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A Risk Model for Pre-Adolescent Disordered Eating

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Abstract

Objective—This study tested this risk model for disordered eating in pre-adolescent girls: pubertal onset is associated with increases in negative urgency (the personality tendency to act rashly when distressed); negative urgency influences eating disorder symptoms by shaping psychosocial learning (expectancy formation), thus indirectly influencing symptom levels; and many influences on purging are mediated by binge eating.

Method—905 fifth grade girls completed questionnaire measures of eating pathology, negative urgency, and dieting/thinness and eating expectancies.

Results—Binge eating and purging behaviors were present in 5th grade girls. As anticipated, pubertal status was associated with higher levels of negative urgency, negative urgency was associated with each expectancy measure, quadratic dieting/thinness and eating expectancies were associated with binge eating, and binge eating was associated with purging.

Discussion—It is important and feasible to develop risk models for pre-adolescent eating disordered behaviors. Our model that integrates puberty, personality, and psychosocial learning appears promising.

Keywords

risk; pre-adolescent; puberty; personality; learning

There is a dearth of research investigating disordered eating and its risk mechanisms in pre-adolescent girls. Research has typically focused on the important post-pubertal years, during which significant eating disorder symptoms are clearly manifested (1). This paper describes an investigation of pre-adolescent eating disordered behaviors and tests a risk model for those behaviors. We will demonstrate that a model specifying a series of predictions involving pubertal onset, personality, and psychosocial learning accounts well for 5th grade girls' binge eating and purging behaviors. To introduce this model test, we first summarize what is known about the prevalence of binge eating and purging in this young population; we then introduce our model and its basis in the empirical literature.

Pre-Adolescent Girls and Eating Disordered Behavior

We consider the onset of adolescence, at least within the United States, as occurring when individuals both go through puberty and enter middle school (where the increased autonomy, increased focus on peer relationships, and relative decline in the centrality of adults that have been defined as characteristic of adolescence occurs: 2,3). To study pre-adolescent risk, we thus sampled fifth grade girls. Some had gone through puberty, but none were yet in middle school.

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Although little is known about girls in this specific pre-adolescent developmental period, Culbert et al. (4) reported on a sample of prepubertal twins (mean age 11.5). They found that 4.82% of the girls in this sample were above the cutoff on the Minnesota Eating Behavior Survey (5), indicating eating disordered behaviors of clinical concern. Considering the full sample of girls, 14.7% reported eating in response to emotion; 39.5% reported stuffing themselves with food; 19.2% reported eating a large amount of food with a lack of control; 16.4% reported frequently thinking about binge eating; 18.6% reported restricting food intake around others, but binge eating when alone; and 1.1% reported thinking about trying self-induced vomiting. Many studies combine girls across the transition into adolescence. Of children ages 8-14, 20%-56% are dieting, 44%-71% are exercising to lose weight, and 1.4%-10% are purging or using laxatives, while 4%-6.5% are binge eating (6-10). Although these studies do suggest the presence of pre-adolescent disordered behavior, they do not provide estimates for pre-adolescent girls specifically. Nevertheless, taken together, the research indicates that disordered eating and dieting behaviors are present even among girls prior to adolescence.

The model we propose to explain risk for disordered eating and dieting behaviors in pre-adolescent girls is an extension of a model proposed by Combs and Smith (11). The model holds that important components of the risk pathway for binge eating and purging include pubertal onset, the personality trait of negative urgency, and thinness and eating expectancies. We next explain each of these risk factors individually, followed by a summary of our integrative model.

Puberty

Puberty is associated with increased levels of eating disordered behaviors and attitudes, even among children at the same age but with different pubertal statuses (12). The mechanism by which the experience of puberty results in higher symptom levels is not fully known, in part because there are profound biological and social changes that girls go through during these years. But one possible mechanism is as follows: Pubertal onset is associated with increased levels of emotional volatility and negative affect (13,14), and also with an increase in rash or impulsive action undertaken when emotional (15,16). In our view, this change reflects a pubertal-based increase in the personality trait of negative urgency, which is the tendency to act rashly when distressed (17,18). We believe that this developmental increase in negative urgency increases eating disorder risk, through a mechanism we will describe below.

