse (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000; Lewinsohn, Streigel-Moore, & Seeley, 2000). Anorexia nervosa involves emaciation, fear of becoming fat, disturbed perception of body shape, undue influence of shape on self-evaluation, denial of the seriousness of low body weight, and amenorrhea. Bulimia nervosa is marked by uncontrollable binge eating, compensatory behavior to prevent weight gain (e.g., vomiting), and undue influence of shape on self-evaluation. Binge eating disorder entails uncontrollable binge eating in the absence of compensatory behaviors. Eating disorders are marked by psychosocial impairment and comorbid psychopathology and have the highest levels of treatment seeking, inpatient hospitalization, suicide attempts, and mortality of the most common psychiatric syndromes (Newman et al., 1996; Wilson, Heffernan, & Black, 1996). Furthermore, eating pathology increases the risk for onset of obesity, depression, and substance abuse (e.g., Stice, Cameron, Killen, Hayward, & Taylor, 1999; Stice, Hayward, Cameron, Killen, & Taylor, 2000).

Although there has been a burgeoning of longitudinal and experimental studies on the risk and maintenance factors for eating pathology, this literature has not been critically reviewed or synthesized. A review of this nature is needed because many of the risk factors that are widely accepted by researchers and clinicians have not been empirically supported. Moreover, there are several less-accepted risk factors that have received empirical support. Thus, the first aim of this article is to provide such a review in the hope that it will advance knowledge of the etiologic and maintenance processes for this serious psychiatric problem. I used a meta-analytic approach to document the magnitude of effects and provide an objective summary of the literature. The second aim is to review multivariate etiologic and maintenance models that have been tested prospectively, which is crucial because little attention has been paid to how risk factors work together. The third aim is to discuss the methodological, statistical, and theoretical limitations of this literature in an effort to promote more rigorous research. The fourth aim is to delineate the preventive and treatment implications of this literature. I conclude the article with a discussion of directions for future research.

## Risk and Maintenance Factor Terminology

First, I define risk and maintenance factor terminology, in support of efforts to standardize nomenclature (Kraemer et al., 1997). A *risk factor* is a variable that has been shown to prospectively predict some subsequent pathological outcome. Temporal precedence can be established by showing that a variable predicts subsequent onset of a disorder among initially disorder-free individuals (for dichotomous outcomes) or growth in symptoms controlling for initial symptom levels (for continuous outcomes). A variable is considered a *causal risk factor* if, among individuals free of diagnostic levels of the disorder, an experimental increase or decrease results in elevated or reduced symptoms, respectively. Randomized experiments are a powerful tool for understanding etiologic processes because they not only document the direction of influence and temporal precedence but also can rule out third-variable explanations for an effect—something prospective studies cannot do. A *proxy risk factor* is one that shows a prospective relation to a pathological outcome solely because it is correlated with a true causal risk factor (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). By definition, manipulation of a proxy risk factor will not result in a subsequent change in the risk for the outcome. A variable that accounts for the relation between a predictor and a criterion is referred to as a *mediator*, wherein the predictor pre-

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sumably causes change in the mediator, which in turn causes change in the criterion. A variable that mitigates the adverse effects of a risk factor is a *protective factor*.

In this context, it is useful to introduce three new terms. A factor that amplifies the adverse effects of a risk factor is referred to as a *potentiating factor*. Both protective and potentiating factors moderate the effects of a risk factor, but these terms better capture the form of the interactions. A factor that predicts symptom persistence over time versus remission among initially symptomatic individuals is a *maintenance factor*. It is important to distinguish between risk and maintenance factors because the former are germane to prevention program design and the latter to treatment intervention design. If an experimental increase or decrease in a factor among initially symptomatic individuals results in symptom expression or suppression respectively, it may be referred to as a *causal maintenance factor.* One could presume that both protective and potentiating factors mitigate or amplify the effects of maintenance and causal maintenance factors in a fashion parallel to that possible with risk and causal risk factors.

## Empirically Documented Risk and Maintenance Factors for Eating Pathology

I confined this review to prospective and experimental studies because it is not possible to differentiate risk and maintenance factors from concomitants or consequences of a disorder with cross-sectional data. I did not include retrospective studies because retrospective data cannot be used to demonstrate temporal precedence and have been found to be inaccurate (Henry, Moffitt, Caspi, Langley, & Silva, 1994). Only prospective studies that tested whether putative risk and maintenance factors predicted subsequent onset or remission of eating pathology or increases or decreases in eating disorder symptoms were included. I did not include studies that only correlated putative risk and maintenance factors with subsequent eating pathology without controlling for initial levels of eating pathology (e.g., Kendler et al., 1991) because this type of analysis does not establish temporal precedence (baseline elevations in the risk factors could be a consequence of initial eating disturbances). Twenty-five prospective studies were excluded for this reason. The exclusive focus on methodologically rigorous studies is intended to permit the strongest etiological and maintenance inferences. It should be noted that this in no way rules out variables that have not yet been investigated in prospective or experimental studies. Furthermore, even variables that are not shown to have univariate effects on increases or persistence in eating pathology may eventually be found to interact with other factors to predict eating disturbances.

Several procedures were used to retrieve published and unpublished articles. First, a computer search was performed on PsycINFO and *MedLine* for the years 1980–2001 using the following key words: *prospective*, *longitudinal*, *experiment*, *eating disorder*, *eating pathology*, *anorexia*, *anorexic*, *bulimia*, *bulimic*, and *binge eating*. A research assistant, a professional librarian, and I performed independent searches to increase the odds that all relevant articles would be retrieved. I reviewed the products of all three searchers to identify pertinent articles. Second, the tables of contents for journals that commonly publish articles in this area were reviewed for this same period (e.g., *International Journal of Eating Disorders*, *Journal of Abnormal Psychology*, *Journal of Consulting and Clinical Psychology*). Finally, I examined the reference sections of all identified articles, past reviews, and books in this area.

It should be noted that although articles focusing on all three of the eating disorders were sought, those located focused on bulimic symptoms, binge eating, or eating disorder symptom composites; none focused solely on anorexic symptoms. This is unfortunate because the distinct symptom profiles of each eating disorder suggest that the risk and maintenance processes for each may differ. This uneven coverage of the eating disorders in the literature has several implications. First, findings from this review likely reveal most about risk and maintenance processes for bulimic symptoms and binge eating because most studies focused on these outcomes. Even those that focused on eating disorder symptom composites may reveal more about bulimic symptoms and binge eating because these outcomes are more prevalent than anorexic symptoms. Thus, conclusions should be generalized with care to anorexic symptoms. Second, the extant literature is not well positioned to differentiate risk factors that are general to the three eating disorders from those that are specific to a particular eating disorder. This information is crucial for isolating the distinct etiologic processes for the different eating disorders. The focus on the eating disorder symptom composites may also obscure the risk and maintenance factors that are specific to the three eating disorders. Thus, this review may reveal more about the risk and maintenance factors for the symptoms that are common to the various eating disorders versus those that are unique to each. To minimize the potential for confusion, the criteria examined in the studies are accurately described in the text and tables so that the boundaries of knowledge are clearly demarcated.

In this meta-analysis, I focused on all potential risk and maintenance factors that have been examined in at least two prospective or experimental studies located in the literature search (the minimum necessary for meta-analytic methods). All of the studies summarized for each factor were independent—no single study contributed more than one effect size. When more than one report provided effect sizes for the relation between two variables from the same data, the effect size from the more methodologically rigorous report was used (e.g., the one with the longer follow-up). When one report provided multiple effect sizes for the relation between two variables, an average effect size and probability value ($p$ value) was calculated using the fixed effects formulas provided in Shadish and Haddock (1994, pp. 265–266). All effect sizes reflect the univariate relation of a risk or maintenance factor to subsequent change in the outcome because the variation in the number and types of covariates included in multivariate models rendered it inappropriate to combine the resulting effect sizes with those from the univariate models. When only multivariate effects were reported, univariate effects were requested from the authors.[1] Because authors were responsive, there was only one effect (0.5%)

[1] The following authors were kind enough to provide univariate effect sizes when only multivariate effects sizes were reported in the original article: Byely, Archibald, Graber, and Brooks-Gunn (2000); Cole, Martin, Peeke, Seroczynski, and Hoffman (1998); Cooley and Toray (2001a, 2001b); Leon, Fulkerson, Perry, Keel, and Klump (1999); Vogeltanz-Holm et al. (2000); Vohs, Bardone, Joiner, Abramson, and Heatherton (1999); and Vohs, Voelz, et al. (2001).

for which the sole estimate of the effect size was from a multivariate model. If effect sizes were not reported, they were directly calculated by reconstituting the data (e.g., using weighted probability values to conduct a chi-square test) or were estimated from the *p* values using the formula provided in Rosenthal (1991, p. 19). If neither of these options was possible because insufficient information was provided, effect sizes were requested from the authors.[2] Authors were again responsive, and there were only two effects (1%) for which there were missing values. Sensitivity analyses assessed the impact of including or excluding the one multivariate effect and the two missing effects (reported below).

I selected the correlation coefficient (*r*) as the index of effect size because of its similar interpretation across different combinations of interval, ordinal, and nominal variables (Pearson's *r*, Spearman's rho, and point biserial; Rosenthal, 1991). I used Cohen's (1988) criteria for small ($r = .10$), medium ($r = .30$) and large ($r = .50$) effects, and considered effects showing a correlation of less than .05 to be trivial.

Separately for each factor, the prospective and experimental studies testing whether the variable is a risk factor or causal risk factor are first presented. In this context, I discuss prospective studies examining the relations of the risk and maintenance factors to the variables that putatively mediate their effects on eating pathology. Next, I present prospective and experimental studies testing whether a variable is a maintenance factor or causal maintenance factor. Evidence for protective factors and potentiating factors from these studies is noted along with the main effects that they qualify. Effect sizes, *p* values, and key methodological features of the studies (follow-up length, age of participants, use of validated scales, and outcome examined) are summarized in the tables. I calculated the average effect sizes and associated *p* values using the fixed effects models detailed in Shadish and Haddock (1994, pp. 265–266).[3] Analyses tested whether there was statistically significant heterogeneity in the effect sizes for the relations between factors and outcomes using the fixed effects *Q* test described in Shadish and Haddock (1994, pp. 266). In the event of significant heterogeneity, univariate weighted least squares analyses tested for factors that moderate the magnitude of the effects using the approach described by Hedges (1994, pp. 295–298).[4] I used a Bonferroni correction for each of these sets of analyses to decrease the risk of false positive findings (Type I errors). Potential moderators were identified on an individual basis for each relation by examining methodological factors that appeared to covary with effect size.

### *Body Mass*

Elevated adiposity theoretically results in increased social pressure to be thin and body dissatisfaction, which putatively lead to dieting, negative affect, and consequent increased risk for eating pathology (Cattarin & Thompson, 1994; Halmi, Falk, & Schwartz, 1981). Elevated adiposity is thought to result in amplified pressure from family and peers to be thinner. This pressure may range from negative comments about weight to attempts to limit the caloric intake of children. Elevated adiposity also likely contributes to body dissatisfaction, as the culturally defined ideal for attractiveness currently favors thinness.

As summarized in Table 1, elevated body mass predicted subsequent increases in perceived pressure to be thin, body dissatisfaction, and dieting (Cattarin & Thompson, 1994; Field et al., 2001; Patton, Johnson-Sabine, Wood, Mann, & Wakeling, 1990; Stice, Mazotti, Krebs, & Martin, 1998; Stice & Whitenton, in press; Vogeltanz-Holm et al., 2000), although some of these relations did not replicate in other studies (Byely et al., 2000; Cooley & Toray, 2001b). Body mass did not predict increases in depression (Lewinsohn et al., 1994; Stice & Bearman, 2001; Stice, Hayward, et al., 2000). Body mass predicted onset of bulimic pathology (Killen et al., 1994) and binge eating (Stice, Presnell, & Spangler, 2002; Vogeltanz-Holm et al., 2000) and increases in eating disorder symptoms (Wichstrom, 2000). However, body mass did not predict onset or increases in bulimic symptoms (Cattarin & Thompson, 1994; Cooley & Toray, 2001b; Killen et al., 1996; Stice & Agras, 1998) or eating disorder symptoms (Gardner et al., 2000; Graber, Brooks-Gunn, Paikoff, & Warren, 1994; Keel, Fulkerson, & Leon, 1997) in other studies. Body mass did not emerge as a significant maintenance factor for bulimic symptoms (Fairburn et al., in press; Stice & Agras, 1998). The magnitude of the average effect of body mass on perceived pressure to be thin was medium, but the effects on body dissatisfaction and dieting were small and the effects on increases in eating pathology were trivial but significant.[5] The effects for negative affect and eating pathology maintenance were trivial and nonsignificant. There was no significant heterogeneity in the effect sizes for body mass.[6] However, the lack of significant heterogeneity in

[2] The following authors were kind enough to provide effect sizes when this information was not provided in the original article: Field, Camargo, Taylor, Berkey, and Colditz (1999); Field et al. (2001); Lewinsohn et al. (1994); Lowe, Foster, Kerzhnerman, Swain, and Wadden (2001); Vogeltanz-Holm et al. (2000); and Wichstrom (2000). Wertheim, Koerner, and Paxton (2001) were generous enough to provide effect sizes for the entire sample, when the effects were reported separately by grade level in the original article. Gardner, Stark, Friedman, and Jackson (2000) were kind enough to share their data so that effect sizes could be calculated for univariate analyses that controlled for initial levels of eating pathology.

