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A Longitudinal Transactional Risk Model for Early Eating Disorder Onset

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Abstract

The presence of binge eating behavior in early middle school predicts future diagnoses and health difficulties. The authors showed that this early binge eating behavior can, itself, be predicted by risk factors assessed in elementary school. We tested the acquired preparedness model of risk, which involves transactions among personality, psychosocial learning, and binge eating. In a sample of 1,906 children assessed in the spring of fifth grade (the last year of elementary school), the fall of sixth grade, and the spring of sixth grade, we found that fifth grade negative urgency (the personality tendency to act rashly when distressed) predicted subsequent increases in the expectancy that eating helps alleviate negative affect, which in turn predicted subsequent increases in binge eating behavior. This transactional risk process appeared to continue to occur at later time points. Negative urgency in the fall of sixth grade was predicted by fifth grade pubertal onset, binge eating behavior, and expectancies. It, in turn, predicted increases in high-risk eating expectancies by the spring of sixth grade, and thus heightened risk.

Eating disorder symptoms, such as binge eating behavior, are present at the beginning of adolescence for many girls (Beato-Fernandez, Rodriguez-Cano, Belmonte-Llario, & Martinez-Delgado, 2004; Bryant-Waugh & Lask, 1995; Cotrufo, Cella, Cremato, & Labella, 2007; Franko & Omori, 1999; Gardner, Stark, Friedman, & Jackson, 2000; Shisslak, Crago, McKnight, Estes, Gray, & Parnaby, 1998; Shomaker, Tanofsky-Kraff, Elliott, Wolkoff, Columbo, Ranzenhofer, Roza, et al., 2010; Tanofsky-Kraff, Shomaker, Olsen, Roza, Wolkoff, Columbo, Raciti et al., 2011). This is important because eating disorder symptoms and risk behaviors in early adolescence are highly predictive of later, diagnosable disorders (Killen, Taylor, Hayward, Wilson, Haydel, Hammer, et al., 1994; Kotler, Cohen, Davies, Pine, & Walsh, 2001; Smith, Simmons, Flory, Annus, & Hill, 2007). Anorexia nervosa and bulimia nervosa symptoms at the beginning of adolescence correlated greater than $r = .40$ with symptoms during adulthood, and diagnosable bulimia nervosa at the beginning of adolescence was associated with a nine-fold increase in bulimia nervosa during late adolescence and a 20-fold increase in anorexia nervosa during late adolescence (Kotler et al., 2001). To the degree that it is possible to predict the presence of eating disorder symptoms at the start of middle school from assessments conducted in elementary school, it will be possible to assess risk earlier than is typically done. The current paper reports on a test of this possibility.

We describe a risk model for the very early onset of binge eating behavior and report on a longitudinal test of the model in children making the transition from elementary school to middle school. Using a developmental psychopathology perspective, we understand risk for very early binge eating behavior to be a function of multiple, mutually influencing factors (Cicchetti, 2006). In particular, we believe that personality traits, psychosocial learning, prior behavior, and the biological experience of pubertal onset all influence one another to alter the risk status of children as they make the important transition into middle school. In what follows, we present the particulars of our risk model and the results of our three wave longitudinal model test. We studied 1,906 children making the transition from fifth grade, which is the last year of elementary school, through sixth grade, the first year of middle school.

The Transition into Middle School

The transition into middle school represents a key part of the contextual change associated with the move from childhood to adolescence. Middle school children encounter larger, more impersonal school contexts (Barber & Olsen, 2004; Eccles, Wigfield, Harold, & Blumenfeld, 1993) and they experience a new level of personal autonomy; they become much more independent of parents than they were in elementary school (Eccles & Midgley, 1989). Even if they have not experienced pubertal onset themselves, the bulk of middle schoolers have, which contributes to a context in which the needs and drives associated with physically mature bodies are manifest. As a result, this transition has been described as a potential turning point in development (Graber & Brooks-Gunn, 1996; Rutter, 1994); that is, a period characterized by significant behavioral and developmental change. We argue that to understand early adolescent behavior and its sequelae, it is necessary to understand the characteristics of elementary school children that influence the nature of the transition into middle school. For this reason, we sampled children prior to middle school (fifth grade), at the beginning of middle school (fall of sixth grade), and at the end of the first year of middle school (spring of sixth grade).

Acquired Preparedness Model of Risk

Risk for maladaptive functioning is no doubt a function of transactions among numerous contextual, environmental, and personal characteristics of individuals, including both the psychological and the biological (Cicchetti, 2006; Gunnar & Vazquez, 2006; Jacobi, Hayward, de Zwann, Karemer, & Agras, 2004; Stice, 2002). In this report, we describe a test of one hypothesized transactional model, known as the acquired preparedness (AP) model of risk. The AP model is adapted from person-environment transaction theory (Caspi, 1993; Caspi & Roberts, 2001). Among the contributions of person-environment transaction theory is the recognition that individuals with different personality traits will respond differently to the same environmental event. The AP model extends that concept to hypothesize that because individuals can respond differently to the same event, they can actually learn different things from the same event. Thus, psychosocial learning is not just a function of experience; rather, one's dispositional characteristics help shape one's learning experiences. One's learning, in turn, influences one's behavior. As a result, the influence of personality disposition on behavior is mediated by psychosocial learning.

