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The Acquired Preparedness Model of Risk for Bulimic Symptom Development

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Abstract

The authors applied person-environment transaction theory to test the acquired preparedness model of eating disorder risk. The model holds that (a) middle school girls high in the trait of ineffectiveness are differentially prepared to acquire high risk expectancies for reinforcement from dieting/thinness; (b) those expectancies predict subsequent binge eating and purging; and (c) the influence of the disposition of ineffectiveness on binge eating and purging is mediated by dieting/thinness expectancies. In a three-wave longitudinal study of 394 middle school girls, they found support for the model. Seventh grade girls' scores on ineffectiveness predicted their subsequent endorsement of high risk dieting/thinness expectancies, which in turn predicted subsequent increases in binge eating and purging. Statistical tests of mediation supported the hypothesis that the prospective relation between ineffectiveness and binge eating was mediated by dieting/thinness expectancies, as was the prospective relation between ineffectiveness and purging. This application of a basic science theory to eating disorder risk appears fruitful, and the findings suggest the importance of early interventions that address both disposition and learning.

The risk process for eating disorders can be understood as consisting of both dispositional characteristics of individuals and of learning processes that are specific to eating and dieting. Our intent with this paper is to test a model, adapted from the basic science literature on person-environment transaction theory (Caspi, 1993), describing a transactional process between broad disposition and specific learning that influences eating disorder risk. In person-environment transaction theory, learning is not just a function of experience; rather, one's dispositional characteristics help shape one's learning experiences, and one's learning in turn

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influences one's behavior. As a result, the influence of disposition on behavior is mediated by psychosocial learning. We applied this model to eating disorder risk, and tested one developmental implication of it: that individual differences in disposition at the start of middle school predicted subsequent high-risk learning, which then predicted increased eating disorder symptomatology.

The dispositional characteristic we studied was ineffectiveness, which has been associated with eating disorder risk and which can be understood to refer to feelings of general inadequacy and ineffectiveness, insecurity, worthlessness, emptiness and lack of control over one's life (Garner, 1991). Items representing the ineffectiveness construct include such statements as "I wish I were someone else" and "I feel ineffective as a person." To represent learning processes related to risk for eating disorder, we relied on eating disorder expectancy theory. Essentially, eating disorder expectancy theory holds that individuals vary in their learned expectancies for reinforcement from eating and from dieting/thinness, and that individual differences in expectancies influence eating disorder risk (Hohlstein, Smith, & Atlas, 1998). Here we focus on the set of expectancies that "dieting and thinness lead to overgeneralized life improvement."

We introduce this study by first reviewing evidence that ineffectiveness and dieting/thinness expectancies each appear to operate as risk factors for eating disorders. We then summarize person-environment transaction theory and specify our application of this theory to eating disorder risk. We then report a test of the following core hypothesis from this theory. Among middle school girls, ineffectiveness predicts subsequent increases in expectancies for overgeneralized life improvement from dieting/thinness; the expectancies, in turn, predict increases in binge eating and purging behavior; and the predictive relationship between ineffectiveness and symptoms is mediated by dieting/thinness expectancies.

Ineffectiveness

There are several pieces of evidence that ineffectiveness plays a role in the eating disorder risk process. Among college women, ineffectiveness predicted subsequent increases in bulimic and restraint behaviors (Cooley & Toray, 2001), and increases in ineffectiveness across the first year of college were associated with maintenance of and increases in eating disordered behaviors (Striegel-Moore, Silberstein, French, & Rodin, 1989). Ineffectiveness also differentiated compulsive from non-compulsive eaters (Attie & Brooks-Gunn, 1989). Among adolescents, ineffectiveness, measured as part of a negative affect/attitudes composite, predicted later disordered eating (Leon, Fulkerson, Keel, & Klump, 1999) and ineffectiveness predicted the subsequent development of partial eating disorder syndromes (Killen et al., 1996).

The content domain of the trait is not specific to eating disorders; it likely involves a general, dispositional risk that is sometimes manifested in eating disorder symptoms. Consistent with this possibility, ineffectiveness is elevated in never-ill relatives of eating-disordered patients as compared to never-ill relatives of controls (Lilenfeld et al., 2000), and scores on the trait remain elevated in women who have recovered from bulimia nervosa and anorexia nervosa (Stein et al., 2002; Casper, 1990; Kaye, et al., 1998).

Eating Disorder Expectancy Theory

Eating disorder expectancy theory is an application of classic expectancy theory as first articulated by Tolman (1932) and as elaborated by Bolles (1972), MacCorquodale and Meehl (1953), Rotter (1954), and others. The basic science literature has identified expectancies as learned anticipations of the likely consequences of behavioral choices. Expectancies are understood to represent summaries of individuals' learning histories; they are formed based on the multitude of direct and vicarious learning experiences that individuals undergo. The

expectancies one forms then influence one's future behavioral choices: one tends to choose behaviors from which one expects rewards and avoid behaviors for which one expects punishment. Application of this perspective has led to useful examinations of many psychological phenomena, including psychopathology (Alloy & Tabachnick, 1984), affect (Carver & Scheier, 1990), gambling behavior (Walters & Contri, 1998), risk for alcohol abuse (Smith, Goldman, Greenbaum, & Christiansen, 1995), and risk for smoking (Brandon & Baker, 1991).

Expectancy theory has been applied to eating disorders: individuals form different eating and dieting/thinness expectancies from one another, in part because they are exposed to different learning experiences concerning eating, dieting, and thinness. To the extent that one comes to associate eating with more powerful reinforcers than do others, one will then hold unusually strong expectancies for reinforcement from eating. One therefore will pursue food with greater vigor. To the extent that one comes to associate thinness with powerful, perhaps overgeneralized, reinforcers, one will then hold strong expectancies for reinforcement from thinness, and thus will be more concerned with dieting and with thinness than will others.