Negative Urgency

The disposition to engage in some form of rash, impulsive action in response to distress, presumably to alleviate the distress, appears to increase the likelihood that some women will engage in binge eating in response to their distress (19,20). In a meta-analysis comparing the associations of several impulsivity-related traits to bulimia nervosa symptoms in late adolescent girls and in women, Fischer et al. (19) found that negative urgency had by far the largest effect size ($r = .40$). In addition, change in negative urgency levels is associated with change in bulimia nervosa symptoms (21), and negative urgency at the start of college interacted with being a victim of sexual assault to predict subsequent bulimia nervosa symptoms (22). Further, women with bulimia nervosa are most likely to binge eat on days when they are experiencing a great deal of negative affect, and their binge eating reduces that distress (23). We propose that individual differences in negative urgency among pre-adolescent girls are associated with binge eating and purging behaviors, just as they are in late adolescents and adults.

Eating Disorder Expectancy Theory

Eating disorder expectancy theory is an application of classic expectancy theory as first articulated by Tolman (24) and as elaborated by Bolles (25), MacCorquodale and Meehl (26), Rotter (27), and others. The basic science literature has identified expectancies as learned anticipations of the likely consequences of behavioral choices. 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 with high levels of these eating expectancies 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.

Expectancies that eating helps one manage negative affect and that dieting/thinness lead to overgeneralized life improvement differentiate among anorexia nervosa, bulimia nervosa, and control patients (28). The expectancies also correlate cross-sectionally with both binge eating and purging behavior in both adolescent and adult samples (28-30) and predict the subsequent onset of those symptoms in middle school girls (31,32). Also, manipulations of dieting/thinness expectancies produce reductions in eating disorder symptoms (33).

Although numerous studies have documented linear relations between dieting/thinness expectancies and symptom reports (28,30-32), Combs et al. (34) demonstrated that the relationship between expectancies and symptoms consists of a combination of linear and quadratic trends: variation in low and moderate levels of dieting/thinness expectancy endorsement are unrelated to symptom levels, but variation within high levels of expectancy endorsement are strongly associated with symptom reports. We believe the same will be true among pre-adolescent girls.

Integration of Dispositional and Learned Risk Factors: the Acquired Preparedness (AP) Model of Eating Disorder Risk Extended

Theoreticians have described several ways in which personal disposition transacts with environmental events to influence behavior: as a function of individual differences in personality, individuals expose themselves to different experiences and they react to those experiences differently (35-38). AP risk models are an extension of this transactional theory. Specifically, individuals are differentially prepared to acquire high risk expectancies as a function of their personalities (39,40).

Combs and Smith (11) applied the AP risk model to eating disorders. They argued the following: Negative urgency predisposes individuals to act rashly when distressed. Many rash actions, such as binge eating, do mitigate one's distress by providing reinforcement (41) and by distraction from the original distress (20,42). Because binge eating is reinforced, individuals come to develop expectancies that eating provides the reward of relief from one's distress; the expectancy, in turn, increases the likelihood of future binge eating.

We thus tested whether the relationships among the predictors, binge eating, and purging were consistent with the theory. Specifically, we anticipated the following. Pubertal status would concurrently predict negative urgency. It would also concurrently predict endorsement of eating expectancies and that relationship would be mediated by negative urgency. Negative urgency would concurrently predict binge eater status, and that relationship would be mediated by eating expectancies. Both eating expectancies and quadratic dieting/thinness expectancies would concurrently predict binge eating, and

through that relationship the expectancies would indirectly predict purging. Figure 1 depicts the model.

Of course, because this test was based on cross-sectional data, it is not a test of the temporal sequence of influences implied by the model. Rather, it is a test of whether the relationships among the variables are consistent with the model: if they are not, the viability of the model would be jeopardized. If they are consistent with the model, then longitudinal tests of the proposed temporal sequence are indicated.

Method

Subjects

Participants in the study ($n=905$) consisted of 5th grade girls from public school systems in urban, rural, and suburban areas. The ethnic breakdown of the sample was as follows: 60.7% Caucasian, 17.6% African America, 6.3 % Hispanic/Latino, 4.0% Asian, 0.5% Arabic, and 10.9% of students endorsing “Other.” The majority of the 5th grade girls, 66.6%, were age 11; 23.8% were age 10; 9.0% were age 12; and 9 and 13 year olds made up 0.2% and 0.3% of the participants respectively.

Measures

The Pubertal Development Scale (PDS; 43) consists of five questions for girls. This measure correlates highly with physician ratings and other forms of self-report (r values ranging from .61 to .67; 44,45). The PDS permits dichotomous classifications as pre- or post-pubertal, which are frequently used (e.g., 4), and which we used in the current study.