[3] Fixed-effects rather than random-effects models were used because the intent of this article was to summarize the studies reviewed in this meta-analysis rather than to generalize my inferences to studies not included in this review. Moreover, random-effects models have less statistical power when there are only a few studies to be summarized (Shadish & Haddock, 1994), which was often the case in this review. Finally, the fact that there was nonsignificant heterogeneity in effect sizes for 78% of the relations examined here provides some evidence that fixed effects models were appropriate (Shadish & Haddock, 1994). Because multiple outcomes were examined for each risk factor, a Bonferroni correction was used for the set of analyses conducted for each risk factor.

[4] It was necessary to use univariate rather than multivariate analyses because (a) in most instances there were insufficient degrees of freedom to permit estimation of the multivariate models and (b) there was often perfect colinearity in the moderators (i.e., singularity) because methodological limitations tended to covary across studies.

[5] Sensitivity analyses indicated that the magnitude of the average effect, test of the significance of the average effect, and test of the heterogeneity of effects was identical when the one multivariate effect size (Keel et al., 1997) was excluded.

[6] Although there was no significant heterogeneity in effect sizes for the relation of body mass to subsequent body dissatisfaction, it should be noted that there is some suggestive evidence that this relation might be weaker in certain ethnic groups (e.g., Black women; Smolak & Striegel-Moore, 2001).

Table 1
*Relation of Initial Body Mass to Subsequent Change in Eating Pathology and Change in the Putative Mediators of This Effect*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in pressure to be thin | | | | | | | |
| Cattarin & Thompson (1994) | 36 mo | Adol | Yes | | 87 | .33 | .001 |
| Increase in body dissatisfaction | | | | | | | |
| Byely et al. (2000) | 12 mo | Adol | No | | 52 | .02 | .916 |
| Cattarin & Thompson (1994) | 36 mo | Adol | Yes | | 87 | .22 | .018 |
| Field et al. (2001) | 12 mo | Child | No | | 4,966 | .16 | .001 |
| Stice & Whitenton (in press) | 12 mo | Adol | Yes | | 484 | .19 | .001 |
| Vogeltanz-Holm et al. (2000) | 60 mo | Adult | Yes | | 709 | .10 | .005 |
| Average | | | | | | .16* | |
| Increase in dieting | | | | | | | |
| Byely et al. (2000) | 12 mo | Adol | No | | 52 | .11 | .574 |
| Cooley & Toray (2001b) | 7 mo | Adult | Yes | | 104 | .17 | .095 |
| Field et al. (2001) | 12 mo | Child | No | | 4,966 | .10 | .001 |
| Patton et al. (1990) | 12 mo | Adol | No | | 176 | .31 | .001 |
| Stice, Mazotti, et al. (1998) | 9 mo | Adol | Yes | | 218 | .13 | .017 |
| Average | | | | | | .11* | |
| Increase in negative affect | | | | | | | |
| Lewinsohn et al. (1994) | 12 mo | Adol | Yes | | 1,508 | .00 | .909 |
| Stice & Bearman (2001) | 20 mo | Adol | Yes | | 231 | .03 | .651 |
| Stice, Hayward, et al. (2000) | 36 mo | Adol | Yes | | 1,024 | .05 | .062 |
| Average | | | | | | .02 | |
| Increase in eating pathology | | | | | | | |
| Cattarin & Thompson (1994) | 36 mo | Adol | Yes | BUL | 87 | .09 | .211 |
| Cooley & Toray (2001b) | 7 mo | Adult | Yes | BUL | 104 | .10 | .310 |
| Gardner et al. (2000) | 24 mo | Child | Yes | Comp | 86 | .14 | .202 |
| Graber et al. (1994) | 96 mo | Adol | Yes | Comp | 85 | .01 | .937 |
| Keel et al. (1997) | 12 mo | Child | Yes | Comp | 80 | .17 | .059 |
| Killen et al. (1994) | 24 mo | Adol | Yes | BUL | 887 | .07 | .050 |
| Killen et al. (1996) | 36 mo | Adol | Yes | BUL | 825 | .06 | .130 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BED | 218 | .10 | .178 |
| Stice et al. (2002) | 20 mo | Adol | Yes | BED | 231 | .18 | .016 |
| Vogeltanz-Holm et al. (2000) | 60 mo | Adult | Yes | BED | 709 | .07 | .038 |
| Wichstrom (2000) | 24 mo | Adol | No | Comp | 7,751 | .02 | .040 |
| Average | | | | | | .04* | |
| Maintenance of eating pathology | | | | | | | |
| Fairburn et al. (in press) | 60 mo | Adult | Yes | BUL | 102 | .04 | .258 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BED | 218 | −.05 | .677 |
| Average | | | | | | −.02 | |

*Note.* ED = eating disorder; mo = month; Adol = adolescent; BUL = bulimic pathology; Comp = symptom composite; BED = binge eating disorder. *$\alpha < .008$.

effect sizes here and below might be a function of the limited statistical power resulting from the small number of studies examined. Results from analyses that focus on a small number of studies should be interpreted cautiously.

Collectively, findings suggest that body mass can be considered a risk factor for perceived pressure to be thin, body dissatisfaction, and dieting but that body mass does not appear to be a risk factor for negative affect and eating pathology or a maintenance factor for eating pathology. This pattern of findings implies that body mass may play a more important role in promoting the risk factors for eating pathology than in directly fostering or maintaining eating disturbances.

### *Sociocultural Pressure to Be Thin*

The sociocultural model of eating pathology posits that social pressure to be thin fosters an internalization of the thin ideal and body dissatisfaction, which in turn place individuals at risk for dieting, negative affect, and eating pathology (Pyle, Mitchell, & Eckert, 1981; Striegel-Moore, Silberstein, & Rodin, 1986). Pressure to be thin from family, peers, and media theoretically contributes to an internalization of the thin ideal and a generalized overvaluation of the importance of appearance. Elevated pressure to be thin is also thought to lead to body dissatisfaction, as repeated messages that one is not thin enough likely causes discontent with one's body. Furthermore, pressure to be thin may directly promote dieting in the absence of body dissatisfaction because people might believe that this would reduce social pressures to be thin.

As indicated in Table 2, perceived pressure to be thin predicted increases in body dissatisfaction, dieting, and negative affect (Cattarin & Thompson, 1994; Field et al., 2001; Stice, 2001; Stice & Bearman, 2001; Stice, Mazotti et al., 1998; Stice & Whitenton, in press; Wertheim, Koerner, & Paxton, 2001), although null findings were also observed (Byely et al., 2000). Perceived pressure to be thin predicted onset of binge eating (Stice et al., 2002) and bulimic

Table 2
*Relation of Initial Perceived Pressure to be Thin to Subsequent Change Eating Pathology and Change in the Putative Mediators of This Effect*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in body dissatisfaction | | | | | | | |
| Byely et al. (2000) | 12 mo | Adol | No | | 52 | .08 | .404 |
| Cattarin & Thompson (1994) | 36 mo | Adol | Yes | | 87 | .25 | .011 |
| Field et al. (2001) | 12 mo | Child | No | | 4,966 | .07 | .001 |
| Stice (2001) | 20 mo | Adol | Yes | | 231 | .14 | .043 |
| Stice & Whitenton (in press) | 12 mo | Adol | Yes | | 484 | .19 | .001 |
| Average | | | | | | .09* | |
| Increase in dieting | | | | | | | |
| Byely et al. (2000) | 12 mo | Adol | No | | 52 | .23 | .092 |
| Field et al. (2001) | 12 mo | Child | No | | 4,966 | .05 | .001 |
| Stice (2001) | 20 mo | Adol | Yes | | 231 | .25 | .001 |
| Stice, Mazotti, et al. (1998) | 9 mo | Adol | Yes | | 218 | .14 | .007 |
| Wertheim et al. (2001) | 8 mo | Adol | Yes | | 432 | .18 | .001 |
| Average | | | | | | .07* | |
| Increase in negative affect | | | | | | | |
| Stice & Bearman (2001) | 20 mo | Adol | Yes | | 231 | .21 | .002 |
| Increase in eating pathology | | | | | | | |
| Field et al. (1999) | 12 mo | Child | No | BUL | 6,928 | .11 | .001 |
| Gardner et al. (2000) | 24 mo | Child | No | Comp | 86 | .07 | .512 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .22 | .001 |
| Stice et al. (2002) | 20 mo | Adol | Yes | BED | 231 | .14 | .038 |
| Wertheim et al. (2001) | 8 mo | Adol | Yes | Comp | 432 | .18 | .001 |
| Average | | | | | | .12* | |
| Maintenance of eating pathology | | | | | | | |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .16 | .245 |

*Note.* ED = eating disorder; mo = month; Adol = adolescent; BUL = bulimic pathology; Comp = symptom composite; BED = binge eating disorder. *$\alpha < .01$.

symptoms (Field, Camargo, Taylor, Berkey, & Coditz, 1999; Stice & Agras, 1998) and increases in eating pathology (Wertheim et al., 2001), but some effects were nonsignificant (Gardner et al., 2000). Perceived pressure to be thin predicted bulimic symptom maintenance (Stice & Agras, 1998). The average effect of perceived pressure to be thin on negative affect was medium, the effects on body dissatisfaction, dieting, and bulimic pathology were small, and the effect on eating pathology maintenance was small but nonsignificant.

There was significant heterogeneity in the effect sizes for the relation of pressure to be thin on dieting, $Q(4) = 16.86$, $p < .005$. Effect sizes were significantly larger ($z = 3.78$, $p < .001$) for studies that used measures with established reliability and validity (mean $r = .19$) versus unvalidated measures (mean $r = .05$; see Table 2). Presumably, validated measures have greater sensitivity to detect effects. There was no statistically significant residual heterogeneity in effect sizes in this model or in any other univariate model examining the predictors of heterogeneous effect sizes reported below. Effect sizes were also significantly larger ($z = 3.99$, $p < .001$) for studies that examined adolescents (mean $r = .19$) versus preadolescents (mean $r = .05$; see Table 2). It may be that studies examining adolescents had greater power to detect effects because this is the developmental period during which eating disturbances typically emerge (Stice, Killen, Hayward, & Taylor, 1998). Length of follow-up was not significantly related to the magnitude of effect sizes.

Numerous lab-based experiments have examined the acute effects of exposure to media-portrayed thin-ideal images, which is one ubiquitous form of pressure to be thin. A recent meta-analysis of this literature (Groesz, Levine, & Murnen, 2002) indicated that exposure to thin-ideal images resulted in immediate increases in body dissatisfaction (mean $r = .15$, range = –.14 to .49, $p < .001$). Groesz et al. (2002) found that the adverse effects of exposure to thin-ideal images on body dissatisfaction were significantly stronger for women with initial elevations in body dissatisfaction ($r = .24$) versus women who were satisfied with their bodies ($r = .05$). Individuals with preexisting body dissatisfaction may be more likely to engage in social comparison processes with thin models, which might result in more adverse effects from media exposure.

Randomized experiments have also provided evidence that exposure to thin-ideal images results in acute increases in negative affect (see Table 3; Cattarin, Thompson, Thomas, & Williams, 2000; Irving, 1990; Stice & Shaw, 1994), although two studies generated null findings (Heinberg & Thompson, 1995; Myers & Biocca, 1992). The average effects of exposure to thin-ideal images on body dissatisfaction and negative affect were small but significant.

There was significant heterogeneity in the effect sizes for the relation of exposure to thin-ideal images to negative affect, $Q(4) = 16.32$. $p < .001$. However, there was no variability in the developmental age of participants or the use of validated measures across studies, so these factors could not explain variation in effect sizes.