Dieting/thinness expectancies, which are the focus of this paper, differentiate among anorexia nervosa, bulimia nervosa, and control patients (Hohlstein et al., 1998), they correlate cross-sectionally with both binge eating and purging behavior in both adolescent and adult samples (Hohlstein et al., 1998; MacBrayer, Smith, McCarthy, Demos, & Simmons, 2001; Simmons, Smith, & Hill, 2002), and they predict the subsequent onset of those symptoms in middle school girls (Smith, Simmons, Flory, Annus, & Hill, 2007; Stice & Whitenton, 2002). Individuals who most strongly expect dieting/thinness to lead to overall life improvement are those at greatest risk. Manipulations of dieting/thinness expectancies produce reductions in some eating disorder symptoms (Annus, Smith, & Masters, 2008) and disrupt the relationship between expectancies and other risk factors (Fister & Smith, 2004). It appears that dieting/thinness expectancies play a role in the risk process.¹

Although numerous studies have documented linear relations between dieting/thinness expectancies and symptom reports (Hohlstein et al., 1998; Simmons et al., 2002; Smith et al., 2007; Stice & Whitenton, 2002), we offer an alternative theory, which is that the relation between expectancies and symptoms likely consists of a combination of linear and quadratic trends. Concerns about weight and shape appear to be normative (Polivy and Herman, 1987), and to some degree expectations for reinforcement from dieting/thinness are likely to be accurate. Therefore, some of the variation in dieting/thinness expectancy endorsement

¹The precise mechanism by which dieting/thinness expectancies influence binge eating and purging behavior has neither been fully articulated nor tested. It is quite possible that strong endorsement of dieting/thinness expectancies contribute to purging behavior fairly directly, in the sense that purging may reflect an effort to pursue thinness (and perhaps a correction for a dieting failure). Concerning binge eating, the expectation that dieting and thinness would be beneficial does not automatically lead to successful caloric restriction: attempts to restrict food intake often fail, perhaps sometimes resulting in excessive consumption. Each such eating event provides reinforcement (the negative reinforcement of hunger alleviation and the direct, positive reinforcement from eating). Girls with strong dieting/thinness expectancies are inclined to attempt to restrict, and hence likely to have frequent experiences of failures in their restricting. Each of those experiences provides reinforcement, both positive and negative, often for excessive eating. Perhaps this process, when combined with other risk factors not studied here (such as expectancies that eating helps alleviate one's negative affect, Hohlstein et al., 1998, and the possibility that binge eating distracts one from one's distress, Heatherton & Baumeister, 1991), increases the probability of repeated episodes of binge eating.

It is important to appreciate that the prospective prediction from dieting/thinness expectancies to binge eating behavior does not constitute evidence for the theory that weight-loss dieting/caloric deprivation causes subsequent binge eating (Polivy & Herman, 1987). To date, no study has tested whether dieting/thinness expectancy endorsement predicts reduced caloric intake; given the existing cross-sectional evidence, it is perhaps more likely that endorsement of the expectancies is associated with a concern for dieting and weight-related issues in the absence of actual caloric deprivation (Hohlstein et al., 1998). We do not hold that dieting/thinness expectancy endorsement predicts weight-loss dieting and caloric deprivation. The theory that weight-loss dieting causes binge eating has been undermined by evidence that (a) scales predictive of subsequent binge eating that were once thought to measure caloric restriction do not measure such restriction (Stice, Fisher, & Lowe, 2004); (b) experimentally induced dieting produces reductions in bulimic symptomatology (Stice, Presnall, Groesz, & Shaw, 2005); and (c) among women with bulimia nervosa, there is an inverse relationship between dieting frequency and binge eating frequency (Lowe, Gleaves, & Murphy-Eberenz, 1998).

probably has little relation to eating disorder symptomatology. Specifically, we believe variation in moderate or low levels of expectancy endorsement is minimally related to symptom reports: for example, whether one is neutral or slightly disagrees with expectancies for improved social skills from thinness is unlikely to relate to binge eating or purging. But there is an increasingly strong relationship between expectancy endorsement and symptoms at above-average expectancy levels: girls who agree strongly with that expectancy are at greater risk than girls who agree only slightly. Thus, those with high symptom levels are likely characterized by unusually strong expectancy endorsement.

We thus expect the relation between expectancies and symptoms to reflect a combination of linear and quadratic trends, such that the slope of the relationship is modest at moderate levels of expectancy endorsement and increases at high levels of expectancy endorsement. We therefore included a quadratic dieting/thinness expectancy variable in our model test.

Integration of Dispositional and Learned Risk Factors: the Acquired Preparedness Model of Eating Disorder Risk

Person-environment transaction theory specifies several ways in which disposition transacts with the environment to influence learning (Caspi, 1993; Caspi & Roberts, 2001; Moffitt, 2005; Shiner & Caspi, 2003; Smith, Williams, Cyders, & Kelley, 2006; Widiger & Smith, 2008). Our focus in this paper is on reactive person-environment transactions: as a function of individual differences in traits, individuals react differently to the same experience. Thus, an objectively common learning event may not be experienced in the same way by two individuals (Hartup & Van Lieshout, 1995). It follows that two individuals may learn different things from the same event.

We applied this perspective to eating disorder risk as follows. Disposition, represented here by the trait of ineffectiveness, influences the learning process, and in that way increases eating disorder risk. Girls high in ineffectiveness (i.e., who feel ineffective and inadequate) are more likely than others to respond to learning events that suggest means to both (a) obtain reinforcers one feels ineffective at obtaining and (b) improve one's feeling of adequacy. It appears to be the case that Western cultural societal messages include the view that one means to obtain life reinforcers and feel adequate, at least for women, involves dieting and thinness (Stice, 2001). In this cultural context girls high in ineffectiveness are perhaps differentially prepared to inculcate that message and thereby form expectancies for overgeneralized life improvement from dieting and thinness. In contrast, girls low in ineffectiveness tend to be less prepared to acquire these high risk expectancies.