The UPPS-R-Child Version (18) was used to measure negative urgency. Responses to this questionnaire follow a four-point Likert-type format. Zapolski et al. (46) reported that the child negative urgency scale was internally consistent ($\alpha = .87$), has good convergent validity across method of assessment, good discriminant validity from other impulsivity-related constructs, and good criterion-related validity in a sample with a mean age of 10.5. In the current sample, negative urgency was again internally consistent ($\alpha = .85$).

Eating Expectancy Inventory (EEI; 28). The EEI provided the measure of the expectancy that eating helps one manage negative affect. In previous work, this scale has proven to be internally consistent and to predict membership in trajectory classes characterized by increased binge eating over time (31). In the current study, the measure was again internally consistent ($\alpha = .92$).

Thinness and Restricting Expectancy Inventory (TREI; 28). The TREI measures overgeneralized expectancies for life improvement from thinness and restricting food intake. The scale has been shown to be internally consistent and predictive of the onset of eating disorder symptomatology (31); interventions that reduce thinness expectancies also reduce eating disorder symptoms (33). An example item is, “If I were thin, I would feel more worthwhile.” The internal consistency of the scale in this sample was $\alpha = .92$.

Eating Disorder Examination-Questionnaire (EDE-Q; 47). The EDE-Q is a self-report version of a semi-structured interview (EDE; 48) which assesses eating disorder symptoms. Overall scale scores, subscales scores, and ratings of binge eating and purging frequency from the EDE and EDE-Q have been found to be correlated (47,49). There is considerable evidence for the validity of the EDE-Q, including evidence for convergent validity, good ability to differentiate recurrent from infrequent bingers, and the ability to validly identify weight and shape concerns (50). The questions on the EDE-Q were modified to reflect the past 14 days (from the past 28 days), following prior work with early adolescents (e.g., 51).

For the present study, we used EDE-Q reports to identify girls who had engaged in binge eating and girls who had engaged in purging. Binge eater status was defined by an affirmative answer on two separate questions on the EDE-Q, one asking about the frequency of having eaten a large amount of food while feeling out of control over the past two weeks and another defining binge eating and asking if the participant had ever engaged in the behavior. If the participant had an affirmative answer on both of these questions, then we considered it a stringent representation of binge eating behavior in the sample. Purger status was determined by the participants' response to a question asking if they had made themselves sick (throw up) in the past two weeks as a means of controlling their shape or weight. We also report the frequency of binge eating and the frequency of purging below.

Procedures

Questionnaire Administration Procedures—The questionnaires were administered in 23 public elementary schools during school hours. A passive consent procedure was used. Out of 978 5th grade girls in the participating schools, 92.5%, or 905 of all of the girls in the system, participated in the study. A total of 73 girls did not participate due to one of the following reasons: families declined to participate, students declined assent, or a variety of other reasons, such as language disabilities that precluded completing the questionnaires.

Questionnaires were administered in school classrooms. It was made clear to the students that their responses on the questionnaire were to be kept confidential and no one outside of the research team would see them. The research team introduced the federal certificate of confidentiality for the project and emphasized that they were legally bound to keep all responses confidential. After each participant signed the assent form, the researchers then passed out packets of questionnaires. The procedure took 60 minutes or less. This procedure was approved by the University's IRB and by the participating school systems.

Data Analytic Method—The aim of this investigation was to test a sequence of predictive relationships: puberty onset predicts higher levels of negative urgency, which predict higher levels of eating expectancies, eating expectancies and quadratic dieting/thinness expectancies predict binge eater status, which predicts purger status. As noted above, we hypothesized a series of mediational relationships among these variables. We adopted a structural equation modeling (SEM) approach. We constructed one primary structural model including several variables: puberty, negative urgency, linear dieting/thinness expectancies and linear eating expectancies, quadratic thinness expectancies, binge eating and purging.¹

We represented negative urgency and the 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 (52,53), we view variability in indicator responses as effects of variability in the underlying construct. We modeled each latent variable using four parcels (or groups) of items as manifest indicators. We used parcels of items for the following reasons: 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

¹An alternative to testing an omnibus model of a specific theory is to do a series of model comparisons, in which one tests whether inclusion of hypothesized pathways improves model fit. In an important substantive way, the two approaches do not differ: if a pathway is significant in the omnibus model, inclusion of it using a stepwise procedure will result in significant model improvement. If a pathway is not significant in the omnibus model, including it will not improve model fit. Because our theory was well enough developed to enable us to specify, a priori, each path as hypothesized either to be significant or to be zero, we presented the results as a single, omnibus model for the sake of parsimony of presentation.

because each estimate is based on more stable indicators. These advantages have been described by Little, Cunningham, Shahar, and Widamon (54). The crucial relevant caution about using parcels is that they could mask multidimensionality in an item set (54,55). Negative urgency and the two expectancy constructs have been shown to be unidimensional in independent, prior factor analyses (28,29,46,56), so that concern is significantly mitigated.