With respect to risk for dysfunction, the AP model holds that individuals are differentially prepared to acquire high-risk expectancies as a function of high-risk personality traits (Smith & Anderson, 2001; Smith, Williams, Cyders, & Kelley, 2006). This process has been shown several ways: (a) In the laboratory (Smith et al., 2006); (b) in one longitudinal eating disorder field study (trait ineffectiveness predicted increased expectancies for reinforcement from thinness, which in turn predicted increased eating disorder symptoms in girls entering high school, (Combs, Smith, Flory, Simmons, & Hill, 2010b); (c) in cross-sectional eating disorder research (negative urgency predicted eating expectancies which predicted binge eating behavior in fifth graders, Combs, Pearson, & Smith 2010a; Pearson, Combs, & Smith, 2010); and (d) in longitudinal research on alcohol misuse (Corbin, Iwamoto, & Fromme, 2011; Settles, Cyders, & Smith, 2010). However, prior to the current research, no study has tested whether this process appears to operate during the formative transition from elementary school to middle school.

The version of the AP model we tested was described by Combs and Smith (2009) and involves transactions among two specific risk factors for binge eating: The high risk personality trait of negative urgency and the high risk psychosocial learning reflected in eating expectancies. We next review those constructs and their relevance for binge eating risk.

Personality risk: Negative urgency

Negative urgency, the tendency to act rashly when distressed, appears to be a particularly important personality risk factor for binge eating behavior (Fischer, Smith, & Cyders, 2008; Whiteside & Lynam, 2001). The trait has been identified as one of five personality traits that disposes individuals to impulsive action; the others are sensation seeking (the tendency to seek out novel, thrilling stimulation), lack of planning (the tendency to act without forethought), lack of perseverance (a limited capacity to maintain focus on tasks, particularly when bored), and positive urgency (the tendency to act rashly when in an unusually positive mood: Cyders, Smith, Spillane, Fischer, Annus, & Peterson, 2007; Whiteside & Lynam, 2001). Among those five traits, negative urgency is the only one with a substantial relationship to bulimia nervosa symptomatology (Cyders et al., 2007; Fischer et al., 2008).[1] Moreover, change in negative urgency levels is associated with change in bulimia nervosa symptoms (Anestis, Selby, & Joiner, 2007), and negative urgency has been found to concurrently predict both binge eating and purging behavior in elementary school-age girls (Combs et al., 2010a) and boys (Pearson et al., 2010). The possibility that the tendency to act rashly when distressed predicts binge eating is consistent with ecological momentary assessment data indicating increased levels of negative affect before binge eating episodes (Haedt-Matt & Keel, 2011; Smyth, Wonderlich, Heron, Sliwinski, Crosby, Mitchell, & Engel, 2007). We thus anticipated negative urgency to be an important risk

[1]Negative urgency is also distinguishable from general negative affectivity (Cyders & Smith, 2008). When the two are considered together, negative urgency but not negative affectivity concurrently predicts several other forms of impulsive or addictive behavior, including preadolescent drinking and smoking (Settles, Fischer, Cyders, Combs, Gunn, & Smith, in press); late adolescent problem drinking, illegal drug use, risky sex, aggression, conduct problems, and intimate partner violence (Derefinko, DeWall, Metze, Walsh, & Lynam, 2011; Settles et al., in press); and adult alcohol dependence diagnosis (Settles et al., in press).

factor for binge eating behavior as children transition into middle school. This study is the first to report a prospective predictive role for the trait.

Psychosocial learning risk: Expectancies for reinforcement from eating

Expectancies represent summaries of individuals' learning histories and are formed based on the multitude of direct and vicarious learning experiences that individuals undergo. They are learned anticipations of the likely consequences of behavioral choices. The expectancies one forms influence one's future behavioral choices. One tends to choose behaviors from which one expects rewards and avoid behaviors for which one expects punishment.

Individual differ in their expectancies about eating. Due to their personalities (as described below) and learning histories, some individuals form unusually strong expectancies that eating helps manage one's negative affect (Annus, Smith, Fischer, Hendricks, & Williams, 2007; Atlas, Smith, Hohlstein, McCarthy, & Kroll, 2002; Boerner, Spillane, Anderson, & Smith, 2004; Hohlstein, Smith, & Atlas, 1998; MacBrayer, Smith, McCarthy, Demos, & Simmons, 2001; Simmons, Smith, & Hill, 2002; Smith et al., 2007). It seems likely that learned expectancies that eating will help one manage one's distress play an important role in the development and maintenance of emotional eating, which is defined as eating in response to negative emotions in an effort to cope with negative affect (Faith, Allison, & Geliebter, 1997; Tanofsky-Kraff, Theim, Yanovski, Bassett, Burns, Ranzenhofer, Glasofer, et al., 2007). Children who hold the belief that eating will alleviate negative affect will most likely be at greater risk to eat as a means of coping with negative emotions. These eating expectancies have been shown to cross-sectionally correlate with symptom level in child (Combs et al., 2010a; Pearson et al., 2010), adolescent (MacBrayer et al., 2001; Simmons et al., 2002) and adult samples (Fischer, Settles, Collins, Gunn, & Smith, in press; Hohlstein et al., 1998). The expectancies also predict binge eating in longitudinal samples of adolescent girls (Smith et al., 2007; Stice & Whitenton, 2002). We thus anticipated that early endorsement of expectancies that eating helps alleviate negative affect would predict binge eating behavior across the transition into middle school.

