Published in final edited form as:
Int J Eat Disord. 2015 March ; 48(2): 187–198. doi:10.1002/eat.22270.

Development and Predictive Effects of Eating Disorder Risk Factors during Adolescence: Implications for Prevention Efforts

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Abstract

Objective—Although several prospective studies have identified factors that increase risk for eating disorders, little is known about when these risk factors emerge and escalate, or when they begin to predict future eating disorder onset. The objective of this report was to address these key research gaps.

Method—Data were examined from a prospective study of 496 community female adolescents (M = 13.5, SD = 0.7 at baseline) who completed eight annual assessments of potential risk factors and eating disorders from preadolescence to young adulthood.

Results—Three variables exhibited positive linear increases: Perceived pressure to be thin, thin-ideal internalization and body dissatisfaction; three were best characterized as quadratic effects: dieting (essentially little change); negative affectivity (overall decrease), and BMI (overall increase). Elevated body dissatisfaction at ages 13, 14, 15, and 16 predicted DSM-5 eating disorders onset in the 4 year period after each assessment, but the predictive effects of other risk factors were largely confined to age 14; BMI did not predict eating disorders at any age.

Discussion—The results imply that these risk factors are present by early adolescence, though eating disorders tend to emerge in late adolescence and early adulthood. These findings emphasize the need for efficacious eating disorder prevention programs for early adolescent girls, perhaps targeting 14 year olds, when risk factors appear to be most predictive. In early adolescence, it might be fruitful to target girls with body dissatisfaction, as this was the most consistent predictor of early eating disorder onset in this study.

Keywords

eating disorders; adolescence; developmental course; predictive effects; body dissatisfaction

Eating disorders show a chronic course and result in significant functional impairment, emotional distress, and medical problems.[1, 2, 3] A cross-sectional retrospective epidemiological study suggested that approximately 7.5% of young women meet criteria for a DSM-IV-TR[4] eating disorder of anorexia nervosa, bulimia nervosa, or binge eating disorder (BED).[1] A recent examination of the natural history of DSM-5 eating disorders[5] in a community sample of 496 female adolescents who completed annual diagnostic interviews

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over an 8-year period found that 13% of participants experienced some form of threshold or subthreshold eating disorder by age 20, with the peak age of onset for bulimia nervosa and BED being 17-18 years of age, and 18-20 for purging disorder.[6] Retrospective data suggest a median age of onset (with interquartile range) of 18 years (16-22) for anorexia nervosa, 18 years (14-22) for bulimia nervosa, and 21 years (17-32) for BED among community-recruited adults.[1]

Although the incidence of eating disorders in early adolescence (ages 10-14) is very low,[3, 7] extant data suggest that risk factors for symptoms of eating disorders[8] and eating disorder syndromes[7,9] escalate in early adolescence. Accordingly, the focus of the present report is on the development and predictive power of potential eating disorder risk factors during early adolescence. The first aim of this study was to describe the developmental changes in potential risk factors from age 13 to 21, using data from a 8-year longitudinal study of community female adolescents.[6] We focused on young women because they are at much higher risk for onset of eating pathology than young men.[1] We examine whether the course of each variable is increasing, decreasing, or stable over this developmental period and, if change is present, whether it is linear or quadratic, as non-linear change might capture critical periods of increases in risk factors. These data should provide direction regarding when to target populations at elevated risk for eating disorders by virtue of possessing these risk factors. Further, if there are qualitatively distinct periods during which different risk factors emerge prior to eating disorder onset, it may be possible to improve the yield of prevention efforts by targeting different risk factors during these distinct developmental periods. It is possible that decreasing these risk factors may reduce future eating disorder onset, though predictive effects cannot establish a causal relation because it is always possible that some third variable explains both the emergence of the risk factor and eating pathology.

Very few studies have examined the course of potential risk factors prior to mid-adolescence. One exception is Bucchianeri, Arikian, Hannan, Eisenberg, and Neumark-Sztainer,[10] who followed a cohort of girls recruited during middle school (*M* age at first assessment = 12.8 years, *SD* = 0.7) over a 10-year period; data revealed a significant upward linear increase in body dissatisfaction from middle school to high school and to young adulthood. However, body mass index (BMI = kg/m2) scores also increased across all three time points for both male and female participants, and after adjusting for BMI, the linear trend in body dissatisfaction was nonsignificant. No studies, to our knowledge, have investigated the temporal development of several risk factors prior to and during peak period of eating disorder onset (i.e., from early adolescence to young adulthood).