We refer to this process with the term acquired preparedness (AP), to denote the theory that girls are differentially prepared to acquire high risk expectancies as a function of stable trait differences. The AP model of risk holds that disposition influences risk indirectly, through its influence on psychosocial learning. Evidence supporting an AP process in domains other than eating disorders has accrued from basic science research in which traits predicted differential learning to the same event (Smith et al., 2006) and from field research on other addictive behaviors, such as alcoholism, in which both cross-sectional (Anderson, Smith, & Fischer, 2003; Barnow et al., 2004; McCarthy, Kroll, & Smith, 2001a; McCarthy, Miller, Smith, & Smith, 2001b; Meier, Slutske, Arndt, & Cadoret, 2007; Trembach, Belyaev, & Lysenko, 2004) and longitudinal studies (Settles, Cyders, & Smith, in press; Fu, Ko, Wu, Cherng, & Cheng, 2007) provided evidence consistent with the theory that traits' influence on problem drinking is mediated by alcohol expectancies (Smith & Anderson, 2001).²

A Prospective Test of the AP Model of Eating Disorder Risk

We tested this proposed integration of disposition and learning in a sample of middle school girls, using a longitudinal design that spanned three years: the two years of middle school and the first year of high school (the school system used a two-year middle school structure). We used the sample first described by Smith et al. (2007), who demonstrated that dieting/thinness expectancies differentiated among girls developing along different eating disorder symptom trajectories and predicted symptom onset over the longitudinal period.

To determine whether development during middle school is consistent with the possibility that ineffectiveness shapes expectancy formation, which in turn influences symptom development, we tested whether ineffectiveness, measured at the start of middle school, predicted subsequent increases in dieting/thinness expectancies, which in turn predicted subsequent increases in binge eating and purging behavior. We further tested whether the predictive relationship between ineffectiveness at the start of middle school (year one) and symptom reports the first year of high school (year three) appeared to be mediated by expectancies measured at year two.

Method

Participants

The participants in this study were 394 middle school girls assessed at three different points in time. They were assessed in the fall of their first year of middle school, which was 7th grade (Time 1), and again in the fall of their 8th grade and 9th grade years. At Time 2, 343 participants remained in the study (87%) and at Time 3, we successfully tested 283 participants (83% of the second year sample, for an overall retention rate of 72%). As described in Smith et al. (2007), missing data appear to have been missing at random, so we used the maximum likelihood estimation procedure available in MPlus (Muthen & Muthen, 2004); this procedure provides relatively unbiased estimates of parameters and standard errors (Schafer & Graham, 2002), enabling us to report results from the full $n = 394$ sample. The mean age of the participants at the initiation of the study was 12.84 years. Most were Caucasian (78.7%), followed by African American (10.5%); the remainder of the sample identified themselves as Asian, Hispanic, American Indian, Arab or East Indian. The socioeconomic makeup of the sample was diverse, with 26% of the reported family incomes less than \$25,000, 50% of the reported family incomes between \$25,000 and \$50,000, and the remaining 24% of reported family incomes more than \$50,000.

Measures

Demographic and Background Questionnaire—This measure provided the assessment of the demographic information reported above. It also asked for self-reported height and weight. Self-reports of height and weight correlate very highly with objective measurements and are reasonably accurate, except that self-reported weight tends to be slightly lower than measured weight (Smith, Hohlstein, & Atlas, 1992). We used these self-reports to calculate body mass index (BMI), in order to investigate whether the effects we observed could be attributed to individual differences in BMI.

²The AP model of risk is consistent with the view that eating disorders constitute addictive behaviors, because the model describes a process by which girls learn to attribute intense, overgeneralized reinforcement to dieting and thinness. As described in this model and elsewhere, this learning increases the risk that girls will become psychologically dependent on the habitual practices of binge eating and purging (Combs & Smith, 2009; Hohlstein et al., 1998). The theory that learned expectancies are risk factors for addictive behaviors has been supported for numerous other addictive behaviors, including alcoholism (Smith et al., 1995), gambling (Walters & Contri, 1998), caffeine addiction (Heinz, Kassel, & Smith, 2009), and smoking (Brandon & Baker, 1991).

Eating Disorder Inventory II, Ineffectiveness Scale (EDI-II; Garner, 1991)—This scale on the EDI-II represents an assessment of feelings of general inadequacy, insecurity, worthlessness, emptiness and lack of control over one's life (Garner, 1991). Items are rated on a 6-point Likert scale ranging from Never to Always. There is considerable evidence for the reliability and validity of this scale (Garner, 1991). As noted above, it predicts subsequent eating disorder symptoms (Cooley & Toray, 2001; Leon et al., 1999; Killen et al., 1996).

Thinness and Restricting Expectancy Inventory (TREI; Hohlstein et al., 1998)—This single scale measure reflects hypergeneralized expectancies about the life benefits of dieting and thinness. Items include such statements as “I would feel less stressed, in general, if I were thin.” The scale has been shown to be unidimensional, internally consistent, correlated with eating disorder symptoms in adolescent and adult samples, and predictive of eating disorder symptom onset (Hohlstein et al., 1998; MacBrayer et al., 2001; Simmons et al., 2002; Smith et al., 2007).