One experiment examined the long-term effects of exposure to media-portrayed thin-ideal images in the natural environment (Stice, Spangler, & Agras, 2001). Results indicated that a 15-

Table 3
*Relation of Exposure to Media-Portrayed Thin-Ideal Images to Subsequent Change in Negative Affect*

| Outcome | Methodological features | | | | |
|---|---|---|---|---|---|
| | Age | Validated scale | Sample size | Effect size (*r*) | *p* |
| Increase in negative affect | | | | | |
| Cattarin et al. (2000) | Adult | Yes | 180 | .41 | .001 |
| Heinberg & Thompson (1995) | Adult | Yes | 138 | .05 | .164 |
| Irving (1990) | Adult | Yes | 162 | .20 | .013 |
| Myers & Biocca (1992) | Adult | Yes | 76 | −.07 | .738 |
| Stice & Shaw (1994) | Adult | Yes | 157 | .15 | .019 |
| Average | | | | .19 | .001 |

month fashion magazine subscription had no main effects on increases in thin-ideal internalization, body dissatisfaction, dieting, negative affect, or bulimic symptoms over time (respective *r*s –.05, .06, .04, .07, .07; all *p* values $>$ .15). Yet, there was evidence that the fashion magazine subscription led to increased negative affect for girls with initial elevations in perceived pressure to be thin ($r$ = .19, $p$ = .004) and body dissatisfaction ($r$ = .14, $p$ = .019), and to increased body dissatisfaction, dieting, and bulimic symptoms for girls with initial deficits in social support ($r$ = .15, $p$ = .033; $r$ = .12, $p$ = .047; $r$ = .13, $p$ = .033, respectively).

Collectively, prospective and experimental findings suggest that perceived pressure to be thin is a causal risk factor for body dissatisfaction, dieting, negative affect, and bulimic pathology, although most of this evidence comes from experiments examining the acute effects of media exposure. There is also evidence that the adverse effects of perceived pressure to be thin are more pronounced for initially at-risk individuals. Perceived pressure to be thin was a nonsignificant maintenance factor for bulimic pathology (though this effect was moderate). The pattern of effects suggested that perceived pressure to be thin might play a more important role in fostering eating pathology than in maintaining it.

### *Modeling*

Direct modeling of body image and eating disturbances by family members and peers is also thought to increase the risk that a youth will emulate these disturbed attitudes and behaviors (Attie & Brooks-Gunn, 1989; Pike & Rodin, 1991). In addition, body image and eating disturbances in one's proximal environment likely reinforces a pursuit of the thin ideal (Stice, 1998).

As summarized in Table 4, modeling of body image and eating pathology did not predict increases in body dissatisfaction or dieting (Byely et al., 2000) but did predict onset of binge eating (Stice et al., 2002) and increases in bulimic symptoms (Stice, 1998). Although the effects for parental modeling of eating pathology might be partially due to genetic factors, effects for peer modeling were also observed (Stice, 1998). The effect for modeling of body image and eating disturbances on body dissatisfaction and dieting were nonsignificant, but the average effect for bulimic pathology onset was small and significant. There was no significant heterogeneity in the effect sizes.

Collectively, findings provide no support for the assertion that modeling is a risk factor for body dissatisfaction or dieting. However, there is emerging evidence that modeling is a risk factor for bulimic pathology. These findings should be interpreted cautiously because of the small number of studies that have examined this factor.

### *Thin-Ideal Internalization*

Theoretically, internalization of the thin ideal and overvaluation of the importance of appearance promote body dissatisfaction, which in turn fosters dieting, negative affect, and eating pathology

Table 4
*Relation of Initial Modeling of Body Image and Eating Disturbances to Subsequent Change in Eating Pathology and Change in the Putative Mediators of This Effect*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in body dissatisfaction | | | | | | | |
| Byely et al. (2000) | 12 mo | Adol | No | | 52 | .01 | .402 |
| Increase in dieting | | | | | | | |
| Byely et al. (2000) | 12 mo | Adol | No | | 52 | .01 | .437 |
| Increase in eating pathology | | | | | | | |
| Stice (1998) | 9 mo | Adol | Yes | BUL | 218 | .16 | .002 |
| Stice et al. (2002) | 20 mo | Adol | Yes | BED | 231 | .16 | .020 |
| Average | | | | | | .16* | |

*Note.* ED = eating disorder; mo = month; Adol = adolescent; BUL = bulimic pathology; BED = binge eating disorder.
*$\alpha < .017$.

Table 5
*Relation of Initial Thin-Ideal Internalization to Subsequent Change in Eating Pathology and Change in the Putative Mediators of This Effect*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in body dissatisfaction | | | | | | | |
| Stice (2001) | 20 mo | Adol | Yes | | 231 | .25 | .001 |
| Stice & Whitenton (in press) | 12 mo | Adol | Yes | | 484 | .14 | .002 |
| Average | | | | | | .18* | |
| Increase in dieting | | | | | | | |
| Stice (2001) | 20 mo | Adol | Yes | | 231 | .29 | .001 |
| Stice, Mazotti, et al. (1998) | 9 mo | Adol | Yes | | 218 | .03 | .098 |
| Average | | | | | | .17* | |
| Increase in negative affect | | | | | | | |
| Stice (2001) | 20 mo | Adol | Yes | | 231 | .17 | .009 |
| Increase in eating pathology | | | | | | | |
| Field et al. (1999) | 12 mo | Child | No | BUL | 6,928 | .13 | .001 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .15 | .005 |
| Stice et al. (2002) | 20 mo | Adol | Yes | BED | 231 | .22 | .003 |
| Wichstrom (2000) | 24 mo | Adol | No | Comp | 7,751 | .03 | .016 |
| Average | | | | | | .08* | |
| Maintenance of eating pathology | | | | | | | |
| Fairburn et al. (in press) | 60 mo | Adult | Yes | BUL | 102 | .22 | .041 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .21 | .015 |
| Average | | | | | | .21* | |

*Note.* ED = eating disorder; mo = month; Adol = adolescent; BUL = bulimic pathology; BED = binge eating disorder; Comp = symptom composite. *$\alpha < .01$.

(Garner, Olmsted, & Polivy, 1983). Thin-ideal internalization and appearance overvaluation putatively contribute to body dissatisfaction because this ideal is difficult to attain. Thin-ideal internalization may also promote dieting in the absence of body dissatisfaction because people may be motivated to pursue a culturally valued body shape to gain social approval. Appearance overvaluation has also been hypothesized to be a maintenance factor for bulimic pathology (Fairburn, 1997). The belief that improved appearance would produce a wide variety of interpersonal and emotional benefits is thought to promote the restrictive dieting that maintains binge eating.

As expected (Table 5), thin-ideal internalization predicted increases in body dissatisfaction, dieting, and negative affect (Stice, 2001; Stice & Whitenton, in press), although one effect was only marginally significant (Stice, Mazotti, et al., 1998). Thin-ideal internalization also predicted the onset of binge eating (Stice et al., 2002) and bulimic symptoms (Field et al., 1999; Stice & Agras, 1998; Stice et al., 2002) and increases in eating disorder symptoms (Wichstrom, 2000), as well as bulimic symptom maintenance (Fairburn et al., in press; Stice & Agras, 1998). The effect of thin-ideal internalization on bulimic pathology maintenance was medium and the effects on body dissatisfaction, dieting, negative affect, and eating pathology were small.

There was significant heterogeneity in the effects for thin-ideal internalization on dieting, $Q(1) = 7.49$, $p < .01$. Although it was not possible to test for predictors of the heterogeneous effects because there were only two studies (and thus only one degree of freedom), the study with the 24-month follow-up had a larger effect than the study with a 9-month follow-up ($r = .29$ vs. .03; see Table 5). Studies with longer follow-up periods may be more likely to find effects because they model change in the outcome more reliably. There was also significant heterogeneity in the effects for thin-ideal internalization on eating pathology, $Q(3) = 7.49$, $p < .01$. Effect sizes were significantly larger ($z = 6.25$, $p < .001$) for studies predicting binge eating or bulimic pathology (mean $r = .14$) rather than general eating pathology (mean $r = .03$; see Table 5). No other methodological feature examined varied across the studies. Findings suggest that subscription to the current thin ideal may play a more important role in the genesis of bulimic symptoms versus general eating disturbances.[7]

An experimental trial found that an intervention that reduced thin-ideal internalization resulted in decreased body dissatisfaction, dieting, negative affect, and bulimic symptoms relative to a waitlist control condition (Stice, Mazotti, Weibel, & Agras, 2000; see Table 6). A second trial that compared this thin-ideal internalization intervention to a healthy weight management intervention found that both interventions produced decreases in these outcomes (Stice, Chase, Stormer, & Appel, 2001). The effects of this thin-ideal internalization intervention on each of the outcomes were medium. There was no significant heterogeneity in effect sizes.

Thus, prospective and experimental findings suggest that thin-ideal internalization is a causal risk factor for body dissatisfaction, dieting, negative affect, and bulimic pathology, as well as a main-

[7] Although length of follow-up and developmental age of study participants were significantly related to effect sizes, the effects were in the opposite direction than expected because of the extremely small effect size for the largest study (which was weighted the most in the analyses). These results are not presented because they seemed to be an artifact of the weighting system resulting from the marked disparity in sample sizes.

Table 6
*Effects of an Intervention That Decreased Thin-Ideal Internalization on Subsequent Change in Eating Pathology and Change in the Putative Mediators of This Effect*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Decrease in body dissatisfaction | | | | | | | |
| Stice, Chase, et al. (2001) | 1 mo | Adult | Yes | | 87 | .23 | .001 |
| Stice, Mazotti, et al. (2000) | 1 mo | Adult | Yes | | 30 | .45 | .004 |
| Average | | | | | | .29* | |
| Decrease in dieting | | | | | | | |
| Stice, Chase, et al. (2001) | 1 mo | Adult | Yes | | 87 | .30 | .001 |
| Stice, Mazotti, et al. (2000) | 1 mo | Adult | Yes | | 30 | .35 | .023 |
| Average | | | | | | .31* | |
| Decrease in negative affect | | | | | | | |
| Stice, Chase, et al. (2001) | 1 mo | Adult | Yes | | 87 | .23 | .001 |
| Stice, Mazotti, et al. (2000) | 1 mo | Adult | Yes | | 30 | .42 | .008 |
| Average | | | | | | .28* | |
| Decrease in eating pathology | | | | | | | |
| Stice, Chase, et al. (2001) | 1 mo | Adult | Yes | BUL | 87 | .23 | .001 |
| Stice, Mazotti, et al. (2000) | 1 mo | Adult | Yes | BUL | 30 | .36 | .020 |
| Average | | | | | | .26* | |

*Note.* ED = eating disorder; mo = month; BUL = bulimic pathology.
*$\alpha < .012$.

tenance factor for bulimic pathology. However, these conclusions should be interpreted with caution because most of the studies were from one research group.

### *Body Dissatisfaction*

Theoretically, body dissatisfaction promotes dieting and negative affect, which in turn increase the risk for eating pathology (Bruch, 1962; Crisp, 1984). Body dissatisfaction putatively leads to dieting because of the belief that this is an effective weight control technique. Body dissatisfaction may also foster negative affect because appearance is a central evaluative dimension for women. Finally, body dissatisfaction may directly promote compensatory behaviors that characterize some eating disorders (e.g., vomiting).

As indicated in Table 7, body dissatisfaction predicted increases in dieting (Cooley & Toray, 2001a; Patton et al., 1990; Stice, 2001; Stice, Mazotti et al., 1998; Wertheim et al., 2001) and negative affect (Cole et al., 1998; Rierdan, Koff, & Stubbs, 1989; Stice & Bearman, 2001; Stice, Hayward, et al., 2000). Body dissatisfaction also predicted bulimic symptom onset (Field et al., 1999; Killen et al., 1994, 1996; Stice & Agras, 1998) and increases in bulimic pathology (Cooley & Toray, 2001a; Stice, 2001) and eating pathology (Graber et al., 1994; Leon, Fulkerson, Perry, Keel, & Klump, 1999; Wertheim et al., 2001; Wichstrom, 2000). However, other studies did not find significant relations between body dissatisfaction and increases in bulimic symptoms (Vohs, Voelz, et al., 2001) and eating disorder symptoms (Gardner et al., 2000; Keel et al., 1997). Finally, body dissatisfaction predicted maintenance of bulimic symptoms (Stice & Agras, 1998). The effects for body dissatisfaction on increases in dieting and bulimic pathology maintenance were medium, whereas the effects on negative affect and eating pathology were small.[8]

There was significant heterogeneity in the effect sizes for the relation between body dissatisfaction and negative affect, $Q(3) = 8.75$, $p < .05$. Effect sizes were significantly larger ($z = 2.50$, $p < .012$) for studies predicting change in depressive symptoms (mean $r = .18$) than for those predicting major depression onset (mean $r = .08$; see Table 7). Presumably, this was because the continuous variables were more sensitive to change than the dichotomous variables. Length of follow-up was not significantly related to effect size, and there was no variability in the age of participants or the use of validated measures across studies.[9]

In sum, results suggest that body dissatisfaction is a risk factor for dieting, negative affect, and eating pathology and a maintenance factor for bulimic pathology. This variable emerged as one

[8] Sensitivity analyses indicated that the magnitude of the average effect for body dissatisfaction on increases in eating pathology and the corresponding test of significance was identical when (a) the one missing effect was excluded (Keel et al., 1997), (b) a value of .00 was inserted, or (c) the mean effect size was inserted.