Guided by the dual-pathway model of eating pathology[11] and prior research with female adolescents,[9,12-15] we focused on six potential risk factors: (1) perceived pressure to be thin, (2) thin-ideal internalization, (3) body dissatisfaction, (4) dieting, (5) temperamental negative affectivity (i.e., the tendency to experience negative emotional states), and (6) body mass. The dual-pathway model posits that perceived pressure to be thin from significant others and the media and internalization of the thin beauty ideal produces body dissatisfaction, which in turn promotes unhealthy dieting behaviors that may progress to an eating disorder. Body dissatisfaction may also contribute to negative affect given the

importance of appearance in Western culture, which in turn might lead people to binge eat to provide comfort and distraction from negative emotions. According to the affect regulation model,[11] people binge eat in an effort to achieve comfort and distraction from their negative emotions. Previous research with adolescents has found that female adolescents in 6-9th grade reporting social pressure to be thin and body image preoccupation showed a higher risk for onset of threshold or subthreshold bulimia nervosa or binge eating disorder over 3-year follow-up,[14] high school women reporting weight and shape concerns and negative affectivity showed a higher risk for onset of threshold or subthreshold bulimia nervosa over 4-year follow-up,[8] and female adolescents (11-15 years of age) reporting elevated dietary restraint showed elevated risk for onset of threshold or subthreshold bulimia nervosa over 5-year follow-up.[16] It is arguably more important to identify youth at elevated risk for *any* eating disorder because prevention programs should ideally target all eating disorders, rather than just one type of eating disorder, and elevated dietary restraint has been found to increase risk for onset of any eating disorder among young women (16-23 years of age) over 2-year follow-up,[12] young women (14-15 years of age) over 3-year follow-up,[14] and 16-year-old women over 1-year follow-up.[15] In an earlier report examining data used in the present study using classification tree analysis to predict onset of threshold and subthreshold eating disorders over 8-year follow-up,[17] a three-way interaction emerged suggesting a body dissatisfaction pathway to eating disorder onset that was amplified by depressive symptoms, as well as a pathway characterized by self-reported dieting among young women who are more satisfied with their bodies. Further, increased adiposity has been theorized to result in both increased social pressure to be thin and body dissatisfaction, which putatively lead to dieting, negative affect, and elevated risk for eating pathology.[18] BMI is known to increase during the adolescent developmental period.[19] For example, one study found that self-reported BMI among female adolescents in their younger cohort increased from an average of 21.7 at the first assessment (*M* age = 12.8) to an average of 26.4 at the third assessment (*M* age = 23.2).[10]

To identify variables that predict pathological eating behavior in children and early adolescents, Gardner, Stark, Friedman, and Jackson examined the degree to which six variables (weight, teasing, body esteem, body dissatisfaction, depression, perceived body size, ideal body size) predicted elevated scores on an eating disorder survey in 216 female and male children ages 6-14 who were assessed annually for 3 years.[20] They examined each predictor at various ages to identify when the factors tended to became significant predictors of elevated eating disorder scores, finding that low body esteem became a significant factor at age 9, depression at age 10, body dissatisfaction and larger perceived body size judgments at age 11, and thinner ideal body sizes at age 12. Using a similar analytic approach, our second aim was to examine whether each of the Aim 1 potential risk factors examined during early adolescence predicted subsequent onset of DSM-5 eating disorders, and whether the predictive effects of these risk factors varied as a function of age. We sought to extend the findings of Gardner and colleagues[20] by (a) following a larger cohort of youth, (b) examining a longer period of time, and (c) by predicting onset of DSM-5 eating disorders as determined by diagnostic interview. To address this aim, we examined the degree to which each of the six potential risk factors at ages 13, 14, 15, and 16 significantly predicted the onset of eating disorders in the four year period following each assessment.