Bulimia Test-Revised (BULIT-R: Thelen, Farmer, Wonderlich, & Smith, 1991)—In two independent studies, the BULIT-R had sensitivity, specificity, and negative predictive values over .90, and positive predictive values over .70 with respect to DSM-IV criteria as diagnosed by interview (Thelen, Mintz, & Vanderl Wal, 1996; Welch, Thompson, & Hall, 1993). It has a four-factor structure, including factors that primarily assessed binge eating and purging among adolescents (Vincent, McCabe, & Ricciardelli, 1999). Smith et al. (2007) slightly modified the scales based on these two factors to ensure that each measured the target symptoms without contamination from other content domains. Ten items measuring binge-eating include “I would presently rate myself a compulsive eater (one who engages in episodes of uncontrolled eating)”; internal consistency for the scale was $\alpha = .88$. Eleven items reflecting purging behavior include such questions as “How often do you intentionally vomit after eating?” Internal consistency for the purging scale was $\alpha = .83$. Responses to items were coded on a 5-point Likert scale; higher scores indicated a higher frequency of the measured behavior. For example, responses to the vomiting frequency question ranged from “less than once a month or never” to “two or more times a week.”

Procedure

Data Collection—Students were tested in the fall, annually, over a one to two day period either in their regular classrooms or in one central location.

Data Analytic Method—The aim of this investigation was to test a sequence of predictive relationships: time 1 ineffectiveness predicts changes in dieting/thinness expectancies at time 2, and time 2 expectancies predict changes in binge eating and purging behaviors at time 3. To complete the test of our hypothesis, we also sought to test whether time 1 ineffectiveness' prediction of time 3 binge eating and purging appeared to be mediated by time 2 expectancies. We therefore adopted a structural equation modeling (SEM) approach. We constructed two primary structural models, each including four variables measured at each of three time points: ineffectiveness, linear dieting/thinness expectancies, quadratic dieting/thinness expectancies, and either binge eating or purging.

We represented ineffectiveness, linear expectancies, and quadratic expectancies as latent variables. We did so because, for each variable, we understand the indicators of the variable to be expressions of a common, underlying construct. Using latent variable theory (Bollen, 1991; Borsboom, Mellenbergh, & Van Heerden, 2003), we view variability in indicator responses as effects of variability in the underlying construct. In contrast, we represented symptom reports as measured variables, calculated as the sum of the binge eating and purging items, respectively. We took this approach because we do not believe, for example, that each

purging item necessarily represents an effect of a common, underlying construct. Instead, we view each indicator, i.e., each report of purging behavior, as a cause of an overall purging score.³

We modeled each latent variable using three parcels (or groups) of items as manifest indicators. Doing so provided both practical and measurement advantages. The practical advantage stemmed from the fact that we had a large number of items measuring each construct: 44 items reflected dieting/thinness expectancies and 10 items reflected ineffectiveness. Our sample of 394 did not provide sufficient degrees of freedom to model covariances among so many items; the use of parcels was necessary to test our model. The measurement advantages of this approach were as follows. First, the reliability of a parcel of items is greater than that of a single item, so parcels can serve as more stable indicators of a latent construct. Second, as combinations of items, parcels provide more scale points, thereby more closely approximating continuous measurement of the latent construct. Third, there is reduced risk of spuriously positive correlations, both because fewer correlations are being estimated and because each estimate is based on more stable indicators. These advantages have been described by Little, Cunningham, Shahar, and Widaman (2002) and partly by Rushton, Brainerd, & Pressley (1983). The crucial relevant caution about using parcels is that they could mask multidimensionality in an item set (Hagtvet & Nasser, 2004; Little et al., 2002). For each of the latent variables we studied, factor analyses on both independent samples (Garner, 1991; Hohlstein et al., 1998) and the current sample confirmed their unidimensionality, mitigating this concern.

To measure quadratic expectancies, we followed the approach recommended by Marsh, Wen, and Hau (2004) for constructing product terms in SEM models of latent variables. Specifically, we first centered all linear dieting/thinness expectancy parcel scores. We then squared each centered parcel, and each of the squared values became indicators of the quadratic expectancy variable. Thus, the indicators of linear expectancies were centered parcels 1, 2, and 3: the indicators of quadratic expectancies were centered parcel 1 squared, centered parcel 2 squared, and centered parcel 3 squared. We centered the parcels to remove nonessential collinearity between the linear and quadratic variables (Cohen, Cohen, West, & Aiken, 2003; Marsh et al., 2004), our squares of the parcels reflect Marsh et al.'s (2004) recommended matched pairs approach, and we included the quadratic term only together with the linear term, so the quadratic term was corrected for its overlap with the linear effect (Cohen et al., 2003).

We measured SEM model fit using four common indices: the Comparative Fit Index (CFI), the Nonnormed Fit Index (NNFI), the root mean square error of approximation (RMSEA), and the standardized root mean square residual (SRMR). Guidelines for these indices vary: CFI and NNFI values of .90 or .95 are described as representing good fit (Hu & Bentler, 1999; Kline, 2005). RMSEA values of .06 are thought to indicate a close fit, .08 a fair fit, and .10 a marginal fit (Browne & Cudeck, 1993; Hu & Bentler, 1999), and SRMR values of approximately .09 tend to indicate good fit (Hu & Bentler, 1999). Overall evaluation of model fit is made by considering the values of each of the four fit indices; models that fit well on most indices are generally considered well-fitting. We used MPlus (Muthen & Muthen, 2004) to run the analyses.

We tested a series of model comparisons, separately for binge eating and purging. For both types of symptoms, the first model (model A) was a simple, autoregressive model: we allowed only prospective prediction from each variable to the same variable one year later. The second model (model B) added in prediction of subsequent symptoms by prior ineffectiveness scores.

³We felt our treatment of binge eating and purging scores as measured variables was the conservative approach. We also ran each model treating binge eating and purging as latent variables, and the same effects emerged.