[9] There is some possibility that the effects of body dissatisfaction might differ for normal weight versus overweight individuals. Unfortunately, because few of the studies reported average body mass of the sample, it was not possible to test whether weight moderated the effect sizes for the relation of body dissatisfaction to subsequent increases in eating pathology. To address this question, post hoc analyses tested whether the relation between initial body dissatisfaction and change in bulimic pathology was moderated by initial body mass with data from four prospective data sets available to the author (Stice, 2001; Stice & Agras, 1998; Stice, Killen, et al., 1998; Stice, Presnell, & Bearman, 2001). The interaction between initial body dissatisfaction and initial body mass in the prediction increases in bulimic symptoms was not significant in any of these four data sets (mean $r = .02$, $p = .231$). Thus, there is little support for the assertion that the relation between body dissatisfaction and increases in bulimic pathology is moderated by body mass.

Table 7
*Relation of Initial Body Dissatisfaction to Subsequent Change in Eating Pathology and Change in the Putative Mediators of This Effect*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in dieting | | | | | | | |
| Cooley & Toray (2001a) | 36 mo | Adult | Yes | | 117 | .38 | .001 |
| Patton et al. (1990) | 12 mo | Adol | No | | 176 | .31 | .001 |
| Stice (2001) | 20 mo | Adol | Yes | | 231 | .35 | .001 |
| Stice, Mazotti, et al. (1998) | 9 mo | Adol | Yes | | 218 | .20 | .001 |
| Wertheim et al. (2001) | 8 mo | Adol | Yes | | 432 | .18 | .001 |
| Average | | | | | | .26* | |
| Increase in negative affect | | | | | | | |
| Cole et al. (1998) | 6 mo | Adol | Yes | | 692 | .14 | .009 |
| Rierdan et al. (1989) | 6 mo | Adol | Yes | | 505 | .20 | .001 |
| Stice & Bearman (2001) | 20 mo | Adol | Yes | | 231 | .26 | .001 |
| Stice, Hayward, et al. (2000) | 36 mo | Adol | Yes | | 1,024 | .08 | .003 |
| Average | | | | | | .14* | |
| Increase in eating pathology | | | | | | | |
| Cooley & Toray (2001a) | 36 mo | Adult | Yes | BUL | 117 | .22 | .018 |
| Field et al. (1999) | 12 mo | Child | No | BUL | 6,928 | .14 | .001 |
| Gardner et al. (2000) | 24 mo | Child | Yes | Comp | 86 | .05 | .649 |
| Graber et al. (1994) | 96 mo | Adol | Yes | Comp | 85 | .24 | .001 |
| Keel et al. (1997) | 12 mo | Child | Yes | Comp | 80 | — | — |
| Killen et al. (1994) | 24 mo | Adol | Yes | BUL | 887 | .15 | .001 |
| Killen et al. (1996) | 36 mo | Adol | Yes | BUL | 825 | .12 | .010 |
| Leon et al. (1999) | 36 mo | Adol | Yes | Comp | 736 | .13 | .001 |
| Stice (2001) | 20 mo | Adol | Yes | BUL | 231 | .22 | .001 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .28 | .001 |
| Vohs, Voelz, et al. (2001) | 1 mo | Adult | Yes | BUL | 70 | .06 | .310 |
| Wertheim et al. (2001) | 8 mo | Adol | Yes | Comp | 432 | .14 | .001 |
| Wichstrom (2000) | 24 mo | Adol | No | Comp | 7,751 | .10 | .001 |
| Average | | | | | | .13* | |
| Maintenance of eating pathology | | | | | | | |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .30 | .003 |

*Note.* A dash indicates a missing effect size. ED = eating disorder; mo = month; Adol = adolescent; BUL = bulimic pathology; Comp = symptom composite.
*$\alpha < .012$.

of the most consistent and robust risk and maintenance factors for eating pathology.

### *Dieting*

The dietary restraint model posits that caloric deprivation increases the risk for onset of binge eating and bulimia nervosa (Hawkins & Clement, 1984; Polivy & Herman, 1985) and that it contributes to the maintenance of binge eating (Fairburn, 1997). Dieting theoretically fosters eating pathology because individuals may binge eat to counteract the effects of caloric deprivation. It has been suggested that dieting results in depletion of tryptophan, a precursor of serotonin, which increases the likelihood of binge eating high-carbohydrate food to restore tryptophan levels (Kaye, Gendall, & Strober, 1998). Dieting might also promote binge eating because violating strict dietary rules can result in disinhibited eating (the abstinence-violation effect; Marlatt & Gordon, 1985). Moreover, dieting entails a shift from a reliance on physiological to cognitive control over eating behaviors, which leaves the individual vulnerable to disinhibited eating when these cognitive processes are disrupted. Dieting may also contribute to negative affect, which increases the odds that an individual might binge eat to improve his or her mood.

Consistent with the dietary restraint model (Table 8), self-reported dieting predicted increases in negative affect (Stice & Bearman, 2001; Stice, Hayward, et al., 2000), bulimic symptoms (Field et al., 1999; Killen et al., 1994, 1996; Stice, 2001; Stice & Agras, 1998), and eating pathology (Leon et al., 1999; Patton et al., 1990; Santonastaso, Friederici, & Favaro, 1999), although null findings have been reported (Cooley & Toray, 2001a). Self-reported dieting predicted bulimic symptom maintenance in one study (Stice & Agras, 1998) but not another (Fairburn et al., in press). The effect of self-reported dieting on maintenance of bulimic pathology was medium, and the effects on increases in negative affect and eating pathology were small.

There was significant heterogeneity in the effect sizes for the relation of self-reported dieting to eating pathology, $Q(8) = 33.08$, $p < .001$. Effect sizes were significantly larger ($z = 3.68$, $p < .001$) for studies that examined adolescents (mean $r = .20$) versus preadolescents or adults (mean $r = .12$). Effect sizes were also significantly larger ($z = 2.95$, $p < .003$) for studies using validated (mean $r = .19$) rather than unvalidated measures (mean $r = .13$; see Table 8). Length of follow-up, average body mass, and the outcome examined were not related to effect sizes.

Table 8
*Relation of Initial Self-Reported Dieting to Subsequent Change Eating Pathology and Change in the Putative Mediators of This Effect*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in negative affect | | | | | | | |
| Stice & Bearman (2001) | 20 mo | Adol | Yes | | 231 | .22 | .001 |
| Stice, Hayward, et al. (2000) | 36 mo | Adol | Yes | | 1,024 | .15 | .001 |
| Average | | | | | | .16* | |
| Increase in eating pathology | | | | | | | |
| Cooley & Toray (2001a) | 36 mo | Adult | Yes | BUL | 117 | .09 | .329 |
| Field et al. (1999) | 12 mo | Child | No | BUL | 6,928 | .12 | .001 |
| Killen et al. (1994) | 24 mo | Adol | Yes | BUL | 887 | .11 | .001 |
| Killen et al. (1996) | 36 mo | Adol | Yes | BUL | 825 | .21 | .010 |
| Leon et al. (1999) | 36 mo | Adol | Yes | Comp | 736 | .18 | .001 |
| Patton et al. (1990) | 12 mo | Adol | No | Comp | 176 | .30 | .001 |
| Santonastaso et al. (1999) | 12 mo | Adol | No | Comp | 72 | .21 | .040 |
| Stice (2001) | 20 mo | Adol | Yes | BUL | 231 | .36 | .001 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .38 | .001 |
| Average | | | | | | .15* | |
| Maintenance of eating pathology | | | | | | | |
| Fairburn et al. (in press) | 60 mo | Adult | Yes | BUL | 102 | .13 | .256 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .26 | .008 |
| Average | | | | | | .22* | |

*Note.* ED = eating disorder; mo = month; Adol = adolescent; BUL = bulimic pathology; Comp = symptom composite.
*$\alpha < .017$.

Experiments that directly manipulated caloric deprivation provided less consistent support for the dietary model (see Table 9).[10] On the one hand, experiments have tended to find experimentally manipulated caloric deprivation to be positively related to lab-assessed caloric intake. Acute caloric deprivation (4 to 19 hr), relative to no-deprivation control conditions, resulted in elevated ad lib caloric intake among individuals without an eating disorder (Hetherington, Stoner, Andersen, & Rolls, 2000; Spiegel, Shrager, & Steller, 1989; Telch & Agras, 1996b), although this effect was only marginally significant in one study (Schachter, Goldman, & Gordon, 1968). Assignment to longer term diets (2 days to 8 weeks), versus no-diet control conditions, resulted in significantly (Wardle & Beales, 1988) and marginally significantly elevated caloric intake (Lowe, Foster, Kerzhnerman, Swain, & Wadden, 2001), but two studies found null effects (Lowe, 1992, 1994). Three studies found that acute caloric deprivation resulted in increased ad lib caloric intake and binge eating among individuals with bulimia nervosa and binge eating disorder (Agras & Telch, 1998; Heatherington et al., 2000; Telch & Agras, 1996b). The effects of caloric deprivation on ad lib caloric intake in individuals without an eating disorder and ad lib caloric intake and binge eating among eating disordered participants were medium. There was no significant heterogeneity in effect sizes.

On the other hand, experiments that assessed the effects of caloric deprivation on changes in affect and eating behavior over time in the natural environment paint a different picture (see Table 9). Several experiments found that assignment to long-term low-calorie diets resulted in reduced negative affect (Epstein, Paluch, Saelens, Ernst, & Wilfley, 2001; Nauta, Hospers, Kok, & Jansen, 2000; Wadden, Foster, & Letizia, 1994), although null findings have occurred (Yanovski & Sebring, 1994). Moreover, a randomized experiment found that assignment to a long-term low-calorie diet, relative to a waitlist condition, resulted in decreased bulimic symptoms for nonobese individuals (Presnell & Stice, in press). Other studies have also found that assignment to a long-term low-calorie diet resulted in decreases in binge eating for obese adults who did not have an eating disorder (Epstein et al., 2001; Telch & Agras, 1993; Wadden et al., 1994). However, the effect was not observed in one study with obese adults (Yanovski & Sebring, 1994) and another study with obese children (Epstein et al., 2001). An experiment also found that assignment to a long-term low-calorie diet, relative to a waitlist control condition, resulted in decreased binge eating for obese individuals with binge eating disorder (Goodrick, Poston, Kimball, Reeves, & Foreyt, 1998). Other studies have similarly found that assignment to a long-term low-calorie diet resulted in decreased binge eating for obese individuals with binge eating disorder (Nauta et al., 2000; Telch & Agras, 1993; Yanovski & Sebring, 1994). Participants in these studies showed significant decreases in body mass in response to the low-calorie diets, verifying that participants achieved lasting caloric deprivation (i.e., were dieting). Thus, caloric deprivation showed negative relations to change in negative affect and eating pathology, and maintenance of eating pathology over time. These effects were medium in size.

There was significant heterogeneity in the effect sizes for the relation of assignment to a low-calorie diet and eating pathology,

[10] Although another experiment that examined the effects of semistarvation on male conscientious objectors found increased binge eating following weight-loss down to 75% of medically healthy weight (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950), the generalizability of these findings to naturalistic dieting as practiced by women is highly suspect. First, the vast majority of female dieters never approach this degree of emaciation. Second, this study was uncontrolled, as all participants were assigned to the restrictive diet.