These data may also advance etiologic models for eating disorders by illuminating the earliest risk factors that predict eating disorder onset.

Method

Participants and Procedures

Participants were 496 female adolescents recruited from public (n = 409) and private middle schools (n = 87) in a large US city. Participants ranged from 12 to 15 years of age (M = 13.5, SD = 0.7) and were in 7th or 8th grade at T1. The sample was composed of 2% Asian/Pacific Islanders, 7% African Americans, 68% Caucasians, 18% Hispanics, 1% Native Americans, and 4% other/mixed, which is generally representative of the ethnic composition of the schools from which we sampled (2% Asian/Pacific Islanders; 8% African Americans, 65% Caucasians, 21% Hispanics; 4% "other or mixed").[6,11] Average parental education, a proxy for socioeconomic status, was 29% high school graduate or less, 23% some college, 33% college graduate, and 15% graduate degree, which was representative of the metropolitan area from which we sampled (34% high school graduate or less; 25% some college; 26% college graduate; 15% graduate degree).[6,11]

The study was described as an investigation of adolescent mental and physical health. An informed consent letter and self-addressed return envelope were sent to parents of middle school students, resulting in a baseline participation rate of 56%, echoing recruitment rates observed in other similar longitudinal studies involving repeated diagnostic interviews.[3] Participants completed a survey and an interview at baseline (T1) and at seven annual follow-ups (T2, T3, T4, T5, T6, T7, & T8). Trained female assessors conducted diagnostic interviews. Assessors had to demonstrate an inter-rater agreement (k > .80) with the project manager using tape-recorded interviews before collecting data. Assessments took place at the school, the participants' place of residence, or the research office. Participants received a gift certificate or cash payment for completing each assessment. The University of Texas at Austin Institutional Review Board approved this study.

Measures

Perceived sociocultural pressure to be thin from family, friends, dating partners, and the media was assessed with the Perceived Sociocultural Pressure Scale.[21] Response options to these 10 items (e.g., "I've felt pressure from my friends to lose weight") ranged from 1 - 5 (1 = *none*, 3 = *some*, 5 = *a lot*). Items for this scale (and those below) were averaged for scoring. This scale has shown internal consistency (α = .88), 2-week test-retest reliability (r = .93), and predictive validity for future onset of bulimic symptoms (α = .85 at T1).[21]

Thin-ideal internalization was assessed with the Ideal-Body Stereotype Scale-Revised,[22] which assesses agreement with statements concerning what attractive women look like. Response options to these six items (e.g., "Slender women are more attractive") were: 1 = *strongly disagree*, 2= *disagree*, 3 = *neutral*, 4 = *agree*, 5 = *strongly agree*. This scale has shown internal consistency (α = .89), 2-week test-retest reliability (r = .80), sensitivity to detecting effects of an eating disorder prevention program that focuses on reducing thin-

ideal internalization, and predictive validity for future bulimic symptom onset (α = .81 at T1).[22]

Body dissatisfaction was assessed with nine items from the Satisfaction and Dissatisfaction with Body Parts Scale[23] that measured satisfaction with body parts that are often of concern to girls and women (e.g., waist, thighs). Response options were: 1 = *extremely dissatisfied*, 2 = *moderately dissatisfied*, 3 = *neutral*, 4 = *moderately satisfied*, 5 = *extremely satisfied*. This scale has shown internal consistency (α = .94), 3-week test-retest reliability (*r* = .90), sensitivity to detecting effects from an eating disorder prevention program, and predictive validity for future onset of eating disorders (α = .94 at T1).[22]

Dieting was assessed with the Dutch Restrained Eating Scale [24] that measures the frequency of dieting behaviors. Response options to these nine items (e.g., "If you have put on weight, do you eat less than you normally would?") were: 1 = *never*, 2 = *seldom*, 3 = *sometimes*, 4 = *often*, 5 = *always*. This scale has shown internal consistency (α = .95), 2-week test-retest reliability (*r* = .82), sensitivity to detecting effects of eating disorder prevention programs, and predictive validity for future onset of eating disorders (α = .91 at T1).[24]