To measure quadratic thinness expectancies, we followed the approach recommended by Marsh, Wen, and Hau (57) for constructing product terms in SEM models of latent variables; this approach was used by Combs et al. (34). 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, 3 and 4: the indicators of quadratic expectancies were centered parcel 1 squared, centered parcel 2 squared, centered parcel 3 squared and centered parcel 4 squared. We centered the parcels to remove nonessential collinearity between the linear and quadratic variables (57,58), our squares of the parcels reflect Marsh et al.'s (57) 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 (58).

We measured SEM model fit using four common indices: the Comparative Fit Index (CFI), the Tucker Lewis Index (TLI), the root mean square error of approximation (RMSEA), and the standardized root mean square residual (SRMR). Guidelines for these indices vary: CFI and TLI values of .90 or .95 are described as representing good fit (59,60). RMSEA values of .06 are thought to indicate a close fit, .08 a fair fit, and .10 a marginal fit (59,61), and SRMR values of approximately .09 tend to indicate good fit (59). 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 (62) to run the analyses.

Results

Descriptive Statistics

The sample showed a 10.6% rate of binge eating in girls. The number of binges over the past two weeks ranged from 1 to 14; the mean was 2.38. The sample had a 6.9% rate of purging among girls, with the mean number of days purged over the past two weeks at 2.19. The lowest number of purges among girls who purged was 1 and the highest was 7. In this sample, 29.3 % of girls reported themselves as having gone through puberty, with 19.8% reporting having begun menstruating.

Correlations Among Pubertal Status, Negative Urgency, Eating Expectancies, Dieting/Thinness Expectancies, Binge Eater Status, and Purger Status

Table 1 presents correlations among the study variables. Because pubertal status, binge eater status, and purger status are dichotomous, correlations between those three variables are phi coefficients and correlations between any of those variables and other variables are point biserial correlations. Correlations among negative urgency and the expectancies are product moment correlations. As the table shows, pubertal onset is associated with increased negative urgency, increased linear and quadratic dieting/thinness expectancies, and higher rates of purging. Negative urgency is associated with higher levels of all expectancies, binge eating, and purging. Eating expectancies are associated with dieting/thinness expectancies, and there is an association between the linear and quadratic dieting/thinness expectancy scores. Binge eater status is related to purger status.²

Test of the Theoretical Model

Our model involved a risk process indicating relationships from puberty to negative urgency, negative urgency to eating expectancies, eating and dieting/thinness expectancies to binge eating and binge eating to purging; also, the latent variables reflecting the expectancies were allowed to correlate with one another. We hypothesized the following set of mediational pathways: (1) from puberty through negative urgency to eating expectancies; (2) from negative urgency through eating expectancies to binge eater status; (3) from negative urgency through binge eater status to purger status; (4) from eating expectancies through binge eater status to purger status; and (5) from quadratic dieting/thinness expectancies through binge eater status to purger status. In addition to these mediational pathways, we also allowed puberty to directly relate to all other variables, negative urgency to directly relate to binge eating and purging, and eating expectancies to relate directly to purging. We did so to allow for the possibility of effects not captured by our proposed mediational pathways. The model is depicted in Figure 1.

It fit well, with a CFI of 0.98, a TLI of 0.98, an RMSEA of 0.04 and an SRMR of 0.04.³ As anticipated, pubertal status was associated with higher levels of negative urgency, negative urgency was associated with eating expectancies, both quadratic dieting/thinness and eating expectancies were associated with binge eating, and binge eating was associated with purging. Interestingly, pubertal status also had a direct effect on purging, i.e., an effect not mediated by other variables in our model. Negative urgency had an unanticipated effect on quadratic dieting/thinness expectancies; it also had direct effects on both binge eating and purging, i.e., effects not mediated by expectancies. Eating expectancies had a direct, non-mediated effect on purging.

To confirm that the nature of the quadratic relationship between dieting/thinness expectancies and binge eating had the form we hypothesized, we conducted follow up tests. Among girls whose dieting/thinness expectancies were at the mean or below, there was no association between the expectancies and binge eating ($r = .04, p = .42$). Among girls whose dieting/thinness expectancies were above the mean, there was a significant association ($r = .26, p < .001$). Thus, just as was true among early adolescents (34), variation at the upper end of dieting/thinness expectancy endorsement is what is associated with binge eating behavior in pre-adolescent girls.