Table 9
*Effects of Experimentally Manipulated Dieting on Subsequent Caloric Intake and Binge Eating*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Change in negative affect over time | | | | | | | |
| Epstein et al. (2001; parents) | 24 mo | Adult | Yes | | 47 | −.18 | .038 |
| Epstein et al. (2001; children) | 24 mo | Child | Yes | | 47 | −.33 | .001 |
| Nauta et al. (2000) | 10 mo | Adult | Yes | | 37 | −.30 | .066 |
| Wadden et al. (1994) | 12 mo | Adult | Yes | | 49 | −.32 | .001 |
| Yanovski & Sebring (1994) | 3 mo | Adult | Yes | | 19 | −.08 | .423 |
| Average | | | | | | −.27* | |
| Laboratory-measured caloric intake | | | | | | | |
| Hetherington et al. (2000) | | Adult | Yes | | 21 | .19 | .189 |
| Lowe (1992) | | Adult | Yes | | 37 | .29 | .070 |
| Lowe (1994) | | Adult | Yes | | 48 | .03 | .834 |
| Lowe et al. (2001) | | Adult | Yes | | 42 | .03 | .840 |
| Schachter et al. (1968) | | Adult | Yes | | 91 | .18 | .078 |
| Spiegel et al. (1989) | | Adult | Yes | | 37 | .33 | .001 |
| Telch & Agras (1996b) | | Adult | Yes | | 26 | .61 | .001 |
| Wardle & Beales (1988) | | Adult | Yes | | 26 | .44 | .001 |
| Average | | | | | | .29* | |
| Change in eating pathology over time | | | | | | | |
| Epstein et al. (2001; parents) | 24 mo | Adult | Yes | BED | 47 | −.59 | .001 |
| Epstein et al. (2001; children) | 24 mo | Child | Yes | Comp | 47 | .06 | .681 |
| Presnell & Stice (2001) | 3 mo | Adult | Yes | Comp | 55 | −.29 | .001 |
| Telch & Agras (1993) | 15 mo | Adult | Yes | BED | 71 | −.33 | .001 |
| Wadden et al. (1994) | 12 mo | Adult | Yes | BED | 49 | −.44 | .001 |
| Yanovski & Sebring (1994) | 3 mo | Adult | Yes | BED | 8 | .13 | .347 |
| Average | | | | | | −.31* | |
| Maintenance of laboratory-measured caloric intake | | | | | | | |
| Agras & Telch (1998) | | Adult | Yes | | 60 | .29 | .001 |
| Hetherington et al. (2000) | | Adult | Yes | | 28 | .40 | .017 |
| Telch & Agras (1996b) | | Adult | Yes | | 52 | .32 | .001 |
| Average | | | | | | .33* | |
| Maintenance of laboratory-measured binge eating | | | | | | | |
| Agras & Telch (1998) | | Adult | Yes | BED | 60 | .26 | .001 |
| Telch & Agras (1996b) | | Adult | No | BED | 52 | .25 | .001 |
| Average | | | | | | .25* | |
| Maintenance of binge eating over time | | | | | | | |
| Goodrick et al. (1998) | 18 mo | Adult | Yes | BED | 219 | −.26 | .001 |
| Nauta et al. (2000) | 10 mo | Adult | Yes | BED | 16 | −.27 | .001 |
| Telch & Agras (1993) | 15 mo | Adult | Yes | BED | 20 | −.57 | .001 |
| Yanovski & Sebring (1994) | 3 mo | Adult | Yes | BED | 11 | −.85 | .001 |
| Average | | | | | | −.38* | |

*Note.* ED = eating disorder; mo = month; BED = binge eating disorder; Comp = symptom composite.
*$\alpha < .008$.

$Q(5) = 11.24$, $p < .05$. Effects were significantly larger ($z = 2.75$, $p < .006$) for studies examining adults (mean $r = -.38$) versus children (mean $r = .06$; see Table 9). This finding again suggests that children may be too young to show adequate variation in eating pathology to permit tests of etiologic processes. Whether participants were obese, length of follow-up, use of validated measures, and type of outcome examined did not predict effect sizes.

Thus, self-reported dieting predicted increased eating pathology over time, and experimentally manipulated caloric deprivation predicted increased lab-assessed caloric intake and binge eating but decreased eating pathology over time in the natural environment. There are several possible explanations for these anomalous findings. The first is that dieting exerts different effects for overweight individuals than it does for nonoverweight individuals and that variation in the average body mass of participants across studies explains the inconsistent findings. Caloric deprivation might exert different effects on those with, versus without, excess caloric reserves. However, the magnitude of the prospective effects of self-reported dieting on increases in eating pathology was not correlated with the average body mass of study participants ($r = .01$), and the effects of short-term and long-term caloric deprivation on lab-assessed caloric intake or change in bulimic symptoms were not different for studies that examined obese versus nonobese samples. A second possibility is that people can only diet for a certain period of time before they give in and binge eat. However, obesity treatment resulted in reduced binge eating and weight loss over periods longer than a year (e.g., Goodrick et al., 1998), whereas self-labeled dieting predicted onset of binge eating and weight gain over periods as short as a few weeks (e.g., Klesges, Klem, & Bene, 1989). A third possibility is that the low-calorie diets used in obesity treatment programs are unrepre-

sentative of real-world dieting (i.e., are more effective). However, if self-reported dieting were merely a weaker version of the dieting achieved in obesity treatment, then self-reported dieting should show similar but weaker relations to change in weight and binge eating—not effects in the opposite direction. Furthermore, the evidence that self-reported dieters gain more weight over time than self-reported nondieters (French, Jeffery, & Wing, 1994; Klesges, Isbell, & Klesges, 1992; Klesges et al., 1989; Stice et al., 1999) indicates that dieters are actually consuming more calories. Thus, in one sense, the inconsistent findings do result because self-reported dieting is unrepresentative of dieting used in obesity-treatment programs but only in that self-report measures of dieting do not appear to be valid indices of dieting.

It seems more likely that the inconsistent findings are a product of the limitations inherent to certain designs. Because randomized experiments rule out third-variable explanations and prospective studies do not, perhaps the positive relation of self-reported dieting to increases in eating pathology emerged because some third variable increases the risk for both variables. It has been suggested that a tendency toward caloric overconsumption may lead to both self-reported dieting and eventual onset of binge eating and bulimic pathology (Stice et al., 1999), based on the evidence that self-reported dieters gain more weight over time than nondieters (French et al., 1994; Klesges et al., 1992). If this is the case, self-reported dieting would be a proxy risk factor for binge eating and bulimic symptoms solely because it is a marker for chronic overconsumption. One would hope that future prospective research will directly test whether a propensity toward overconsumption is a risk factor for onset of dieting and binge eating. Ideally, such a propensity would be operationalized through unobtrusive observation of caloric intake to ensure that social desirability biases do not obscure the relations. Research might also test whether dieting potentiates the relation between overconsumption tendencies and risk for binge eating onset, as it has been suggested that dieting disrupts the biological processes that govern eating satiety (Blundell, 1995). Even if this is not the specific third variable that accounts for the positive relations from the prospective studies, greater interpretational weight was given to the experimental studies because this design putatively rules out possible third variables.

The fact that experimentally manipulated caloric deprivation was positively related to lab-assessed caloric intake and binge eating but negatively related to changes in caloric intake (as reflected by decreases in weight), binge eating, and bulimic pathology in the natural environment is vexing. Demand characteristics may explain the pattern of findings. In the lab studies, the researchers first placed the participants on a diet, then exposed them to tempting foods (e.g., ice cream) and indicated that they should indulge. Participants may simply have felt that they had permission to overeat these tempting foods. Moreover, the fact that participants in these long-term dieting studies consistently lose weight indicates they are not routinely overeating. The most likely explanation for the inconsistent findings is that lab measures of caloric intake and binge eating may be biased by demand characteristics. Thus, the experimental data suggest, not that dieting is causally related to binge eating and bulimic pathology, as predicted by the restraint model, but rather that effective dieting curbs overeating tendencies. Similarly, the experimental data imply that dieting results in positive rather than negative affect. This analysis has two implications. First, there does not appear to be a valid self-report measure of dieting in that the prospective studies indicated that dieting scales predicted elevated increases in body mass over time. Second, most people may not be able to successfully diet (i.e., enter a state of negative energy balance) for a prolonged period of time without therapeutic intervention. Future research needs to evaluate these implications with measurement approaches that are not subject to distortion (e.g., direct measures of weight change to verify dieting status).

In sum, although prospective studies suggest that self-reported dieting is a risk factor for negative affect and bulimic pathology as well as a maintenance factor for bulimic pathology, results from experiments that manipulated caloric deprivation appear incompatible with this conclusion. On the basis of the strengths of the various designs, I conclude that dieting is not a risk factor for eating pathology but rather attenuates overeating tendencies.

### *Negative Affect*

According to the affect regulation model (Hawkins & Clement, 1984; McCarthy, 1990), people binge eat in an effort to provide comfort and distraction from adverse emotions. Individuals might also use radical compensatory behaviors, such as vomiting, to reduce anxiety about impending weight gain consequent to overeating or because they believe that purging serves as an emotional catharsis. It has been proposed further that acute negative affect might adversely affect body satisfaction (Williamson, 1990).

As summarized in Table 10, negative affect predicted increases in eating disorder symptoms (Wertheim et al., 2001; Wichstrom, 2000) and bulimic symptoms (Cooley & Toray, 2001a; Field et al., 1999; Killen et al., 1996; Stice, 2001; Stice & Agras, 1998), although this effect was only marginally significant in one study (Vohs, Voelz, et al., 2001). Other studies have found nonsignificant relations of negative affect to increases in bulimic symptoms (Vohs et al., 1999) and eating pathology (Gardner et al., 2000; Keel et al., 1997; Leon et al., 1999) or binge eating onset (Vogeltanz-Holm et al., 2000). Negative affect was a marginally significant predictor of bulimic symptom maintenance in one study (Fairburn et al., in press), but this effect was nonsignificant in another (Stice & Agras, 1998). The effects for negative affect on increases in eating pathology and maintenance of bulimic pathology were small.[11]

There was significant heterogeneity in the effect sizes for the relation of negative affect to eating pathology, $Q(11) = 25.62, p < .01$. Effects were significantly larger ($z = 3.38$, $p < .001$) for studies predicting binge eating or bulimic symptoms (mean $r = .10$) versus general eating pathology (mean $r = .07$; see Table 10), suggesting that affective disturbances might be specific to binge eating problems. Age of participants, use of unvalidated measures, and length of follow-up were not related to the magnitude of effects.

Experiments also examined the acute effects of negative affect (see Table 11). Negative affect inductions (primarily inducing depressive feelings), versus positive or neutral mood inductions,

[11] Sensitivity analyses indicated that the magnitude of the average effect for negative affect on increases in eating pathology and the corresponding test of significance was identical when (a) the one missing effect was excluded (Keel et al., 1997), (b) a value of .00 was inserted, or (c) the mean effect size was inserted.

Table 10
*Relation of Initial Negative Affect to Subsequent Change in Eating Pathology*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in eating pathology | | | | | | | |
| Cooley & Toray (2001a) | 36 mo | Adult | Yes | BUL | 117 | .18 | .049 |
| Field et al. (1999) | 12 mo | Child | No | BUL | 6928 | .10 | .001 |
| Gardner et al. (2000) | 12 mo | Child | Yes | Comp | 86 | .11 | .301 |
| Keel et al. (1997) | 12 mo | Child | Yes | Comp | 80 | — | — |
| Killen et al. (1996) | 36 mo | Adol | Yes | BUL | 825 | .11 | .010 |
| Leon et al. (1999) | 36 mo | Adol | Yes | Comp | 736 | .05 | .179 |
| Stice (2001) | 20 mo | Adol | Yes | BUL | 231 | .18 | .006 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .25 | .001 |
| Vogeltanz-Holm et al. (2000) | 60 mo | Adult | Yes | BED | 709 | .03 | .243 |
| Vohs et al. (1999) | 9 mo | Adult | No | BUL | 342 | .01 | .476 |
| Vohs, Voelz, et al. (2001) | 1 mo | Adult | Yes | BUL | 70 | .16 | .095 |
| Wertheim et al. (2001) | 8 mo | Adol | Yes | Comp | 432 | .21 | .001 |
| Wichstrom (2000) | 24 mo | Adol | No | Comp | 7751 | .07 | .001 |
| Average | | | | | | .09* | |
| Maintenance of eating pathology | | | | | | | |
| Fairburn et al. (in press) | 60 mo | Adult | Yes | BUL | 102 | .21 | .075 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .09 | .234 |
| Average | | | | | | .13* | |

*Note.* A dash indicates a missing effect size. ED = eating disorder; mo = month; BUL = bulimic pathology; Adol = adolescent; Comp = eating disorder symptom composite; BED = binge eating disorder.
*$\alpha < .025$.

produced acute increases in body dissatisfaction (Stice, Little, & Foerster, 2001; Taylor & Cooper, 1992), but this effect was nonsignificant in a third study (Baker, Williamson, & Sylve, 1995). Negative affect inductions, relative to neutral mood inductions, did not result in changes in ad lib caloric intake among individuals who did not have an eating disorder in several studies (Abramson & Wunderlich, 1972; Baucom & Aiken, 1981; Frost, Goolkasian, Ely, & Blanchard, 1982; Heatherton, Herman, & Polivy, 1991; Herman & Polivy, 1975; Herman, Polivy, Lank, & Heatherton, 1987; Lowe & Maycock, 1988; McKenna, 1972; Polivy, Herman, & McFarlane, 1994; Reznick & Balch, 1977; Ruderman, 1985; Schachter, Goldman, & Gordon, 1968; Steere & Cooper, 1993; Telch & Agras, 1996a). However, two studies found that negative versus neutral mood inductions resulted in increased caloric intake among individuals who did not have an eating disorder (Cools, Schotte, & McNally, 1992; Schotte, Cools, & McNally, 1990). Negative mood inductions did not result in increased caloric intake among individuals with binge eating disorder, suggesting that negative affect is not a maintenance factor for caloric intake (Agras & Telch, 1998; Telch & Agras, 1996a). However, Agras and Telch (1998) found that a negative versus a neutral mood induction resulted in elevated investigator-coded binge eating. The effects for negative affect inductions on body dissatisfaction and maintenance of binge eating were medium, the effect on caloric intake was small and nonsignificant, and the effect on maintenance of caloric intake (among individuals with an eating disorder) was trivial and nonsignificant. When interpreting these findings, it is important to note that negative mood inductions may not be representative of more chronic affective disturbances that occur in the real world.