Negative affectivity was assessed with Buss and Plomin's Emotionality Scale,[25] which measures agreement with statements regarding tendencies to become affectively distressed. Using a response format modified from the original scale, participants indicated how much they agreed with eight items regarding their tendency to become emotionally distressed or aroused (e.g., "I frequently get upset") using the following response options: 1 = *strongly disagree*, 2 = *disagree*, 3= *neutral*, 4 = *agree*, 5 = *strongly agree*. This scale showed internal consistency (α = .80 at T1), convergent validity with other measures of negative affect[25] and predictive validity for future increases in bulimic symptoms.[11]

Body mass was determined by the body mass index (BMI = kg/m^2), which was obtained by physical measurements. Height was measured to the nearest millimeter using portable stadiometers. Weight was assessed to the nearest 0.1 kg using digital scales with participants wearing light indoor clothing without shoes or coats. Height and weight were measured twice each and averaged. Height and weight were directly assessed in the 3324 diagnostic interviews conducted in person, but were based on self-report for the 534 diagnostic interviews conducted over the phone because subjects had moved from the Austin area (13.8% of all interviews).

Eating pathology was assessed by the Eating Disorders Diagnostic Interview (EDDI), which probed for eating disorder symptoms over the past 12-months at each assessment.[17] The EDDI is a semi-structured interview that was adapted from the Eating Disorder Examination (EDE)[26] by inquiring about the presence of each eating disorder symptom on a monthly basis over the past year and by omitting non-diagnostic items from the weight concerns, shape concerns, eating concerns, and dietary restraint subscales. We used these data to determine whether participants met criteria for the proposed DSM-5 eating disorders.[6] Specifically, we diagnosed the following DSM-5 disorders: anorexia nervosa, bulimia nervosa, BED, and other specified feeding or eating disorder (which includes atypical anorexia nervosa, bulimia nervosa of low frequency or limited duration, BED of

low frequency or limited duration, and purging disorder; a description of the operationalizations of subthreshold conditions described previously[6]). Test-retest reliability was assessed by randomly selecting a subset of 184 participants who were interviewed by the assessors and then re-interviewed by the same assessor within a week; the test-retest reliability was $K = .79$ for DSM-5 eating disorders. Inter-rater agreement for the eating disorder diagnoses was assessed by randomly selecting subset of 207 participants who were re-interviewed by a second blinded assessor; the inter-rater agreement was $K = .75$ for DSM-5 eating disorders. In support of the validity of the EDDI, participants with versus without DSM-5 eating disorders (both threshold disorders and "other specified feeding or eating disorders") report elevated functional impairment, emotional distress, suicidal ideation, and mental health treatment, as well as abnormalities in body mass[6] and the EDDI has been shown to detect effects of eating disorder prevention programs.[22]

Statistical Methods

We investigated the first aim by fitting linear and quadratic relations wherein each of the six potential risk factors was regressed on the participant's age for linear models and regressed on age and quadratic age for the quadratic models. Prior to the analysis, age and quadratic age were centered at age 13, so that the intercept represents the estimated level of the outcome at age 13. All models were fit using mixed models implemented with nlme package from the R project.[27] We linked the T1-T8 assessments to the participant's closest age because we were interested in developmental changes in risk factors based on participant age, rather than the assessment wave. Effect sizes were estimated by converting *t* values to Pearson's *r*. We quantified the change in each risk factor using the change in the marginal means between age 13 and 21 divided by age 13 standard deviation which represents the change in standard deviations.

We investigated the second aim using logistic regression models implemented using the glm function from the R Project.[28] The dependent variable was first incidence of any DSM-5 eating disorder (ED) reported within four years following each respective assessment, henceforth referred to as adolescent eating disorder onset, which permitted us to compare the predictive power of potential risk factors each year of early adolescence (participants diagnosed with an eating disorder in the same assessment or prior to the assessment point were excluded from the analysis, as well as participants diagnosed with an eating disorder more than four years following that particular assessment). Adolescent eating disorder onset was regressed on each of the potential risk factors measured at ages 13, 14, 15, and 16 (number of eating disorder onset cases for each period were 25, 25, 31, and 39, respectively); six participants had an eating disorder at or before age 13 and were not included in the Aim 2 analyses. Potential risk factors were transformed to *z* scores within age prior to model fitting to facilitate the interpretation of odds ratios: odds ratio thus represent a one standard deviation change in the independent variables.