To understand the magnitude of these associations, we computed odds ratios for each predictor with binge eating and purging. The odds of binge eating were 2.59 times higher, and the odds of purging were 1.46 times higher, for each one unit increase in negative urgency. The odds of binge eating were 1.16 times higher for those who reported high thinness expectancies (more importantly, for those girls above the dieting/thinness expectancy mean, the odds ratio for binge eating was 1.84). The odds of purging were 1.20 times higher for those who reported high eating expectancies, 1.84 times higher for those who had already gone through puberty, and 2.30 times higher for participants who had higher rates of binge eating.

²In order to determine whether there was significant covariance among the study variables due to participants attending the same school, we calculated intraclass coefficients for each of our major model components (using school membership, $n = 23$, as the nesting variable). Example intraclass coefficients were as follows: negative urgency = .03; linear thinness expectancies = .00, quadratic thinness expectancies = .00; pubertal onset = .02. These low values indicate that very little or no shared variance among study variables was due to attending the same school.

³We also ran the model excluding the direct relationships between puberty and negative urgency, thus testing only the pathways we hypothesized. The fit of this alternative model was also good (CFI = .98; TLI = .97; RMSEA = .04, 90% confidence interval: .04 - .05; and SRMR = .04). Although this model fit slightly worse than the model reported in the text (comparison $\chi^2 = 34.74, p < .001$), the magnitude of the difference in fit was quite small. We reported the unrestricted model in the text so researchers can be aware of significant pathways in addition to those we hypothesized.

Tests of Mediation Pathways—We present the statistical tests of mediation for each of the five hypothesized mediational effects. First, statistical tests of mediation were consistent with the hypothesized mediational processes involving pubertal status and negative urgency: negative urgency appeared to mediate puberty's effect on eating expectancies ($z = 3.49, p < .001, b = .04$). Second, results were consistent with the AP hypothesis that expectancies mediate negative urgency's influence on symptoms: eating expectancies appeared to mediate the influence of negative urgency on binge eater status ($z = 4.37, p < .001, b = .05$). Third, binge eater status appeared to mediate the influence of negative urgency on purger status ($z = 2.20, p < .05, b = .02$). Fourth, binge eater status appeared to mediate the influence of eating expectancies on purger status ($z = 2.22, p < .05, b = .02$), and fifth, binge eater status also appeared to mediate the influence of quadratic dieting/thinness expectancies on purger status ($z = 2.19, p < .05, b = .02$). Thus, the patterns of covariation in the data were consistent with each of the five mediational processes hypothesized in the model.

Discussion

One implication of this research is that it is both clinically important and feasible to develop risk models to understand eating disorder behaviors that occur very early in life, typically before the onset of diagnosable disorders. It is important because in this sample of 5th grade girls, just as has been reported in similarly aged children in the past (4,5), eating disordered behaviors are present: over 10% of the girls in our sample reported binge eating and almost 7% reported having purged their food through self-induced vomiting. It is feasible, in that we applied a risk model previously developed and partially tested among late adolescents and adults (11,19,28,31,34) successfully to explain the presence of binge eating and purging behavior in this young sample.

The concurrent pattern of associations in this sample were consistent with our model that pubertal onset is associated with increases in negative urgency (the tendency to act rashly when distressed); that negative urgency influences eating disorder symptoms in the acquired preparedness-specified way of shaping psychosocial learning (expectancy formation), thus indirectly influencing symptom levels; and that many influences on purging are indirect, through their influence on binge eating (as purging tends to follow binge eating).

Several aspects of this empirically supported risk model are noteworthy. First, it proposes a mechanism by which pubertal onset increases risk: pubertal onset may increase levels of negative urgency, which has been shown to be associated with symptom level in late adolescents and adults (19) and which may operate similarly in pre-adolescent girls. Second and relatedly, the model specifies a process by which negative urgency increases risk: girls high in negative urgency appear to be differentially prepared to acquire the high risk expectancies that eating helps one manage negative affect. This expectancy, in turn, is associated with symptom expression in 5th graders, as is the expectancy that dieting/thinness lead to overgeneralized life improvement. These relationships also exist in early adolescents (29,31,34), late adolescents (30), and adults (28). Thus, if puberty leads to higher levels of negative urgency, it may create a facilitative condition for high-risk learning.