The third model (model C) tested our hypothesized model: in addition to the paths in model B, we specified that time 1 ineffectiveness predicted time 2 quadratic expectancies, and time 2 quadratic expectancies predicted increases in time 3 symptoms (either binge eating or purging). The test of model C included a test of the indirect path from time 1 ineffectiveness, through time 2 quadratic expectancies, to time 3 symptoms. We also conducted follow-up tests, as described below. For each of these models, all time 1 latent and manifest variables were allowed to correlate, as were cross-construct, within-wave residuals.⁴

Results

Participant Attrition Analysis, Treatment of Missing Data, and Descriptive Information

Smith et al. (2007) described attrition analyses for this sample. Briefly, they noted that retention rates were acceptable, given the rate at which students moved out of the school system. Study dropouts were more likely to live with only one parent, but the two groups did not differ on binge eating, purging, or any expectancy score. We found that the two groups' scores on ineffectiveness also did not differ. Because the two groups did not differ on any variable of interest to the study, we proceeded on the assumption that scores were missing at random. We therefore used the maximum likelihood estimation procedure in MPlus, which enabled us to make use of all available data. This method has been shown to produce relatively unbiased population parameter estimates and to be superior to alternative methods, such as deleting cases with missing data, conducting mean substitutions for missing values, or using other single imputation methods (Schafer & Graham, 2002).

As noted by Smith et al. (2007), 92 girls (23.35%) reported having engaged in binge eating behavior and 71 (18.02%) reported having engaged in purging behavior during the course of the study. They found that some girls reported increases and others reported decreases in their symptomatic behavior during these years, with the result that there is little mean change at the group level. Table 1 provides descriptive statistics for ineffectiveness, dieting/thinness expectancies, binge eating, and purging for each of the three years.

Nature of Quadratic Relationship between Dieting/Thinness Expectancy Endorsement and Symptom Endorsement

We first tested our hypothesis that the nature of the relationship between dieting/thinness expectancy endorsement and symptom level was characterized by a combination of linear and quadratic curves, such that variation in low and moderate levels of expectancy endorsement would have little relationship to either binge eating or purging, but variation in above average levels of expectancy endorsement would be strongly related to those symptom reports. Figure 1 depicts curves summarizing the relationship between expectancy scores and binge eating scores, and between expectancy scores and purging scores. The figure includes a Lowess line and the accompanying scatterplot for each relationship. The Lowess line maps the smoothed, specific relationship in this data set without imposing any prior, hypothesized structure to the relationship (Cohen et al., 2003). Visually, the lines appear to be consistent with our hypotheses.

In preliminary multiple regression analyses, we found that inclusion of the quadratic dieting/thinness expectancy term (a) significantly improved prospective prediction of binge eating both from year 1 to year 2 and from year 2 to year 3, and (b) significantly improved prospective prediction of purging both from year 1 to year 2 and from year 2 to year 3. Inclusion of the quadratic term increased the explained variance in this set of analyses by a median amount of

⁴An alternative approach would have been to include all longitudinal paths and simply report those that were significant. We re-ran the analyses using that approach, and found that the same pathways were significant and the model fit improved slightly.

5%. We therefore expected the quadratic component of dieting/thinness expectancies to predict subsequent increases in binge eating and purging.⁵

Ineffectiveness, Dieting/Thinness Expectancies, and Binge Eating

As the top half of Table 2 shows, each of the models we tested with respect to binge eating fit the data fairly well. Model comparisons indicated that model B fit significantly better than did model A, and that model C, the hypothesized model, fit significantly better than did model B. Figure 2 depicts model C. Despite the stability in the individual differences reported for each construct, as reflected in the good fit for the autoregressive model (model A), addition of the AP sequence of additional prospective predictions significantly improved the fit of the model to the data. Year 1 ineffectiveness predicted year 2 quadratic dieting/thinness expectancies ($b = .31, p < .01$), and year 2 quadratic expectancies predicted increased binge eating behavior at year 3 ($b = .10, p < .05$). The test of the indirect pathway, from year 1 ineffectiveness through year 2 quadratic expectancies to year 3 binge eating, which is the statistical representation of the AP mediation hypothesis, was significant ($z = 2.24, p < .05$). Table 3 presents the correlations among the variables in the model.

We followed these model comparisons with three forms of follow-up tests. First, we tested whether the indirect pathway remained significant, even when we added in prospective prediction of year 2 quadratic expectancies from all other predictors in the model (year 1 linear expectancies, year 1 binge eating) and prediction of year 3 binge eating from all other predictors (year 2 ineffectiveness, year 2 linear expectancies). It did, and inclusion of these additional pathways did not improve model fit significantly.

Second, we explored the nature of the quadratic effect by conducting a form of piecewise prediction. We divided the sample into two groups: girls whose time 2 expectancy scores were at the mean or below, and girls whose time 2 expectancy scores were above the mean. Then for both groups, we tested a model that included autoregressions for ineffectiveness, dieting/thinness expectancies, and binge eating, along with the AP indirect effect from year 1 ineffectiveness through year 2 expectancies to year 3 binge eating. We did so to test the hypothesis that dieting/thinness expectancies predict increases in binge eating for those who endorse the expectancies at an above average level, but do not for those who do not. Thus, we modeled linear expectancies for those at the mean or below, and then we modeled linear expectancies for those above the mean. As hypothesized, for the low expectancy group, there was a nonsignificant relationship between year 1 ineffectiveness and year 2 expectancies ($b = -.11$) and a nonsignificant relationship between year 2 expectancies and year 3 binge eating ($b = -.08$). It follows that the indirect effect was nonsignificant. For the high expectancy group, year 1 ineffectiveness predicted year 2 expectancies ($b = .41, p < .01$) and year 2 expectancies predicted year 3 binge eating ($b = .14, p < .01$), and the indirect effect, representing the hypothesized mediation, was significant ($z = 2.21, p < .05$). For both groups, model fit was reasonably good (respective values for the low and high groups were: CFI = .91, .92; NNFI = .89, .90; RMSEA = .10, .09; SRMR = .07, .06). We also conducted a multigroup analysis in which we tested the hypothesis that constraining the AP pathways (year 1 ineffectiveness to year 2 expectancies, year 2 expectancies to year 3 binge eating) to be equal would significantly reduce model fit. It did (comparison $\chi^2(2) = 25.43, p < .01$).