There was significant heterogeneity in the effect sizes for the relation of negative mood inductions on caloric intake, $Q(15) = 33.81$, $p < .001$. Effects were significantly larger ($z = 4.63$, $p < .001$) for studies that allowed participants to eat during (mean $r = .31$) rather than after (mean $r = -.01$) the negative affect induction (see Table 11). Effects were also significantly larger ($z = 3.33$, $p < .001$) for studies that manipulated general negative affect (mean $r = .18$) versus anxiety (mean $r = -.02$). There was no variability in the developmental age of participants, length of follow-up, use of validated measures, or outcome measure.[12]

Collectively, findings indicate that negative affect is a risk factor for eating pathology and a causal risk factor for body dissatisfaction and caloric intake, as well as a causal maintenance factor for binge eating (but not caloric intake) among individuals with an eating disorder. It was noteworthy that the effects tended to be most potent for general negative affect, rather than anxiety. There was also evidence that this negative affect results in the greatest caloric intake if individuals have access to food during the negative mood induction.

### *Perfectionism*

Theorists have proposed that perfectionism is a risk factor for eating pathology, as this personality trait may promote a relentless pursuit of the thin ideal (Bruch, 1973). In addition, perfectionism has been implicated as a maintenance factor for bulimic pathology (Fairburn, 1997), as it supposedly fosters the rigid dieting that perpetuates the binge–purge cycle.

[12] Although there is evidence that negative mood inductions resulted in greater caloric intake among dieters than among nondieters (e.g., Baucom & Aiken, 1981), dieting was not manipulated in these studies.

Table 11
*Effects of Experimentally Manipulated Negative Affect on Subsequent Caloric Intake and Binge Eating and Putative Mediators of This Effect*

| Outcome | Methodological features | | | | | |
|---|---|---|---|---|---|---|
| | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in body dissatisfaction | | | | | | |
| Baker et al. (1995) | Adult | Yes | | 72 | .13 | .136 |
| Stice, Little, & Foerster (2001) | Adult | Yes | | 56 | .26 | .022 |
| Taylor & Cooper (1992) | Adult | Yes | | 73 | .27 | .020 |
| Average | | | | | .22* | |
| Increase in laboratory-measured caloric intake | | | | | | |
| Abramson & Wunderlich (1972) | Adult | Yes | | 66 | −.08 | .674 |
| Baucom & Aiken (1981) | Adult | Yes | | 56 | .10 | .435 |
| Cools et al. (1992) | Adult | Yes | | 91 | .51 | .001 |
| Frost et al. (1982) | Adult | Yes | | 55 | .12 | .484 |
| Heatherton et al. (1991) | Adult | Yes | | 75 | −.04 | .725 |
| Herman & Polivy (1975) | Adult | Yes | | 42 | −.05 | .744 |
| Herman et al. (1987) | Adult | Yes | | 80 | .02 | .464 |
| Lowe & Maycock (1988) | Adult | Yes | | 60 | .09 | .495 |
| McKenna (1972) | Adult | Yes | | 80 | .00 | .501 |
| Polivy et al. (1994) | Adult | Yes | | 96 | .17 | .094 |
| Reznick & Balch (1977) | Adult | Yes | | 64 | .05 | .680 |
| Ruderman (1985) | Adult | Yes | | 105 | .01 | .978 |
| Schachter et al. (1968) | Adult | Yes | | 91 | −.16 | .131 |
| Schotte et al. (1990) | Adult | Yes | | 60 | .32 | .012 |
| Steere & Cooper (1993) | Adult | Yes | | 48 | −.20 | .070 |
| Telch & Agras (1996a) | Adult | Yes | | 30 | −.04 | .850 |
| Average | | | | | .07 | |
| Maintenance in laboratory-measured caloric intake | | | | | | |
| Agras & Telch (1998) | Adult | Yes | | 60 | .12 | .358 |
| Telch & Agras (1996a) | Adult | Yes | | 30 | −.13 | .488 |
| Average | | | | | −.01 | |
| Maintenance of binge eating | | | | | | |
| Agras & Telch (1998) | Adult | Yes | BED | 60 | .26 | .045 |

*Note.* ED = eating disorder; BED = binge eating disorder.
*$\alpha < .012$.

As revealed in Table 12, one study found that perfectionism predicted bulimic pathology onset (Killen et al., 1994), although others did not find significant effects for onset of bulimic pathology (Killen et al., 1996) or for increases in bulimic symptoms (Vohs et al., 1999; Vohs, Voelz, 2001) or eating pathology (Leon et al., 1999). Santonastaso et al. (1999) found that perfectionism predicted maintenance of eating pathology. The effect of perfectionism on increases in eating pathology was small but significant, whereas the effect on eating pathology maintenance was medium (Table 12). There was no significant heterogeneity in effects sizes.

Collectively, findings provide support for the assertions that perfectionism is a risk factor for bulimic pathology and a mainte-

Table 12
*Relation of Initial Perfectionism to Subsequent Change in Eating Pathology*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in eating pathology | | | | | | | |
| Killen et al. (1994) | 24 mo | Adol | Yes | BUL | 887 | .09 | .010 |
| Killen et al. (1996) | 36 mo | Adol | Yes | BUL | 825 | .06 | .100 |
| Leon et al. (1999) | 36 mo | Adol | Yes | Comp | 736 | .04 | .268 |
| Vohs et al. (1999) | 9 mo | Adult | Yes | BUL | 342 | .02 | .372 |
| Vohs, Voelz, et al. (2001) | 1 mo | Adult | Yes | BUL | 70 | .03 | .245 |
| Average | | | | | | .06* | |
| Maintenance of eating pathology | | | | | | | |
| Santonastaso et al. (1999) | 12 mo | Adol | No | Comp | 72 | .22 | .030 |

*Note.* ED = eating disorder; mo = month; Adol = adolescent; BUL = bulimic pathology; Comp = symptom composite.
*$\alpha < .025$.

Table 13
*Relation of Early Menarche to Subsequent Change in Eating Pathology and Change in the Putative Mediators of This Effect*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in body dissatisfaction | | | | | | | |
| Stice & Whitenton (in press) | 12 mo | Adol | Yes | | 484 | −.03 | .501 |
| Increase in dieting | | | | | | | |
| Cooley & Toray (2001a) | 36 mo | Adult | Yes | | 117 | .03 | .718 |
| Increase in negative affect | | | | | | | |
| Hayward et al. (1997) | 72 mo | Adol | Yes | | 758 | .07 | .055 |
| Increase in eating pathology | | | | | | | |
| Cooley & Toray (2001a) | 36 mo | Adult | Yes | BUL | 117 | −.01 | .961 |
| Graber et al. (1994) | 96 mo | Adol | Yes | Comp | 85 | .13 | .237 |
| Hayward et al. (1997) | 72 mo | Adol | Yes | Comp | 758 | .11 | .003 |
| Smolak et al. (1993) | 24 mo | Child | Yes | Comp | 79 | .24 | .108 |
| Wichstrom (2000) | 24 mo | Adol | No | Comp | 7,751 | .03 | .019 |
| Average | | | | | | .04* | |

*Note.* ED = eating disorder; mo = month; Adol = adolescent; BUL = bulimic pathology; Comp = symptom composite.
*$\alpha < .012$.

nance factor for eating pathology. It is also noteworthy that there is emerging evidence that perfectionism may interact with other risk factors to predict eating disturbances (see below).

### *Early Menarche*

It has been suggested that early pubertal development fosters body image and eating disturbances (Rierdan & Koff, 1991). Because early menarche leads to increased adipose tissue, it moves girls away from the normative body shape of adolescents (i.e., developmental deviance) and the current thin ideal, which theoretically increases body dissatisfaction and consequent dieting and eating disturbances.

As shown in Table 13, early menarche did not predict increases in body dissatisfaction (Stice & Whitenton, in press) or dieting (Cooley & Toray, 2001a) and was only marginally related to increases in negative affect (Hayward et al., 1997). Two studies found that early menarche predicted increases in eating disorder symptoms (Hayward et al., 1997; Wichstrom, 2000), but others found null effects for increases in bulimic symptoms (Cooley & Toray, 2001a) and eating disorder symptoms (Graber et al., 1994; Smolak, Levine, & Gralen, 1993).[13] The effects of early menarche on increases in body dissatisfaction, dieting, and negative affect were small to trivial and nonsignificant, and the effect for eating pathology was trivial but significant (Table 13). There was no significant heterogeneity in the effect sizes.

Together, the findings provide little support for the claim that early menarche is a risk factor for body dissatisfaction, dieting, negative affect, and eating pathology. One possible explanation for the null findings is that the adverse effects of early menarche are developmentally localized and only occur during the period in which youth are developmentally deviant (i.e., during early adolescence). This interpretation accords with the observation that the effects of pubertal timing on psychological functioning dissipate by late adolescence (Tobin-Richards, Boxer, Kavrell, & Petersen, 1984). The possibility of developmentally localized effects of early menarche implies that it would be useful to examine the effects of early menarche in short-term longitudinal studies focusing on early adolescence. Nonetheless, there is evidence that the adverse effects of early puberty on body image and eating disturbances may be amplified when early development co-occurs with other life stressors (see below).

### *Impulsivity*

Theorists have suggested that a general deficit in impulse control is a risk factor for eating pathology (Hawkins & Clement, 1984), as deficits in impulse control are thought to leave individuals vulnerable to episodes of uncontrollable binge eating. However (see Table 14), impulsivity did not predict increases in bulimic symptoms (Stice, 2001; Stice & Agras, 1998; Stice, Presnell, & Bearman, 2001) or eating pathology (Leon et al., 1999).[14] The average effect size for impulsivity was small but significant (Table 14). There was no significant heterogeneity in the effect sizes. There is also some indirect evidence for this factor. As shown in Table 15, substance use predicted an increased risk for onset of bulimic pathology (Killen et al., 1996; Stice, Presnell, & Bearman, 2001; Vogeltanz-Holm et al., 2000), although this effect was only marginally significant in one study (Stice & Agras, 1998) and nonsignificant in another (Leon et al., 1999). There was no significant heterogeneity in effect sizes. The average effect size for substance use was also small but significant. Because substance use is at least partially rooted in impulse-control deficits (Sher & Trull, 1994), these findings might be interpreted as providing indirect evidence that impulsivity is a risk factor for the development of eating pathology. Deficits in inhibitory control that are

[13] Several studies have investigated the effects of stage of pubertal development rather than timing of pubertal development. However, the former studies were not reviewed because they confound the effects of menarche timing and the age of participants, with the precise mixture of these two effects depending on the age range of the sample.

[14] It should be noted that the effects for impulsivity and substance use from Leon et al. (1999), Stice (2001), Stice and Agras (1998), and Stice, Presnell, and Bearman (2001) were from post hoc analyses not reported in the original articles.

Table 14
*Relation of Impulsivity to Subsequent Change in Eating Pathology*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in eating pathology | | | | | | | |
| Leon et al. (1999) | 36 mo | Adol | Yes | Comp | 736 | .05 | .156 |
| Stice (2001) | 20 mo | Adol | Yes | BUL | 231 | .08 | .206 |
| Stice & Agras (1998) | 9 mo | Adol | Yes | BUL | 218 | .06 | .368 |
| Stice, Presnell, & Bearman (2001) | 12 mo | Adol | Yes | BUL | 484 | .06 | .185 |
| Average | | | | | | .07 | .034 |

*Note.* ED = eating disorder; mo = month; Adol = adolescent; Comp = symptom composite; BUL = bulimic pathology.

putatively due to functional and structural abnormalities in a cortical-basoganglionic neural network (Barkley, 1997) may increase the risk for overconsumption of both food and psychoactive substances. Alternatively, there might be other shared risk factors for these outcomes (i.e., that substance use is a proxy risk factor for eating pathology). Perhaps a hypersensitivity of the mesolimbic dopamine reward system places individuals at risk for any behavior that activates this reinforcement system. This explanation accords with the evidence that individuals with bulimia nervosa show an increased family history of substance use disorders (Bulik, 1987; Fairburn, Welch, Doll, Davies, & O'Connor, 1997; Garfinkel et al., 1995). A third possibility is that substance use may disinhibit people's efforts at dietary restraint and lead to binge eating (Polivy & Herman, 1985). Additional research is needed to more fully understand these relations.