The AP model for eating disorder risk (Combs & Smith, 2009) uses these two risk factors and is as follows. Children high in negative urgency are disposed to act to alleviate distress; often, their actions are rash or impulsive (Settles et al., in press). When children high in this trait are exposed to learning experiences (whether direct or modeled) that involve eating when distressed, they are biased to form expectancies associating the behavior of food consumption with the negative reinforcement of distress relief (Fischer et al., 2008). This expectancy, in turn, is understood as the proximal risk factor that increases the likelihood of binge eating behavior (Smith et al., 2007). Thus, over time, negative urgency should predict increased endorsement of expectancies that eating will help alleviate distress, and those expectancies will, in turn, predict subsequent binge eating behavior. We tested this transactional process across the transition into middle school.

In addition to testing the full AP risk model by predicting binge eating at the end of sixth grade, we also tested whether this risk process was ongoing. There were two aspects to this test. First, we tested whether negative urgency levels at wave 2 (the fall of sixth grade) predicted further increases in the proximal risk factor of eating expectancies at the end of

our longitudinal period (wave 3, the end of sixth grade). Second, we tested whether it was possible to predict wave 2 negative urgency levels from wave 1 levels of negative urgency, binge eating behavior, eating expectancies, and pubertal onset.

Predictors of Negative Urgency

We anticipated some stability in negative urgency levels across the transition from late fifth grade to early sixth grade. However, even with this stability, it is also true that personality traits are somewhat malleable, particularly at important developmental transitional periods, such as the shift into middle school (Roberts, Caspi, & Moffitt, 2001). There is evidence that one's behavior in childhood leads to subsequent personality change (Garmezy & Tellgen, 1984; Masten, Coatsworth, Neeman, Gest, Tellegen, & Garmezy, 1995; Masten, Hubbard, Gest, Tellegen, Garmezy, & Ramirez, 1999), although we found no studies of personality change over a time interval as short as the one in the current study. Nonetheless, we anticipated that engagement in the rash, impulsive act of binge eating behavior would lead to increases in negative urgency over time. We anticipated that a similar process would operate with respect to eating expectancies. The expectancy that eating helps manage one's distress is endorsement of a behavior geared toward negative reinforcement that does not address the original source of one's distress. Learning of this kind, that is, learning to engage in a behavior because it is thought to provide immediate distress relief even though it does not solve the original problem, could increase the overall tendency to act in ways that alleviate distress without furthering one's interests. Such actions are often viewed as rash; in this way, eating expectancies could contribute to increased negative urgency over time.

We also anticipated that the biological experience of pubertal onset would lead to increased negative urgency. Pubertal onset is associated with increased levels of emotional volatility and negative affect (Allen & Matthews, 1997; Compas, Connor-Smith, Sasltzman, Thomsen, & Wadsworth, 1995; Spear, 2000), and also with an increase in rash or impulsive action undertaken when emotional (Luna & Sweeney, 2004; Steinberg, 2004). These changes can be understood to reflect a pubertal-based increase in the trait of negative urgency.

The model we tested is depicted in Figure 1. It includes pathways representing each of the effects we have described, in addition to (a) immediate and delayed autoregressive effects, and (b) prediction from fifth grade pubertal onset to binge eating in the fall of sixth grade. Given the increase in eating disorder symptomatology across the adolescent years, it is important to test whether pubertal onset directly predicts increased binge eating behavior prospectively. Interestingly, one prior study that involved prediction from elementary school risk factors to middle school behavior did not find a positive association between pubertal status and subsequent Eating Attitudes Test (EAT) scores (Keel, Fulkerson, & Leon, 1997). The EAT is thought to measure attitudes and behaviors consistent with anorexia nervosa and includes one subscale assessing binge-related behaviors (Garner, Olmstead, Bohr, & Garfinkel, 1982). We therefore modeled direct effects from pubertal onset to binge eating, in addition to the indirect effect we modeled from pubertal onset to negative urgency and from negative urgency to eating expectancies.

Method

Participants

The participants in this study were 1906 students (50.2% boys, 49.8% girls) assessed at three different time points: The end of their fifth grade year in elementary school, the beginning of their sixth grade in middle school, and the end of their sixth grade in middle school. The mean age of the participants at the initiation of the study was 10.86 years. Most were European American (60.9%), followed by African American (18.7%); the remainder of the sample identified themselves as Hispanic (8.2%), Asian (2.9%), Middle Eastern (0.4%), or other (8.8%).

Measures

The Pubertal Development Scale (PDS; Peterson, Crockett, Richards, & Boxer, 1988)—The PDS consists of five questions for girls and five questions for boys, using a likert-type rating format. This measure correlates highly with physician ratings and other forms of self-report (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987; Coleman & Coleman, 2002). The PDS permits dichotomous classifications: Following prior research, we rated scores of 2.5 or greater as reflective of pubertal onset.

Eating Expectancy Inventory (EEI; Hohlstein et al., 1998)—This five-factor measure reflects expectancies for reinforcement from eating. For this study, we used the measure of the expectancy that eating helps one manage negative mood states. This scale has been shown to predict subsequent onset of binge eating (Smith et al., 2007). In the current sample, as in past samples, the scale was internally consistent (Wave 1 α = .93, Wave 2 α = .95, Wave 3 α = .95).