We implemented multiple imputation for Aim 2 so models at each of the four ages at which the risk factors were assessed included all participants. For the models examining body mass, we used an age-standardized BMI (i.e., the number of standard deviations ± the BMI for a putative age). Missing data values were imputed using the Amelia II package

developed for the R project, which uses available data to impute missing values via a bootstrapping approach. The observed and imputed data were compared to ensure that imputed values were in the same range as observed data. Missing data were replaced with imputed values in 20 data sets and each data set was analyzed separately; model parameters and standard errors from models fit to each of the twenty data sets were combined for the final results.

Results

Preliminary Analyses

With respect to attrition, the percentages of participants missing self-report and diagnostic interview data from were 0% (N = 496), 1% (N = 491), 3% (N = 481), 3% (N = 481), 2% (N = 486), 3% (N = 481), 4% (N = 476), and 6% (N = 466) for each of the eight annual assessments. We were often able to collect data from participants who did not provide data at an earlier assessment (99% of participants provided data at baseline and at least one additional assessment). Attrition was not significantly correlated with any of the potential risk factor variables or with eating disorder diagnosis.

Regarding the incidence of DSM-5 eating disorders between ages 14-20, a total of 59 participants (12.0%) experienced at least one eating disorder (atypical AN n = 12, anorexia nervosa n = 3, subthreshold bulimia nervosa n = 21, bulimia nervosa n = 12, subthreshold BED n = 14, BED n = 14, purging disorder n = 17; 33 participants had more than one disorder).

Developmental Course of Potential Risk Factors from Ages 13 to 21

Results for Aim 1 models are presented in Table 1. Three of the potential risk factors exhibited positive linear increases across the age range, but did not exhibit a quadratic effect: Perceived pressure to be thin [$t(2771) = 10.49$, $p < .001$], thin-ideal internalization [$t(3248) = 6.71$, $p < .001$], and body dissatisfaction [$t(3248) = 4.97$, $p < .001$]. Dieting exhibited a significant negative linear effect [$t(3246) = -2.66$, $p = .008$], indicating an instantaneous decrease at the intercept, and a significant positive quadratic effect [$t(3246) = 2.94$, $p = .003$], indicating an increasingly positive increase in dieting across time. Negative affectivity exhibited a significant linear decrease across time [$t(2353) = -5.01$, $p < .001$]. BMI exhibited a significant positive linear effect [$t(3246) = -13.74$, $p < .001$], indicating an instantaneous increase at the intercept, and a significant negative quadratic effect [$t(3246) = -5.63$, $p < .001$] indicating that the positive increase in BMI decelerated across time. Figure 1 through Figure 6 plot the fitted lines showing linear or quadratic effects for average levels of the potential risk factors for the full sample, as well as the observed average levels for participants who remain free of an eating disorder and those who exhibited eating disorder onset at age 16 or later. Perceived pressure to be thin exhibited a 0.49 standard deviation increase between 13 and 21; thin-ideal internalization exhibited a 0.27 standard deviations increase between 13 and 21; body dissatisfaction exhibited a 0.17 standard deviations increase between 13 and 21; dieting exhibited a 0.03 standard deviations increase between 13 and 21; negative affectivity exhibited a 0.37 standard deviations decrease between 13 and 21; and BMI exhibited a 0.61 standard deviations increase between 13 and 21.

Degree to which Risk Factors at 13-16 Predict Future Eating Disorder Onset

Results for Aim 2 models are presented in Table 2. Five of the six variables were significant predictors of subsequent eating disorder onset at one or more of the early adolescent timepoints, although the variables differed markedly in their degree of significance. One risk factor – body dissatisfaction – significantly predicted future eating disorders at all four of the examined assessment points. A second variable – negative affectivity –significantly predicted eating disorder onset at two of the four assessment points (ages 14 and 15), and three risk factors – perceived pressure to be thin, thin-ideal internalization, and dieting – predicted eating disorder onset at only one of the four early adolescent assessment points (age 14). Body mass index failed to predict eating disorder onset at any of the four examined age points.