⁵We also used multiple regression, including the expectation maximization procedure for estimating missing data, to estimate the minima values for both relationships (expectancies with both binge eating and purging). Minima values refer to the point where the curve bends. Using centered scores, minima estimations were -0.19 for the year 2 expectancy to year 3 binge eating relationship, and -0.30 for the year 2 expectancy to year 3 purging relationship. Both of those values are near the mean expectancy score. The maximum likelihood estimation procedure we used for the structural models does not provide the raw regression coefficients necessary to obtain minima estimates (Cohen et al., 2003). Although the values we obtained are consistent with visual inspection of the relevant lowest lines, and thus are likely to be reasonable estimates of the minima in this sample, they should be understood to be estimates obtained using a different modeling procedure from the maximum likelihood SEM procedure we used to present the substantive findings.

Third, because model C represents the quadratic effect but not the linear effect (in model C, the linear effect is controlled for the quadratic effect), we re-ran models A, B, and C, leaving out the quadratic expectancy latent variable, in order to test the linear effect (so this version of model C included an indirect effect from year 1 ineffectiveness through year 2 linear expectancies to year 3 binge eating). Each of these models fit the data well. For example, the fit values for this version of model C were CFI = .94, NNFI = .93, RMSEA = .08, SRMR = .06. However, the indirect effect was not present: year 1 ineffectiveness predicted year 2 linear expectancies significantly ($b = .20, p < .01$), but year 2 linear expectancies did not predict year 3 binge eating significantly ($b = .02$). Thus, modeling of only the linear component of dieting/thinness expectancies did not result in evidence for the AP mediational process.

Ineffectiveness, Dieting/Thinness Expectancies, and Purging

We tested the analogous set of models for the prediction of purging behavior. The bottom half of Table 2 presents the fit results for the models we tested. It was again the case that purging model C (the one depicting the AP indirect effect from year 1 ineffectiveness through year 2 quadratic expectancies to year 3 purging) fit better than model B (which included direct effects from ineffectiveness), which fit better than model A (the autoregressive model). In purging model C, the path from year 1 ineffectiveness to year 2 quadratic expectancies was significant ($b = .31, p < .01$), and the path from year 2 quadratic expectancies to year 3 purging was marginally significant ($b = .06, p < .06$). The test of the hypothesized indirect effect relating those three variables was also marginally significant ($z = 1.54, p < .07$). Table 3 presents the correlations among the variables in the model.

We again conducted three forms of follow-up tests. First, the marginally significant indirect effect, which represents the mediational process we hypothesized, remained marginally significant after including all other prospective predictors in the model. Including those additional predictors did not significantly improve model fit.

Second, we again explored the nature of the quadratic effect using the same piecewise model fitting. We divided the sample into the same two groups: girls whose time 2 expectancy scores were at the mean or below, and girls whose time 2 expectancy scores were above the mean. For both groups, we tested a model that included autoregressions for ineffectiveness, dieting/thinness expectancies, and purging, along with the AP indirect effect from year 1 ineffectiveness through year 2 expectancies to year 3 purging. We thus tested the hypothesis that dieting/thinness expectancies predict increases in purging behavior for those who endorse the expectancies at an above average level, but do not for those who do not. For the low expectancy group, the relationship between year 1 ineffectiveness and year 2 expectancies ($b = -.13$) was significantly lower than zero ($p < .05$), opposite to the direction that was predicted. There was a nonsignificant relationship between year 2 expectancies and year 3 purging ($b = -.03$). It follows that the indirect effect was nonsignificant. For the high expectancy group, year 1 ineffectiveness predicted year 2 expectancies ($b = .36, p < .01$) and year 2 expectancies predicted year 3 purging ($b = .10, p < .05$), and the indirect effect, representing the hypothesized mediation, was significant ($z = 1.87, p < .05$). It was again true that for both groups, model fit was reasonably good (respective values for the low and high groups were: CFI = .92, .91; NNFI = .90, .89; RMSEA = .09, .11; SRMR = .07, .06). The multigroup analysis again tested the hypothesis that constraining the AP pathways (year 1 ineffectiveness to year 2 expectancies, year 2 expectancies to year 3 purging) to be equal would significantly reduce model fit. It did for the prediction of purging, just as it did for the prediction of binge eating (comparison $\chi^2(2) = 16.96, p < .01$).

Third, we again re-ran models A, B, and C, leaving out the quadratic expectancy latent variable, in order to test the linear effect (so this version of model C included an indirect effect from year 1 ineffectiveness through year 2 linear expectancies to year 3 purging). Each of these

models fit the data well. For example, the fit values for this version of model C were CFI = .94, NNFI = .93, RMSEA = .08, SRMR = .06. However, the indirect effect was not present: year 1 ineffectiveness predicted year 2 linear expectancies significantly ($b = .18, p < .01$), but year 2 linear expectancies did not predict year 3 purging significantly ($b = -.05$). Just as was true with binge eating, modeling of only the linear component of dieting/thinness expectancies did not result in evidence for the AP mediational process.⁶

Discussion

The AP model of eating disorder risk represents an application of person-environment transaction theory to the study of eating disorders. The model holds that high scores on the trait of ineffectiveness differentially prepare girls to develop high-risk expectancies that dieting/thinness lead to overgeneralized life improvement, and that development of the expectancies increases the likelihood of early onset binge eating and purging. Thus, the influence of ineffectiveness on symptom expression is indirect, through its influence on expectancy development.