UPPS-P Negative Urgency Scale (Lynam, Smith, Cyders, Fischer, & Whiteside, 2007)—Negative urgency is related to bulimic symptom expression in late adolescents and adults (Fischer et al., 2008). A child version of the negative urgency scale recently proved to be internally consistent (α = .87), have good convergent validity across assessment method, good discriminant validity from other impulsivity-related measures, and predictive of criteria in theoretically consistent ways (Zapolski, Stairs, Settles, Combs, & Smith, 2010). In the current sample, the internal consistency of the scales at each wave are as follows: Wave 1 α = .85, Wave 2 α = .86, Wave 3 α = .87.

Eating Disorder Examination- Questionnaire *(EDE-Q;* Fairburn & Beglin, 1994*)*—The EDE-Q is a self-report version of the Eating Disorders Examination semi-structured interview (Cooper & Fairburn, 1993) designed to assess the full range of behavioral and cognitive or attitudinal features of the specific psychopathology of eating disorders during the preceding four weeks, including patients' extreme concerns about their shape and weight. The EDE-Q has been shown to have good reliability and validity, particularly in clinical samples (Cooper & Fairburn, 1993; Luce & Crowther, 1999; Mond, Hay, Rodgers, Owen, & Beumont, 2004). In this study, we adapted the EDE-Q for use with a younger population by using age-appropriate wording, defining concepts that could possibly be difficult to understand, and shortening the length of time referred to in the questions to the

past two weeks, per past recommendations (Carter, Stewart, & Fairburn, 2001). For instance, we changed the word “restrict” to “cut back on” and the word “influence” to “control” throughout the measure. We also defined terms such as “laxatives”, “diuretics”, and “binge eating”. Our measure of binge eating emphasized binge eating with loss of control; that is, children had to endorse two items, one that assessed episodes of objective binge eating and one that assessed loss of control during these episodes. We used the following 7-point scale: 0=the absence of binge eating; 1= binge eating with no loss of control; 2= one to two loss of control episodes; 3= three to four loss of control episodes; 4= five to seven loss of control episodes; 5= eight to ten loss of control episodes; 6= eleven to thirteen loss of control episodes; 7 = fourteen or more loss of control episodes.

Procedure

Data collection—The questionnaires were administered in 23 public elementary schools during school hours for the first wave and in 19 public middle schools during school hours for the second and third waves. A passive consent procedure was used. Each family was sent a letter, through the U.S. Mail, introducing the study. Families were asked to return an enclosed, stamped letter or call a phone number if they did not want their child to participate. Out of 1988 fifth grade students in the participating schools, 95.8%, or 1906, of the students participated in the study. A total of 82 students 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.

The first data collection took place in the last month of the students’ fifth grade year in their elementary schools. The second and third data collections took place in the second and last month, respectively, of the students’ sixth grade year in middle school. Questionnaires were administered in school classrooms or cafeterias.

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. Participants who moved out of the study’s school districts were contacted and asked to complete the forms by mail and were paid $30 for doing so. The questionnaire administration took 60 minutes or less. This procedure was approved by the University’s IRB and by the participating school systems and was utilized at all three waves of the study. There was no compensation for participations (except for those who moved out of district).

Data analytic method—We adopted a structural equation modeling (SEM) approach to test this predictive model. We constructed one primary structural model that included puberty, negative urgency, eating expectancies, and binge eating. We measured SEM model fit using four common indices: The Comparative Fit Index (CFI), the Tucker Lewis Index (TLI: also known as the Nonnormed Fit Index), 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 (Brown & 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 (Muthén & Muthén, 2004) to run the analyses.

We conducted these analyses in two ways. First, we used maximum likelihood parameter estimates and an adjusted chi-square statistic that is robust to non-normality (the MLR method). Second, because there were a large number of zero values on our key criterion of binge eating (most children were not binge eating), we conducted zero-inflated poisson regression (known as ZIP models). Using the ZIP model, frequency of binge eating is treated as a count variable, and the method provides two kinds of regression results. The first predicts variation in the count of binge eating (from zero to the highest reported count) and the second is a logistic regression that predicts the probability of being unable to assume any value except zero. The MLR method provides the capacity to test for indirect (or mediated) effects; ZIP models do not. We tested mediation by using a bias-corrected bootstrapping method that increases statistical power and does not impose the assumption of normality on the data. The procedure generated 1,000 bootstrapped samples to empirically approximate the true sampling distribution because the assumption of a normal sampling distribution is not likely to be accurate. This procedure is available in MPlus (Muthén & Muthén, 2004).

The model we specified allowed for the following: (a) All autoregressive predictive effects, from wave 1 to wave 2, wave 2 to wave 3, and wave 1 to wave 3; (b) predictive effects representing the AP model (i.e., from wave 1 negative urgency to wave 2 eating expectancies, and from wave 2 eating expectancies to wave 3 binge eating); (c) predictive effects representing the ongoing nature of the AP model (i.e., from wave 1 pubertal onset, eating expectancies, and binge eating to wave 2 negative urgency, and from wave 2 negative urgency to wave 3 eating expectancies); (d) direct predictive effects from pubertal onset to binge eating (i.e., from wave 1 pubertal onset to wave 2 binge eating); and (e) all cross-sectional associations among risk factors or their disturbance terms at each wave.

We tested whether the predictive model was invariant across sex (prior cross-sectional research with this fifth grade sample indicated very similar correlations among risk factors and behavior for girls and boys: Combs et al., 2010a; Pearson et al., 2010) and across the two racial groups present in sufficient numbers for model testing: European American (n = 1,069) and African American (n = 328). We used measured variables, rather than latent variables, in part to preserve sufficient degrees of freedom for invariance testing that included the African American subsample.