### *Non-Established Risk Factors*

It is also import to note that certain widely accepted risk factors for eating pathology have not yet been supported in prospective or experimental studies. Theorists have suggested that childhood sexual abuse is a risk factor for eating pathology (Connors, 2001). However, childhood sexual abuse did not emerge as a significant predictor of binge eating onset in the only prospective study that investigated this factor (Vogeltanz-Holm et al., 2000). Thus, it seems premature to conclude that sexual abuse is a risk factor for eating pathology. Similarly, there has been speculation about the role of stress, control issues, dysfunctional family systems, and deficits in parental affection in the genesis of eating pathology (e.g., Minuchin, Rosman, & Baker, 1978). However, no empirical support emerged in the prospective studies that tested these assertions (Attie & Brooks-Gunn, 1989; Ball & Lee, 2002; Graber et al., 1994; Vogeltanz-Holm et al., 2000). Unfortunately, many believe these are empirically established risk factors for eating pathology despite the null findings from available prospective studies. Nevertheless, it is possible that these putative risk factors will be supported in future prospective and experimental studies. Within this context, it is acknowledged that some of these factors will be more challenging to study with these types of designs (but see below). Moreover, even though these risk factors may not show univariate relations to increases in eating pathology, it is still possible that they interact with other risk factors to promote eating disturbances. Finally, because these putative risk factors are so distal in time from the period during which eating pathology emerges, they may exert their effects on more proximal factors (e.g., negative affect) and may only show weak relations to increases in eating pathology.

## Summary of Risk and Maintenance Factor Findings

Findings suggest that elevated body mass is a risk factor for perceived pressure to be thin, body dissatisfaction, and dieting, and that modeling of body image and eating disturbances are risk factors for bulimic pathology. Body dissatisfaction is a risk factor for dieting, negative affect, and eating pathology. Negative affect, perfectionism, impulsivity, and substance use are risk factors for eating pathology. Perceived pressure to be thin and thin-ideal internalization can be considered causal risk factors for body dissatisfaction, dieting, negative affect, and eating pathology because of the experimental findings. Negative affect can also be

Table 15
*Relation of Substance Use to Subsequent Change in Eating Pathology*

| Outcome | Methodological features | | | | | | |
|---|---|---|---|---|---|---|---|
| | Follow-up | Age | Validated scale | ED outcome | Sample size | Effect size (*r*) | *p* |
| Increase in eating pathology | | | | | | | |
| Killen et al. (1996) | 36 mo | Adol | Yes | BUL | 825 | .11 | .001 |
| Leon et al., 1999 | 36 mo | Adol | Yes | Comp | 736 | .00 | .991 |
| Stice & Agras, 1998 | 9 mo | Adol | Yes | BUL | 218 | .13 | .059 |
| Stice, Presnell, & Bearman (2001) | 12 mo | Adol | Yes | BUL | 484 | .09 | .049 |
| Vogeltanz-Holm et al. (2000) | 60 mo | Adult | Yes | BED | 709 | .09 | .007 |
| Average | | | | | | .07 | .001 |

*Note.* ED = eating disorder; mo = month; Adol = adolescent; BUL = bulimic pathology; Comp = symptom composite; BED = binge eating disorder.

considered a causal risk factor for body dissatisfaction and caloric intake. There was evidence that thin-ideal internalization, body dissatisfaction, and perfectionism are maintenance factors for bulimic pathology and that negative affect is a causal maintenance factor for binge eating. Prospective and experimental findings for dieting were at odds, prompting a conclusion that dieting is not a risk factor for eating pathology but rather attenuates overeating tendencies. Finally, there was some indication that perceived pressure to be thin, thin-ideal internalization, and body dissatisfaction are potentiating factors that amplify the effects of other risk factors and that social support is a protective factor that mitigates the effects of other risk factors.

There are several noteworthy features of the findings. Most important is that the average univariate effects for the risk factors were generally small. The methodological limitations of these studies likely attenuated the magnitude of the effects (e.g., unreliability of measurement). This account is consistent with the current meta-analytic findings indicating that the effects were larger in the more methodologically rigorous studies. The generally small effects may also imply that no single factor can account for a large proportion of the variance in change in eating pathology. If so, multivariate models should have greater explanatory power. In addition, there may be other important risk factors for eating pathology that remain to be discovered. These factors likely include biological processes. For instance, it has been suggested that elevated serotonin levels may be a risk factor for onset of dieting, which results in decreased serotonin levels because it reduces the precursors to this neurotransmitter (tryptophan; Kaye et al., 1998). This dietary restriction in turn putatively increases the risk that the individual will binge eat to restore serotonin levels. Although the maintenance factors tended to show somewhat larger effects, it is possible that biological processes serve to perpetuate eating disorder symptoms once they are initiated. For instance, Kaye et al. (1998) suggested that starvation induces neuropeptide abnormalities that promote further obsessions with food and weight in a downward spiral.

Meta-analytic results also indicate that the effects were larger for studies predicting change in binge eating and bulimic symptoms versus general eating pathology (no studies predicted increases in anorexic pathology). This may imply that the risk factors are somewhat different for the specific eating disorders and that predicting change in an overall eating disorder symptom composite obscures these differences and attenuates the effects of the risk factors. Future research should examine this possibility.

A final noteworthy trend was that findings from randomized experiments tended to be larger than those from prospective studies. Presumably this is because experiments afford greater control of extraneous variables, which results in large effect sizes.

## Multivariate Risk Factor Models

Numerous multivariate models have been proposed that attempt to explain how risk factors work together to promote or maintain eating pathology. Some models are interactive in nature in that they posit that a particular confluence of risk factors places individuals at increased risk for eating pathology, whereas others propose a mediational sequence that links risk factors developmentally over time. In this section, I review the few multivariate models that have been prospectively tested.

Greenberg and Harvey (1986) posited that the confluence of dieting and depression gives rise to binge eating. This assertion accords with the suggestion from restraint theory that negative affect disrupts the cognitive controls over eating that characterize dieting (Polivy & Herman, 1985). However, the only study to prospectively test this model indicated that there was no significant interaction between dieting and negative affect ($r = .05$, $p = .723$) in the prediction of binge eating onset over a 9-month period in adolescent girls (Stice, Akutagawa, Gaggar, & Agras, 2000).

Theorists have also proposed a cumulative stressor model of body image and eating disturbances (Smolak et al., 1993). It has been hypothesized that the co-occurrence of certain stressors during adolescence—puberty-related weight gain, dating onset, and intensified academic demands—interact to precipitate dieting and eating disturbances. A prospective test of aspects of this model revealed that girls who experienced onset of early puberty and dating during the same year showed greater increases in eating pathology ($r = .26$, $p < .01$) than girls who did not experience these events simultaneously (Smolak et al., 1993). Similarly, early menarche shows a stronger relation to subsequent negative affect when it co-occurs with other life stressors such as school transitions ($r = .14$, $p < .05$; Blyth, Simmons, & Zakin, 1985). That there was converging support for this model from two independent studies suggests that this model is promising and should be investigated further.

Vohs and colleagues (1999) proposed that the confluence of perfectionism, body dissatisfaction, and low self-esteem promotes bulimic pathology. The combination of body dissatisfaction and perfectionism theoretically results in effective weight control behaviors for individuals with high self-esteem because such individuals are likely to view being overweight as a temporary, changeable situation. In contrast, perfectionistic individuals with low self-esteem putatively respond to being overweight with less effective coping skills. The Perfectionism $\times$ Body Dissatisfaction $\times$ Low Self-Esteem interaction predicted increases in bulimic symptoms over a 5-week period ($r = .17$, $p = .048$; Vohs, Voelz, et al., 2001) and over a 9-month period among college women ($r = .08$, $p = .028$; Vohs et al., 1999). Although the effect sizes were relatively small for a multivariate model, it is encouraging that the effects replicated in two studies. Furthermore, these may have been conservative tests of this model because they were conducted with young adults who are generally past the period of greatest risk for onset of bulimic pathology.

The dual pathway model posits that sociocultural pressure to be thin fosters body dissatisfaction, as repeated messages that one is not thin enough likely promotes discontent with one's body, and that internalization of the thin ideal contributes to body dissatisfaction because this ideal is difficult to attain (Stice, Nemeroff, & Shaw, 1996). Body dissatisfaction in turn is thought to promote dieting and negative affect, which consequently increase the risk for bulimic pathology. Body dissatisfaction putatively leads to dieting because of the belief that this is an effective weight control technique and contributes to negative affect as appearance is a central evaluative dimension for women. Dieting is thought to increase the risk for bulimic pathology because people may binge eat to counteract the effects of caloric deprivation or because violation of strict dietary rules results in disinhibited eating. Negative affect may foster bulimic symptoms because it is commonly believed that eating provides comfort and distraction from nega-

tive emotions. Perceived pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and negative affect predicted bulimic symptom onset in three independent studies of adolescent girls (Stice & Agras, 1998; Stice, Killen, et al., 1998; Stice et al., 2002). Moreover, a technique for prospectively testing all linkages in a mediational model evaluated this account with data from the third study (Stice, 2001). As expected, pressure to be thin ($r = .14$, $p = .043$) and thin-ideal internalization ($r = .25$, $p = .001$) predicted increases in body dissatisfaction, body dissatisfaction predicted increases in dieting ($r = .35$, $p = .001$) and negative affect ($r = .21$, $p = .001$), and dieting ($r = .36$, $p = .001$) and negative affect ($r = .18$, $p = .006$) predicted increases in bulimic symptoms. This model collectively accounted for 23% of the variance ($r = .48$) in growth of bulimic pathology.

Only one eating pathology maintenance model has apparently been tested with prospective data: Fairburn's (1997) cognitive model of bulimia nervosa. Fairburn posited that appearance overvaluation is of primary importance in maintaining binge eating, the core feature of bulimia nervosa. Theoretically, individuals who consider their body shape one of the most important aspects of self-evaluation and who believe that achieving thinness will bring social and psychological benefits engage in severe dieting. This dieting is based on absolutist and inflexible thinking and is thought to place individuals at risk for binge eating. This binge eating in turn putatively results in redoubled dietary efforts and potentially more radical weight control behaviors such as vomiting. In this way, appearance overvaluation is thought to maintain the cycle of extreme dieting and bulimic pathology. Thus, dieting ostensibly mediates the relation between appearance overvaluation and binge eating. Fairburn et al. (in press) tested this model with prospective data from women with bulimia nervosa. As expected, appearance overvaluation showed a significant relation to change in dietary restriction ($r = .28$, $p = .007$) and a marginally significant relation to change in binge eating ($r = .19$, $p = .078$), and change in dietary restriction predicted change in binge eating ($r = .22$, $p = .049$). The relation between initial appearance overvaluation and change in binge eating became nonsignificant when the effects of change in dietary restriction were controlled ($r = .13$, $p = .241$), suggesting that the relation between appearance overvaluation and binge eating was mediated by change in dietary restriction. These factors collectively showed a moderate relation to change in binge eating ($r = .25$). Thus, results provide preliminary support for the cognitive model of bulimic symptom maintenance and imply it would be fruitful for future research to investigate this account.

## Summary of Prospective Tests of Multivariate Etiologic and Maintenance Models

Findings provided support for the cumulative stressor model, the Perfectionism × Body Dissatisfaction × Low Self-Esteem model, and the dual pathway model of the etiology of bulimic pathology, as well as for the cognitive model of bulimic symptom maintenance. However, most of these models have not been independently tested and some accounted for a modest amount of variance. Findings also provided evidence that negative affect, perfectionism, and early menarche are potentiating factors that amplify the effects of other risk factors. It was noteworthy that despite the fact that early menarche did not emerge as a significant risk factor for eating pathology in the univariate tests, it appears to interact with life stressors to predict emergence of negative affect and eating disturbances. Thus, despite the scarcity of prospective tests of etiologic and maintenance models, several promising accounts have emerged.

## Methodological Limitations of Past Research

Advances in understanding of the processes that promote and maintain eating pathology have been constrained by certain methodological limitations. This meta-analysis reveals several methodological factors that attenuated effect sizes. First, use of measures with unestablished reliability and validity was associated with smaller effect sizes. This factor likely attenuated the estimates of effect sizes. One key example is that some studies used measures of eating pathology that have not been demonstrated to assess clinically significant levels of impairment. Thus, it is not clear whether the etiologic processes are related to clinically meaningful eating pathology or more innocuous eating abnormalities.