This paper describes the first test of this model. In a three-wave longitudinal study, we found that 7th grade girls' ineffectiveness scores predicted subsequent increases in their endorsement of high risk dieting/thinness expectancies, and that those expectancies in turn predicted subsequent increases in binge eating behavior. The prospective prediction of 9th grade binge eating by 7th grade ineffectiveness was indirect, i.e., mediated by 8th grade dieting/thinness expectancies. These longitudinal findings are consistent with the AP model of eating disorder risk.

One important feature of this process was that the relationship between dieting/thinness expectancy endorsement and binge eating was curvilinear. In particular, at low or average levels of expectancy endorsement, variation in expectancy scores was unrelated to variation in binge eating. But at above average levels of expectancy endorsement, expectancy scores were strongly related to subsequent binge eating. This finding is consistent with the theoretical position that variation among girls within the moderate to low levels of endorsement of the expectancy that dieting/thinness leads to overgeneralized reinforcement is not associated with variation in risk for early onset binge eating behavior. Variation among girls who endorse the expectancy at higher levels, operationalized here as endorsement at or above mean levels, is strongly associated with variation in risk.

This paper also reports the first test of the AP model with respect to purging behavior. In the full sample, prediction of increased 9th grade purging from 8th grade expectancies was only marginally significant, as was the test of whether 8th grade expectancies mediated the prediction of 9th grade purging by 7th grade ineffectiveness. However, the hypothesized indirect prediction was observed among girls who endorsed the expectancy at or above mean levels within the sample. For the girls who endorsed dieting/thinness expectancies at this higher level, individual differences in expectancy endorsement did predict subsequent individual differences in purging behavior, and results were consistent with the hypothesized process in which year 1 ineffectiveness predicted increased year 2 expectancies, which in turn predicted increased year 3 purging behavior.

It thus seems possible that ineffectiveness contributes to eating disorder risk in the following way. Perhaps girls high in ineffectiveness, i.e. who feel inadequate, insecure, and ineffective

⁶For both the binge eating and purging models, we tested whether our findings could be a function of BMI (body mass index). We re-ran each model including BMI at all three time points and allowing all cross-sectional and prospective correlations with BMI. We found that all findings were unchanged.

at meeting their needs, tend to be more receptive than other girls to messages describing steps toward greater effectiveness and adequacy. Perhaps that receptiveness in this culture leads them to be more highly prepared to learn that thinness and dieting make every area of one's life better. To the degree that they form that expectancy, their risk for eating disorder behavior increases. Perhaps girls low in ineffectiveness are at reduced risk compared to their peers, because they are less prepared to form high-risk dieting/thinness expectancies. Indeed, one way to view the present findings is that low levels of ineffectiveness and low endorsement of dieting/thinness expectancies may provide protection against the early onset of binge eating and purging behavior.

The AP risk model is one that describes causal processes: traits shape learning, which then influences behavior. In this study, we did not test, nor did we show, the operation of causal processes. Rather, we conducted a series of prospective tests that demonstrated change as predicted by the theory, in the presence of tight statistical controls. Had the prospective tests failed to support the theory, the theory would have been placed in doubt. The theory survived those tests; the results of this study are consistent with, but of course not proof of, the AP model of eating disorder risk. We cannot rule out the possibility that the prospective relationships we found were artifactual of other, unspecified causal processes.

There is some independent evidence that dieting/thinness expectancies play a causal role in eating disorder symptomatology. Annus et al. (2008) found that reduction of dieting/thinness expectancies produced a decline in body dissatisfaction and cognitive symptom endorsement, and Fister and Smith (2004) found that learning information counterfactual to dieting/thinness expectancies disrupted expectancies' relations to other risk factors. The present findings supplement that experimental evidence concerning causality. Of course, it is difficult to develop evidence that a trait operates in a causal way, because one cannot experimentally manipulate trait levels. However, the demonstration by Smith et al. (2006) that trait levels predicted subsequent, differential expectancy formation, even though all participants were exposed to precisely the same learning environment, certainly suggests a causal process. It does seem that longitudinal data collected in the field, such as that provided in this report, when combined with laboratory controlled demonstrations like Smith et al. (2006), constitutes good, albeit not definitive, evidence in the absence of the ability to conduct experiments.

At the same time, it is important to recognize that the specific mechanisms of the putative causal process has not been specified or tested. We have described a process by which high ineffectiveness could bias girls to learn to form high-risk expectancies, but the prospective correlational data we have provided does not test the validity of any one particular process or mechanism of influence. Likewise, we have offered the speculation that strong endorsement of dieting/thinness expectancies may contribute fairly directly to purging behavior, as purging may be viewed as a means to pursue thinness, or perhaps to avoid failing to achieve thinness. We have suggested that strong endorsement of the expectancy may lead to concern with dieting and attempts to restrict, followed by failures in those attempts, such that the subsequent eating and overeating is reinforcing. But we have not tested either of these speculations in this study.

It is of course very likely that these processes, if they do operate, work in conjunction with other processes not tested in this study to increase risk. For example, expectancies that eating helps alleviate negative affect predict subsequent binge eating (Smith et al., 2007); this paper does not address the possible trait influences on that expectancy, nor does it provide a test a risk process that integrates all those factors. Other processes, such as genetic liability (Klump, McGue, & Iacono, 2003), negative affect-based impulsivity (Fischer, Smith, & Cyders, 2008), perfectionism (Vohs, Bardone, Joiner, Abramson, & Heatherton, 1999), and others are likely to play a role as well.