For bivariate correlations, we used $p < .01$ to determine statistical significance. The SEM model involved tests of the net effects of each variable, over and above the effects of other variables. We therefore included pathways significant at $p < .05$, if the corresponding bivariate correlation was significant at $p < .01$.

Results

Attrition and Treatment of Missing Data

The retention rate from wave 1 to wave 2 was 96%; from wave 1 to wave 3 it was 92%. Analyses comparing retained and non-retained participants on all study variables indicated no significant differences. We therefore assumed data were missing at random, and we used the expectation maximization (EM) procedure to impute values for the missing data points. This procedure has been shown to produce relatively unbiased population parameter estimates and to be superior to traditional methods, such as deleting cases with missing data or conducting mean substitutions for missing values (Little & Rubin, 1989). As a result, we were able to make full use of the sample of n = 1,906.

Descriptive Statistics

The sample of 1906 children includes 949 girls and 957 boys. By the spring of fifth grade, 27.7% of the girls and 21.8% of the boys had experienced pubertal onset. Table 1 presents the means and standard deviations for negative urgency and eating expectancies at each wave for both boys and girls and Table 2 presents the frequencies of binge eating behavior by sex over the two weeks preceding the assessments. Because we understand the transition to middle school to be a time of flux for children, we did not know whether to expect a change in the number of children binge eating across the three waves. As the table shows, there was a slight decrease in binge eating over time ($F(2, 1903 = 9.46$, $p < .001$). Specifically, 403 children (21.1%) reported decreases in the number of binge episodes during the preceding two weeks, 335 (17.6%) reported increases in the number of binge episodes, and most (1,187: 61.4%) reported no change. At each wave, over 10% of girls and 8% or more of boys reported at least some objective binge eating over the preceding two weeks.

Correlations among Pubertal Status, Negative Urgency, Eating Expectancies, and Binge Eating Across all Three Waves

Table 3 presents correlations among the study variables. Pubertal status is dichotomous so correlations between that variable and any others are point biserial correlations. All other correlations are product moment correlations. As the table shows, pubertal onset was associated positively with negative urgency and binge eating in all three waves and with eating expectancies in wave 3. All three waves of negative urgency were associated with higher levels of all other variables, including eating expectancies at all waves and binge eating at all waves. The same is true for binge eating and eating expectancies: Binge eating in all waves was associated positively with negative urgency and eating expectancies at all waves, and eating expectancies were associated positively with negative urgency and binge eating in all three waves.

Test of the Theoretical Model

Using the MLR estimation method, the structural model fit well, with a CFI of .98, a TLI of .94, an RMSEA of 0.06 (90% confidence interval: 0.05 – 0.07), and an SRMR of 0.02.[2]

The risk model explained 21% of the variance in binge eating behavior at wave 3. We next summarize the results of the model test.

Autoregressive Predictive Effects

Negative urgency at wave 1 predicted negative urgency at wave 2, and both wave 1 and wave 2 negative urgency predicted wave 3 negative urgency. The same was true for eating expectancies and binge eating behavior. There was stability in participant reports on all three variables, although binge eating reports were less consistent than were the trait and expectancy reports.

Acquired Preparedness

The test of the AP model applied to children undergoing this developmental transition was supported. Wave 2 eating expectancies appeared to mediate the effect of wave 1 negative urgency on wave 3 binge eating ($z = 2.37$, $p < .01$, $b = .01$). The predictive relationship from negative urgency measured in the spring of fifth grade to binge eating behavior measured in the spring of sixth grade can be understood to be indirect. Negative urgency predicted subsequent increases in the expectancy that eating helps alleviate negative affect, which in turn predicted later increases in binge eating behavior.

Ongoing Acquired Preparedness and Prediction of Negative Urgency

Consistent with our hypotheses, wave 2 negative urgency predicted wave 3 eating expectancies, just as wave 1 negative urgency predicted wave 2 eating expectancies. In addition, wave 2 negative urgency was predicted by wave 1 negative urgency, wave 1 pubertal onset, wave 1 eating expectancies, and wave 1 binge eating behavior. Statistical tests of mediation were consistent with the hypothesized mediational processes of transactional risk: (a) Puberty predicted increases in wave 3 eating expectancies through wave 2 negative urgency ($z = 1.90$, $p < .05$, $b = .01$); (b) wave 1 eating expectancies predicted increases in wave 3 eating expectancies through wave 2 negative urgency ($z = 1.82$, $p < .05$, $b = .01$); and (c) wave 1 binge eating predicted increases in wave 3 eating expectancies through wave 2 negative urgency ($z = 1.87$, $p < .05$, $b = .01$).

Direct Prediction of Binge Eating from Pubertal Onset and Cross-Sectional Associations

Wave 1 pubertal onset also directly predicted wave 2 binge eating behavior. Cross-sectional associations among negative urgency, eating expectancies, and binge eating were significantly greater than zero at each wave.

Model Invariance Across Sex and Race

We next tested whether the structural model represented in Figure 1 was invariant across sex. When we specified the same model for boys and girls, the model fit did not decline: CFI = .98; TLI = .94; RMSEA = .04 (90% confidence interval: .02 - .07). We then constrained each pathway that was significantly greater than zero in the overall model to be equal for

[2]We also ran our model with age as a covariate and our results were unchanged. This supports previous findings (Graber & Brooks-Gunn, 1996; Rutter, 1994) highlighting the importance of the developmental transition from elementary school into middle school. The developmental transition, not just age progression, appears to be important.

boys and girls, one at a time. This process involved 21 comparisons in which we tested whether there was a significant drop in fit due to equality constraints, using the chi-square difference test. Only one time did the constraint reduce model fit: There was a significance difference between boys and girls in the pathway from wave 1 eating expectancies to wave 2 negative urgency; the coefficient was higher for girls than for boys, $p < .05$. We viewed this result skeptically because one significant effect at $p < .05$ out of 21 comparisons is what would be expected by chance. We therefore concluded that the risk model was invariant across sex.