Second, effect sizes were smaller for etiologic studies focusing on preadolescents and adults rather than adolescents. This suggests that it may not be ideal to test etiologic hypotheses with data from developmental periods that are not characterized by increases in eating pathology. Eating pathology does not emerge or increase markedly during preadolescence or adulthood (Keel et al., 1997; Rizvi, Stice, & Agras, 1999; Vohs, Heatherton, & Herrin, 2001) but rather emerges during middle adolescence (Lewinsohn et al., 2000; Stice, Killen, et al., 1998). Thus, studies should follow participants through the period of greatest risk for the emergence and growth in eating pathology (or other outcome of interest) to provide an optimal test of etiologic processes.[15]

Third, results indicate that prospective studies that followed participants for shorter periods of time generated smaller effects than those with longer follow-ups. This may signify that a longer follow-up period is necessary for sufficient numbers of participants to show change in the outcome of interest or that multiple waves of data allow one to model change more reliably (Rogosa, Brandt, & Zimowski, 1982). This limitation also likely attenuated the effect size estimates.

Fourth, effects were weaker for studies that predicted overall eating disorder symptom composites versus bulimic symptoms or binge eating. This may have resulted because these outcomes tap more objective behavioral markers rather than abstract attitudinal constructs. Alternatively, this may indicate that the risk and maintenance processes are specific to the three eating disorders and that focusing on overall eating disorder symptom composites obscures these relations and attenuates effects. This brings to light a key lacuna of the literature—no prospective study has tested whether the risk and maintenance factors differ for the three eating disorders. A related gap is that no prospective study has focused solely on anorexic pathology. This state of affairs makes it difficult to draw conclusions regarding risk and maintenance processes that are specific for each of the eating disorders. One implication is that

[15] It might be noted that high test–retest correlations for eating pathology measures during adolescence should not be interpreted as indicating that the frequency of these behaviors remains constant. Equivalent test–retest correlations can be observed when the average frequency increases, decreases, or remains stable over time (test–retest correlations reflect consistency of rank ordering across assessments).

certain risk factors might have received greater support if studies had focused on anorexic symptoms (e.g., perfectionism). Other risk factors might have received more support if eating disorder subtypes had been used as the criteria (e.g., impulsivity might predict onset of the purging subtype of bulimia nervosa).

There were other methodological limitations that were not examined as moderators of effect sizes (because studies with these limitations were excluded or because there was no variability across studies). First, the overreliance on cross-sectional designs represents a key shortcoming of the literature because it is not possible to differentiate risk factors from concomitants or consequences of eating pathology with cross-sectional data. Greater use should be made of prospective and experimental studies. It is particularly important to use experiments to test etiologic and maintenance hypotheses because prospective studies are vulnerable to third-variable explanations. Randomized prevention and treatment trials can be used to provide experimental tests of etiologic and maintenance factors, respectively.

The use of mono-method and mono-reporter data is another limitation. Increased use of collateral reports, observational data, and biological measures could advance our understanding of the etiology and maintenance processes. For instance, it was long accepted, on the basis of self-report data, that obese individuals did not consume more calories than nonobese individuals. However, this finding was refuted with the advent of unobtrusive measures of caloric intake. Studies with doubly labeled water, a measure of energy expenditure, indicate that obese individuals underreport caloric intake (Bandini, Schoeller, Dyr, & Dietz, 1990; Prentice et al., 1986). For example, Prentice and associates (1986) found that 33% of obese individuals underreported caloric intake compared with 2% of lean individuals.

Finally, there is some concern about the discriminant validity of the various risk and maintenance factors. Although no study has examined all of the factors considered here, some conducted confirmatory factor analysis of sets of these risk factors and provided evidence that the factors represent distinct constructs (Stice et al., 1996; Thompson, Coovert, Richards, Johnson, & Cattarin, 1995). For example, the average correlation between a set of these factors was .33 (Stice et al., 1996). Nonetheless, it is important that future studies more clearly establish the discriminant validity of these risk and maintenance factors.

## Statistical Limitations of Past Research

There were also a number of statistical limitations of past studies. First, approximately half of the prospective studies analyzed their data in a way that did not establish temporal precedence. It is necessary for longitudinal analyses to control for initial levels of the outcome so that analyses are predicting change in eating pathology.

Second, many studies have not taken advantage of powerful analytic techniques for modeling longitudinal data. Standard multiple regression or logistic regression models are one of the least powerful techniques for modeling change. Analytic techniques such as random regression growth curve models and hazard models offer more sensitive approaches to modeling change (Rogosa et al., 1982; Willett & Singer, 1993).

Third, most etiologic models have not been evaluated in rigorous studies that provide tests of the hypothesized mediational or moderational relations. There are many proponents of structural equation modeling, yet most applications are cross-sectional and therefore do not permit inferences regarding the nature of the observed correlations. There is nothing causal about this type of statistical technique (even with prospective data), as inferential power is a function of design rather than analysis. Furthermore, using indicators that share a single method or reporter incorporates method and reporter biases into the latent construct (i.e., they should not be considered error free). Future research should use more powerful approaches that permit prospective tests of each link in mediational models (e.g., sequential growth curve models). Also, regression and classification tree analysis can provide a more sensitive test of moderational models.

Fourth, the meaning behind the correlations among risk and maintenance factors should be handled thoughtfully. Risk factors that do not show unique effects in multivariate models are sometimes dismissed, even though these factors show univariate effects. However, such correlations can occur for vastly different reasons. Risk factors might be correlated because they assess the same construct or share reporter biases, but they may also be correlated because they are related to the outcome in a complex mediational chain or co-occur in a cumulative fashion.

Fifth, greater attention should be paid to the magnitude of effects. Correlations, the percentage of variance explained, or some other relevant index of effect size should be routinely reported and considered when interpreting findings.

## Theoretical Limitations of the Literature

There are also theoretical limitations of this literature. First, more attention should be directed at identifying new risk factors because most of the established risk factors have modest effect sizes. This search should include a focus on potentiating and mitigating factors that qualify the main effects of risk factors. Promising variables include hypersensitivity to negative interpersonal transactions, cognitive factors, (e.g., affect regulation expectancies), feeding avidity, and individual differences in reinforcement from eating. Increased research should also be directed at biological risk factors (e.g., serotonin abnormalities, functional aberrations in the mesolimbic dopamine pathway, and structural differences in the orbitofrontal cortex). It will be crucial to use prospective studies to test whether biological factors predict eating pathology because virtually all biological factors that have been examined normalize with treatment, suggesting that these disturbances are a consequence rather than a cause of eating pathology (Kaye et al., 1998). Although such prospective studies are more challenging, they are necessary to establish the biological factors that increase risk for eating pathology. Greater use should also be made of experimental designs (e.g., one could test the diet-induced serotonin deficit model of binge eating by experimentally manipulating tryptophan). The current paradigm used to study biological factors (comparing individuals with an eating disorder with those who have recovered) rests on the assumption that eating disorders have no long-term neurological effects—an assumption that has not been verified. Prospective and experimental studies have been used to examine biological risk factors for other psychiatric disorders with much success (Hill, Steinhauer, Lowers, & Locke, 1995; Rogers et al., 1999).

Second, more theoretical attention should focus on factors that maintain eating pathology because etiologic and maintenance processes may differ. Theoretical attention should also be directed at the protective and potentiating factors that moderate the effects of maintenance factors. The distinction between risk and maintenance factors is key because an understanding of maintenance processes is necessary to develop optimal treatments. Biological factors hold promise in explaining symptom maintenance, although natural history studies that test whether these factors predict persistence of eating pathology in untreated community samples of individuals with eating disorders is necessary.

Greater theoretical attention should also focus on elucidating how risk and maintenance factors work together to promote and perpetuate eating pathology. That even the most comprehensive model accounted for only a portion of the variance in eating pathology suggests that more complex explanatory models are needed. The predictive power of these models might be enhanced by incorporating more of the established risk and maintenance factors, and by including mediational and moderational components in these models. It is likely that there are multiple pathways to the development of any psychopathology, which would reflect moderated mediation. There has also been little theoretical work on how psychosocial and biological factors, including genetic risk, might work together to foster eating disturbances.

## Prevention and Treatment Implications

Prevention programs should focus on reducing malleable risk factors for eating pathology, such as thin-ideal internalization, body dissatisfaction, and negative affect, as well as on decreasing factors that potentiate the effects of other risk factors, such as pressure to be thin and perfectionism. They should also strive to increase protective factors, such as social support and self-esteem. Although some of these risk factors have been targeted in past prevention efforts, few interventions focused on multiple risk and potentiating factors, and fewer still attempted to increase protective factors. There would also be benefits to targeting more general risk factors that have been shown to predict multiple adverse outcomes (e.g., social support has been found to decrease risk for depression and substance abuse as well [Monroe, Imhoff, Wise, & Harris, 1983; Windle, 1992]) because such interventions should produce greater overall improvements in mental health.

As the risk factors were able to predict emergence of eating pathology, they could also be used to identify high-risk groups for selective prevention programs. This appears to be an important application given the trend for prevention programs to produce the greatest benefits for those with initial elevations in pathology (e.g., Killen et al., 1993). Selective programs should consider using a composite of several risk factors for screening purposes, as this approach should identify a higher risk population than a single screening scale would identify.

Treatment implications are limited by the scarcity of prospective and experimental studies on maintenance factors. Nonetheless, findings suggest that treatment of bulimia nervosa and binge eating disorder should focus on reducing thin-ideal internalization, body dissatisfaction, and negative affect. Whereas some of these areas are targeted in cognitive behavior therapy (Fairburn, 1997), the efficacy of this intervention might be enhanced by expressly focusing on all of these constructs. Perfectionism emerged as a maintenance factor for general eating pathology and therefore might be usefully targeted in eating disorder treatments as well.

It might be useful to profile patients regarding the specific maintenance factors on which they show elevations. This would permit individualized interventions that are tailored to reduce the particular factors that serve to maintain their eating disturbance. My hope is that such an approach would improve the rate of treatment success. Only about 30% of individuals receiving the treatment of choice show a complete cessation of bulimic behaviors for at least a 1-month period (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000).

## Directions for Future Research

Several directions for future research are suggested by this review. First, investigators should make greater use of prospective and experimental designs if advances in understanding of etiologic and maintenance processes are to be made. Results from this meta-analysis suggest that these studies should use validated measures, developmentally appropriate samples, and at least three assessments to maximize the reliability with which change is measured. Studies should also use multiple methods of data collection, multiple-reporter data, and analytic techniques that model change with greater sensitivity.

Second, more prospective research is needed on the factors that maintain eating pathology, as there are only a few preliminary studies. This information is vital for the design of effective treatments. It would be ideal for these longitudinal studies to use community, rather than clinical, samples because the latter are unrepresentative (they overrepresent psychiatric comorbidity—Berkson's bias—and the chronicity and severity of disorders—clinicians' illusion).

Third, prospective and experimental studies concerning the mechanisms by which factors work together to promote and maintain eating pathology are needed. The promising multivariate models that have been proposed should be tested with rigorous designs that permit the demonstration of temporal precedence for each link in these accounts. Studies should also focus more energy on how psychosocial, biological, and genetic factors work together.

Fourth, more research should be directed at elucidating the risk and maintenance factors for the three specific eating disorders—particularly anorexia nervosa. It is my hope that these studies will span the full spectrum of eating pathology, ranging from symptom onset to the emergence of full syndrome disorders.

I offer the following approach to empirically investigating risk and maintenance factors for eating pathology in the hope that it might promote more rigorous programmatic research. The first step is to establish that there is a correlation between the putative risk or maintenance factor and eating pathology because costly longitudinal or experimental studies should not be conducted needlessly. The second step is to demonstrate that the putative risk or maintenance factor prospectively predicts onset or persistence of eating pathology respectively. The third step is to explore how the risk or maintenance factor works together with other factors to promote or perpetuate eating pathology (mediation and moderational relations). Again, a moderator does not have to show a significant univariate relation to the outcome to qualify the direction and/or strength of the relation between another risk or maintenance factor and the outcome. The fourth step is to conduct a

randomized laboratory experiment that assesses these relations with tight experimental control. The fifth step is to conduct an ecological experiment that reduces the risk or maintenance factor and assesses whether there is a consequent change in the outcome over time (with randomized prevention or treatment trials). This set of studies would more definitely establish the nature of the relation between risk and maintenance factors and the development and perpetuation of eating pathology.

## Conclusion

A number of conclusions emerged from this meta-analytic review of the risk and maintenance factors for eating pathology. First, several widely accepted risk factors for eating pathology have not received empirical support, such as sexual abuse and stress, or have very conflicting research support, such as dieting. Second, there is surprisingly consistent support for other risk factors that are not as well accepted, including thin-ideal internalization and negative affect. Third, although prospective studies are a vast improvement over cross-sectional designs, randomized experiments are necessary to permit greater confidence in inferences regarding etiologic and maintenance processes. Finally, the predictive power of individual risk and maintenance factors was limited, suggesting it will be important to search for additional risk and maintenance factors, develop more comprehensive multivariate models, and address methodological limitations that attenuate effects.

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