The results of this study should be understood in the context of its limitations. First, a larger sample size would have increased power in the analyses and perhaps have contributed to making significant those findings that were marginal. We did experience relatively high attrition rates (28% of original participants dropped out by Time 3); although there is good evidence for the validity of the maximum likelihood method for addressing missing data (Schafer & Graham, 2002), less attrition might nevertheless increase confidence in these findings. Second, all risk and symptom reporting was done by questionnaire. Although there is considerable evidence for the validity of both the expectancy and behavior measures, face-to-face interviews might well have provided opportunities for more precise assessment.

The problem of eating disordered behavior is a very serious one that involves significant harm to adolescent girls. It is therefore crucial to develop risk models that integrate the contributions of different kinds of risk factors. In this report, we described such a model and found empirical support for it in a longitudinal sample of middle school girls. There is good reason to believe that dispositional risk, particularly the trait of ineffectiveness, transacts with learned expectancies to increase risk for some middle school girls.

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Figure 1.

Figure 1 depicts the relationship between binge eating at Time 3 and purging at Time 3 as predicted by dieting/thinness expectancies at Time 2. The best fit lowess line is shown, as well as the scatter plot describing the relationships. Scatterplots report raw data, so the points that appear to be in bold simply indicate that there are more cases at those specific points. For these scatterplots, dieting/thinness expectancies are not centered.

Figure 2.

Figure 2 depicts model C for binge eating. Included are relationships between ineffectiveness (IE), binge eating, linear thinness expectancies (LTE) and quadratic thinness expectancies (QTE) at Years 1 (7th grade), 2 (8th grade), and 3 (9th grade). The arrows represent the significant tested pathways; the corresponding numbers refer to the maximum likelihood coefficient for each respective relationship. The effect of quadratic thinness expectancies was modeled with the inclusion of the corresponding path from linear thinness expectancies, in order to test the quadratic effect with the linear effect controlled. The linear quadratic expectancy pathways are not included when they are non-significant. Also modeled, but excluded from the figure, were parcel loadings, time 1 correlations among all latent and measured variables, residual and disturbance terms, and cross-construct, within-time correlations of residual terms. $*p < 0.05$; $**p < 0.01$.

Figure 3.

Figure 3 depicts model C for purging. Included are relationships between ineffectiveness (IE), purging, linear thinness expectancies (LTE) and quadratic thinness expectancies (QTE) at Years 1 (7th grade), 2 (8th grade), and 3 (9th grade). The arrows represent the significant tested pathways; the corresponding numbers refer to the maximum likelihood coefficient for each respective relationship. The effect of quadratic thinness expectancies was modeled with the inclusion of the corresponding path from linear thinness expectancies, in order to test the quadratic effect with the linear effect controlled. The linear quadratic expectancy pathways are not included when they are non-significant. Also modeled, but excluded from the figure, were parcel loadings, time 1 correlations among all latent and measured variables, residual and disturbance terms, and cross-construct, within-time correlations of residual terms. ^a $p < 0.06$; * $p < 0.01$.

Means and Standard Deviations of Binge Eating, Purging, Dieting/Thinness Expectancies, and Ineffectiveness at Years 1, 2, and 3

Table 1

	Year 1		Year 2		Year 3	
	M	SD	M	SD	M	SD
Bingeing	15.122	6.681	13.788	5.284	14.759	5.853
Purging	15.950	6.078	14.886	5.198	16.764	6.336
Dieting/Thinness Expectancies	3.217	1.506	3.432	1.616	3.541	1.544
Ineffectiveness	1.087	1.330	0.925	1.288	1.227	1.524

Note: binge eating and purging means represent total scores, whereas expectancy and ineffectiveness means represent average item scores. The expectancy scores indicate that average endorsement fell near the middle (3.5) of the severity scale; ineffectiveness was coded such that the middle of the severity scale was 1.5, so mean scores fell below the middle of that scale.

Table 2

Models with SEM fit indices

Binge Eating		DF	Chi Square	Chi Square Change	CFI	NNFI	RMSEA (95% CI)	SRMR
Model A	388	1299.22 *	--		.92	.91	.08	.10
Model B	386	1275.18 *	24.04 *		.93	.92	.08	.09
Model C	384	1247.01 *	28.17 *		.93	.92	.07	.05

Purging		DF	Chi Square	Chi Square Change	CFI	NNFI	RMSEA (95% CI)	SRMR
Model A	388	1330.46 *	--		.92	.92	.08	.09
Model B	386	1308.00 *	22.46 *		.93	.92	.08	.09
Model C	384	1282.30 *	25.70 *		.93	.92	.08	.07

NOTE: CFI = comparative fit index; NNFI = non-normed fit index; RMSEA = the root mean square of approximation; CI = confidence interval; SRMR = the standardized root mean square residual

* Significant at $p < .01$

Table 3

Correlations between ineffectiveness (IE), centered linear thinness expectancies (LTE), quadratic thinness expectancies (QTE), binge eating, and purging at times 1, 2, and 3.

	IE	LTE	QTE	Binge	Purge
Time 1	IE	.38, .38	.32, .31	.54	.60
	LTE	--	.26, .26	.33	.32
	QTE		--	.21	.24
Time 2	IE	.16, .16	.23, .12	.40	.19
	LTE	--	.15, .08	.19	.14
	QTE		--	.21	.10
Time 3	IE	.29, .29	.18, .18	.22	.25
	LTE	--	-.02, -.02	.11	.17
	QTE		--	.11	.09

NOTE: For each relationship, the first correlation is the estimate from the binge eating SEM model and the second is from the purging SEM model. All correlations of .14 and higher are significant at $p < .05$.