We tested whether the structural model was invariant across race. In this analysis, we specified the same model for European American and African American youth. That model fit the data well: CFI = .98; TLI = .90; RMSEA = .06 (90% confidence interval: .04 - .08). Each index indicates good model fit, although the fit was very slightly worse than the fit of the overall model. We next constrained each of the 21 significant pathways, one by one, to be equal across ethnic group. None of those constraints produced a statistically significant decrement in model fit. We concluded, provisionally, that the model was invariant across these two racial groups.

Analysis using ZIP Modeling

As noted above, because of the large number of zero values on binge eating, we tested the model using zero inflated poisson regression modeling. Prediction of variation on the count variables of wave two and wave three binge eating was virtually identical to what was found using the MLR estimation method. Logistic regression prediction of likelihood of being able to take on any value other than zero found that the likelihood of non-zero wave two binge eating was predicted by wave one binge eating, wave one eating expectancies, and wave one pubertal onset. The likelihood of non-zero wave three binge eating was predicted by wave two binge eating, wave two eating expectancies, and wave two negative urgency. Because of the consistency of results using the two approaches, we reported the MLR estimation method results, which allowed for tests of our mediation hypotheses.

Discussion

We found that middle school or early adolescent binge eating, which has been used to predict subsequent dysfunction, can itself be predicted by characteristics of children during their elementary school years. We specified and tested a transactional model among risk factors for the early onset of binge eating behavior, and found that transactions among risk factors measured in the last year of elementary school and in the fall of the first year of middle school (sixth grade) predicted binge eating behavior at the end of sixth grade. The success of this model test (a) provides support for developmental risk models characterized by multiple risk factors that transact over time to increase the risk profile and (b) further highlights the potential value of assessing risk for binge eating as early as the elementary school years (Combs, Pearson, Zapolski, & Smith, 2011; Keel et al., 1997; Tanofsky-Kraff et al., 2011).

The particular transactional model we tested was the acquired preparedness (AP) model of risk, an extension of person-environment transaction theory (Caspi, 1993; Caspi & Roberts,

2001). Consistent with this model, we found that elementary school levels of negative urgency predicted increased levels of expectancies that eating will help manage negative affect, and expectancy endorsement predicted subsequent binge eating behavior, above and beyond prediction from prior levels of binge eating behavior. In addition, as predicted by the AP model, the predictive relationship from wave 1 negative urgency to wave 3 binge eating behavior was mediated by wave 2 eating expectancies. These findings are consistent with the hypothesis that transactions among dispositional characteristics of individuals (such as negative urgency) and their psychosocial learning experiences (reflected in eating expectancies) increase the risk for binge eating behavior across the transition into middle school.

The findings of this three-wave longitudinal study were also consistent with the possibility that this risk process continues to operate at later time points. Negative urgency measured in the fall of sixth grade predicted increased eating expectancies at the end of the longitudinal period (the end of sixth grade); thus, for some children, eating disorder risk was higher at the end of sixth grade than it was at the start of the year. Additionally, the personality trait of negative urgency in the fall of sixth grade was itself predictable from characteristics of the children when they were in fifth grade. First, pubertal onset in fifth grade predicted increases in negative urgency by sixth grade. This finding is consistent with evidence that pubertal onset is associated with increased levels of emotionality and impulsive action (Compas et al., 1995; Maggs, Almeida, & Galambos, 1995; Spear, 2000; Steinberg, 2004). Second, increases in negative urgency were predicted by prior eating expectancies: Children in fifth grade who believe that eating alleviates negative affect are more likely to develop higher levels of the personality trait negative urgency when they enter middle school. Perhaps ongoing learning regarding behavior and its consequences influences personality development. Third, increases in negative urgency were also predicted by prior binge eating behavior in fifth grade. Perhaps having engaged in binge eating led to increases in the trait of negative urgency during this developmental transition.

In addition to these findings supporting our transactional model of risk, we also found that pubertal onset by the spring of fifth grade had a direct and positive predictive effect on binge eating levels by the fall of sixth grade. The positive relationship observed in this study differs from the negative relationship between pubertal status and EAT scores observed by Keel et al. (1997) across a similar age period. However, the two tests differed in important ways. The criterion variable in Keel et al. (1997), EAT scores, reflects attitudes and behaviors consistent with anorexia nervosa. It includes binge eating, but also includes dieting and other factors. The criterion in the current study was limited to binge eating. In addition, the two studies controlled for different variables. Keel et al. (1997) controlled for body mass index, which we did not do. We controlled for prior binge eating; Keel et al. (1997) did not. Further research is necessary to understand the relationships among pubertal status, binge eating, and symptoms of anorexia nervosa.