Third, it appears to be true for 5th graders, just as for adolescents, that dieting/thinness expectancies relate to symptom expression quadratically. Just as was true in adolescents (34), for 5th grade girls falling at or below the mean on dieting/thinness expectancies, their expectancies did not predict whether they engaged in either binge eating or purging. In contrast, for 5th grade girls reporting expectancies above the mean, prediction of binge eater and purger status was strong. It may well be that moderate endorsement of the expectancy for overgeneralized life improvement from dieting and thinness is both normative and,

perhaps to some degree, accurate. Only young girls and adolescents who endorse that expectancy to an unusually strong degree are at increased risk for eating disordered behavior.

There were also findings not specified by our theory that were noteworthy. Puberty was related to purging behavior, in part independent of all the predictors in our model: there appears to be another mechanism of influence on purging beyond what we have specified. Negative urgency was related to both binge eating and purging not just through the mediated paths we hypothesized, but also independent of those paths. Again, there appears to be another mechanism by which negative urgency influences symptom levels beyond what is included in our model. Negative urgency was related to dieting/thinness expectancies. We had not hypothesized this association and we do not have a ready explanation for it.

It is important to appreciate the distinction between the model driving this research and the nature of the empirical test we conducted. The model we have specified describes a set of causal processes leading to disordered eating that occur in a temporal sequence. This empirical test did not address the causal nature of the process, nor did it assess the temporal sequence of influences implied by the model. Rather, it tested whether the pattern of associations among these variables in 5th grade girls was consistent with the model. The data were consistent with the model, indicating the merit in investigating this model prospectively.

It is critical to note that though this risk model may capture one pathway, there is certainly evidence that other personality factors may contribute to other forms of eating disorder expression (63). Personality is also one of many risk factors that may contribute to disordered eating; other important factors include genetics (4,12), along with family dynamics and environmental factors (64).

Of course the biggest limitation of this study is its cross-sectional nature: the model needs to be tested with more rigorous longitudinal methods. Nevertheless, there are good empirical and conceptual reasons for our choices to order the variables as we did. First, surely pubertal onset precedes changes in negative urgency, and not the other way around. Second, both longitudinal laboratory work and longitudinal field work support the premise that personality predicts subsequent changes in expectancies (34,40,65). Third, eating and dieting/thinness expectancies predict the subsequent onset of binge eating and of purging (31), and dieting/thinness expectancy manipulations reduce symptom levels (33). Thus, although all variables were measured at the same time, the ordering of influences implied by our model and implicit in this model test are well-supported by prior research and theory.

This study had other important limitations as well. This sample was not clinical, so the results reported do not necessarily reflect clinically important eating disorder behavior. Because the model is one of risk, this limitation may not be serious. Also, the data were all from self-report questionnaires. It is possible that interview assessments could have provided opportunities for clarification and increased precision. However, it may also be true that 5th grade girls would be reluctant to admit thoughts and behaviors in a face-to-face encounter with an adult that they would admit in a confidential questionnaire.

In summary, even pre-adolescent 5th grade girls report engaging in eating disorder behaviors of obvious clinical concern. It is both important and feasible to develop an understanding of the risk process in children this age. We hope the model described here generates further advances toward this important goal.

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Figure 1.

Visual representation of the empirical test of the risk model. The arrows represent the significant tested pathways; the corresponding numbers refer to the maximum likelihood coefficient for each respective relationship. Variables represented in ovals are latent variables; variables represented in squares are measured variables. Relationships modeled but not visually represented are as follows. We did not include the loadings of the various parcels onto the latent variables in the interest of clarity; these loadings ranged from .74 to .94. We did not include either error terms or disturbance terms, nor did we include correlations among the three expectancy measures. In the measurement model of dieting/thinness expectancies, each parcel for linear expectancies was allowed to correlate with its square, which was a parcel for quadratic expectancies.

Table 1

Correlations among the variables.

	Puberty	Urgency	LTE	QTE	EE	Binge	Purge
Puberty	--						
Urgency	.13**	--					
LTE	.08*	.21**	--				
QTE	.12**	.09**	.41**	--			
EE	.03	.24**	.32**	.12**	--		
Binge	.05	.25**	.27**	.24**	.27**	--	
Purge	.10**	.12**	.12**	.06	.13**	.15**	--

NOTE: LTE = Linear Thinness Expectancies; QTE = Quadratic Thinness Expectancies; EE = Eating Expectancies;

* = significant at the p < .01 level;
** = significant at the p < .001 level.