The success of the specific risk factors chosen in this study is consistent with findings from prior studies and has important theoretical implications. Among personality traits related to rash or impulsive behavior, negative urgency has consistently emerged as the best concurrent predictor of binge eating and bulimia nervosa symptomatology (Fischer et al.,

2008). Researchers interested in the role of impulsivity in eating disordered behaviors might profitably focus on negative urgency, rather than other impulsivity-related traits. Among personality traits involving negative affect, negative urgency concurrently predicts a number of different forms of addictive and externalizing behavior, including binge eating, problem drinking, illegal drug use, risky sex, aggression, and delinquency, but general negative affectivity does not (Settles et al., in press).

The expectancy that eating helps alleviate negative affect has consistently predicted binge eating and bulimia nervosa symptomatology both cross-sectionally (Hohlstein et al., 1998; MacBrayer et al., 2001) and longitudinally (Smith et al., 2007). The concept of measuring learned expectancies is based in classic learning theory and dates back to the work of Tolman (1932), Rotter (1954), and Bolles (1972). It is consistent with findings highlighting the importance of emotional eating (Faith et al., 1997; Tanofsky-Kraff et al., 2007): From an expectancy theory perspective, individuals who have formed the expectancy that eating helps alleviate negative affect are at greatest risk to engage in emotional eating, that is, eating to help cope with negative emotions.

One important characteristic of the children's self-reported binge eating behavior is that there appears to be considerable flux in the behavior during this transitional period. Of particular interest is that different children reported different directions of change in their binge eating behavior during this one year period. Over 20% reported a drop in binge eating across the 3 waves, but almost 18% reported an increase. Strikingly, many of the children reporting the highest levels of binge eating reported increases across the longitudinal period. These findings suggest the value of person-centered approaches to understanding the changes over time. Different children are having different experiences, and the group-level analyses we have conducted cannot represent those differences. With additional waves of data, it will likely emerge that different children move along different trajectories with respect to binge eating. More extended longitudinal periods of assessment are clearly necessary, and person-centered analytic approaches could help clarify whether different binge eating trajectories this young predict diagnosable dysfunction in late adolescence and adulthood.

The support for this risk model was invariant across sex. Although many more adult women than men report eating disorders, this is less true for binge eating behavior (Streigel-Moore & Bulik, 2007). Other prior research has identified common risk processes across sex as well (Boerner et al., 2004; Pearson et al., 2010; Spillane, Boerner, Anderson, & Smith, 2004). There are also differences in risk across sex. For example, puberty appears to influence the heritability of disordered eating in girls but not boys (Klump, Culbert, Slane, Burt, Sisk, & Nigg, 2011). Further investigation of similarities and differences in eating disorder risk by sex are clearly warranted.

We did not find evidence that the risk model varied across the racial/ethnic groups of European American and African American. This finding is consistent with prior research that the rates of binge eating do not differ in African American, Hispanic, and Caucasian youth (e.g., Johnson, Rohan, & Kirk, 2002; Striegel-Moore, Schreiber, Lo, Crawford, Obarzanek, & Rodin, 2000). At the same time, the current research, like so much past

research, was characterized by the presence of many more European Americans than children from other ethnic groups in the sample. Further examination of risk across group should be conducted with comparably large samples from each ethnic group.

Our risk model describes causal processes that occur in a temporal sequence. 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 demonstrate change as predicted by the theory, in the presence of tight statistical controls. The theory survived those tests; the results of this study are consistent with, but of course not proof of, the transactional model of eating disorder risk.

It is also very likely that these processes work in conjunction with other factors not tested in this study to increase risk. Genetic liability (Klump, McGue, & Iacono, 2003), perfectionism (Vohs, Bardone, Joiner, Abramson, & Heatherton, 1999), a history of weight suppression (Lowe, Thomas, Safer, & Butryn, 2007), ineffectiveness (Combs et al., 2010b), and other factors are likely to play a role as well.

The results of this study should be understood in the context of the study's limitations. First, this study did not test any specific process or mechanism of influence and so does not provide information on the mechanisms by which the influences implied by prospective prediction operate. Second, though we experienced relatively low attrition rates and there is good evidence for the validity of the maximum likelihood method for addressing missing data, we cannot know if the results would have differed with even higher retention.

Third, we relied on questionnaires to assess each attribute and behavior in our sample. Although the EDE-Q has good reliability and validity data for clinical samples (Carter et al., 2001; Cooper & Fairburn, 1993; Luce & Crowther, 1999; Mond et al., 2004), very few studies have examined its validity in community-based, pre-adolescent samples. There is evidence to suggest that when the EDE-Q is used with a younger or community-based sample, individual subscales that involve ambiguous features, such as binge eating, should be interpreted with caution (Black & Wilson, 1996; Decaluwé & Braet, 2004; Fairburn & Beglin, 1994; Passi, Bryson, & Lock, 2003). It is possible that the measure used in this study to assess binge eating may have over-estimated the occurrence of binge eating in this young community sample. However, terms were clearly defined in each item for participants in an effort to minimize this concern and this measure is widely and routinely used in similar research. Nonetheless, interview assessments may have provided the opportunity for clarification of questions and thus a possible increase in precision. Fourth, we did not assess the context of the eating disorder behavior. As a result, we know very little about the nature of these early binge eating experiences. Fifth, we did not include a measure of body mass index (BMI). Prior research using similar constructs to those used in this study found that the inclusion of BMI did not alter the predictive relationships (Combs et al., 2010b). However, unlike the current study, those studies did not involve prediction from pubertal status. We cannot know whether inclusion of BMI would have altered the findings in the current study.

In summary, eating disorder behavior begins at a very young age and is of obvious clinical concern. A risk model that specifies transactions among pubertal onset, negative urgency, the expectancy that eating helps manage negative affect, and prior behavior predicted binge eating behavior across the transition from elementary school to middle school. In the past, binge eating at the beginning of middle school is part of what predicts subsequent diagnoses and health difficulties. As is true of a small number of other studies (e.g., Keel et al., 1997; Tanofsky-Kraff, 2011), the current study showed that middle school binge eating can itself be predicted by characteristics of children during elementary school. It appears that risk for very early binge eating can be understood to result from transactional processes among biological experiences (pubertal onset), personality, and psychosocial learning.

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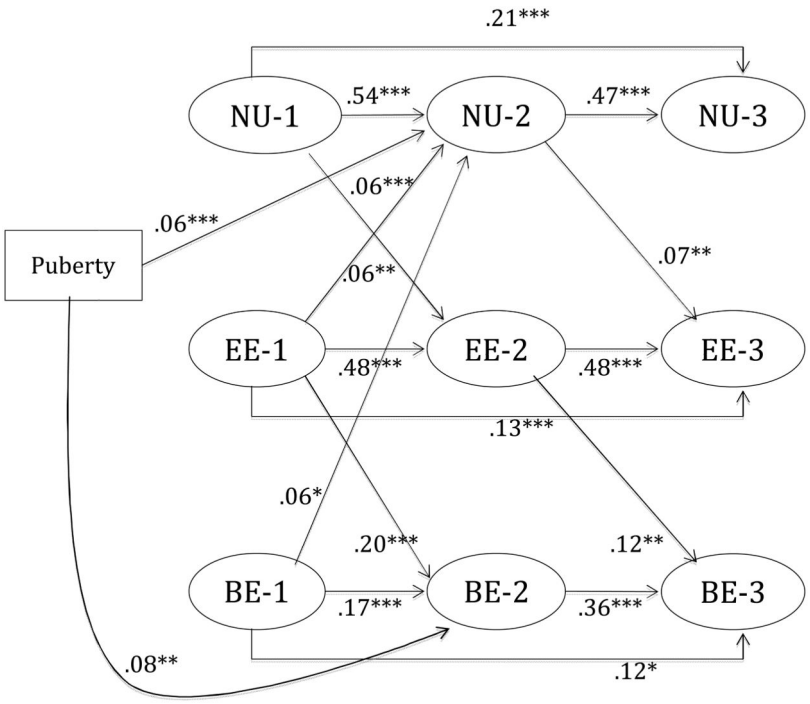

Figure 1. Structural Model with Significant Pathways

*p < .05. **p< .01. ***p< .001; NU = Negative Urgency; EE = Eating Expectancies; BE = Binge Eating; each number following the variable represents the wave in which the data was collected (i.e., NU-2 = Negative Urgency, Wave 2).

Table 1

Descriptive Statistics: Negative Urgency and Eating Expectancies

Sex	Factor and Wave	Mean	Standard Deviation
Girls	NU-1	2.21	0.71
	NU-2	2.10	0.71
	NU-3	2.20	0.74
Boys	NU-1	2.20	0.70
	NU-2	2.12	0.65
	NU-3	2.10	0.66
Girls	EE-1	2.19	1.31
	EE-2	1.89	1.23
	EE-3	1.90	1.27
Boys	EE-1	2.33	1.42
	EE-2	1.92	1.27
	EE-3	1.80	1.17

Note: NU = Negative Urgency; EE = Eating Expectancies; each number following the variable represents the wave in which the data was collected (i.e., NU-2 = Negative Urgency, Wave 2).

Table 2

Descriptive Statistics: Binge eating

Sex	Number of binges in last 14 days	Frequencies		
		Wave 1	Wave 2	Wave 3
Girls (n=949)	None	818	850	846
	Binge, no loss of control	36	37	32
	1 - 2	60	34	35
	3 - 4	24	15	16
	5 - 7	5	6	6
	8 - 10	1	2	4
	11 - 13	1	3	0
	14 or more	4	2	10
Boys (n=957)	None	807	860	882
	Binge, no loss of control	64	43	44
	1 - 2	46	25	16
	3 - 4	26	15	7
	5 - 7	8	2	0
	8 - 10	2	5	1
	11 - 13	3	2	1
	14 or more	1	5	6

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Table 3

Correlations among the variables

	Puberty	**NU-1**	**NU-2**	**NU-3**	**EE-1**	**EE-2**	**EE-3**	**Binge-1**	**Binge-2**
Puberty	--	--	--	--	--	--	--	--	--
NU-1	.13**	--	--	--	--	--	--	--	--
NU-2	.13**	.57**	--	--	--	--	--	--	--
NU-3	.13**	.53**	.66**	--	--	--	--	--	--
EE-1	.03	.22**	.20**	.18**	--	--	--	--	--
EE-2	.05	.20**	.29**	.20**	.51**	--	--	--	--
EE-3	.08**	.21**	.27**	.29**	.41**	.58**	--	--	--
Binge-1	.07**	.21**	.19**	.18**	.29**	.21**	.20**	--	--
Binge-2	.09**	.14**	.18**	.15**	.26**	.37**	.30**	.23**	--
Binge-3	.05*	.13**	.16**	.16**	.20**	.27**	.35**	.23**	.42**

Note: NU = Negative Urgency; EE = Eating Expectancies; each number following the variable represents the wave in which the data was collected (i.e., NU-2 = Negative Urgency, Wave 2);

* $p < .01$.

$p < .001$.