Another method of summarizing the Aim 2 results involves the number of predictive effects at each age. With this focus, the greatest number of risk factors were significant at the age 14 assessment; five of six measures at that timepoint significantly predicted eating disorder onset within the next four years. Conversely, only one variable was significant at age 13 and two were significant at age 15.

Discussion

The goals of this study were to describe developmental changes in the levels of six potential risk factors for eating disorder onset from ages 13 to 21 and to examine whether the presence of each risk factor at ages 13-16 significantly predicted the first incidence of eating disorders within the four years following that assessment point. We focused on the following six potential risk factors: perceived pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, negative affectivity, and body mass.

Regarding the development of potential risk factors during this critical and complex developmental period, three of the six variables exhibited a positive linear increase, two exhibited a positive quadratic increase, and one a negative linear decrease. Of the three variables that significantly increased in a linear fashion – perceived pressure to be thin, thin-ideal internalization, and body dissatisfaction – the first variable (perceived pressure to be thin) appeared to have the largest increase over time; thin-ideal internalization and body dissatisfaction had statistically significant increases, but these changes both reflected only slight increases over an 8-year period. Self-reported dieting was most appropriately characterized as a quadratic line, first decreasing slightly and then increasing from 13 to 21 years of age, appearing to exhibit the least change over eight years. BMI was also most appropriately charted as a quadratic line, first increasing markedly and then decelerating over time, for an overall total increase. Negative affectivity was the only variable to exhibit a significant decrease from ages 13 to 21. Thus, although full-threshold eating disorders are rare in early adolescence, inspection of fitted lines in Figures 1-6 suggest that, with the exception of body mass and possibly also perceived pressure to be thin, the potential risk factors examined in this report appear to be present by age 13 and do not show marked increases from early adolescence into young adulthood. The lack of substantial change across such a long and important developmental period in the lifespan for four of the six examined risk factors is striking.

An examination of the developmental course of potential eating disorder risk factors in this degree of detail and scope has not been previously conducted. One of the most relevant studies to examine the issue of risk factor course of development[10] reported a significant upward linear increase in body dissatisfaction from middle school to young adulthood. However, the linear trend in body dissatisfaction in that study became nonsignificant after adjusting for BMI, which also increased during this time period. We conducted a similar control by fitting the linear trend in body dissatisfaction controlling for age-standardized BMI, and found that the linear trend for body dissatisfaction remained significant [$t(3192) = 4.03$, $p < .001$] controlling for zBMI, which was treated as a time-varying covariate in the model. Thus, the increase in body dissatisfaction in our sample could not be attributed solely to changes in body composition from 13 to 21 years of age. The discrepancy between the effects reported in the present study and those reported previously by Bucchianeri and colleagues may have been due to greater accuracy in modeling growth in the present study (8 data points rather than 3), greater statistical power ($N = 496$ vs. 250 girls in the younger cohort with self-reported BMI), or the fact that the three assessment points in their study went further into early adulthood (*M* age at the third assessment = 23.2 years).

Overall, the results of Aim 1 imply that, though eating disorders tend to emerge in late adolescence and early adulthood, most of the examined risk factors are present by early adolescence. The pattern of results also supports the notion that a general dissatisfaction with one's body emerges before rather than after perceived increases in the pressure to attain an unrealistic version of thinness promoted for women in many Western cultures. Levels of body dissatisfaction also appeared to be elevated prior to significant increases in body mass that occur in middle and late adolescence. Given that four of the six examined factors did not markedly increase after age 13 indicates the importance of longitudinal studies that track the course of these factors starting even earlier in the lifespan to better capture their emergence and developmental course.

Our second aim involved the degree to which each of these six risk factors at ages 13, 14, 15, and 16 significantly predicted the first incidence of an eating disorder within the following 4-year period, in an effort to determine whether it would be optimal to implement prevention programs in early adolescence and, if so, which high risk populations should be targeted. Only one variable emerged as a significant predicted at age 13 (body dissatisfaction), whereas five of the six variables at age 14 significantly predicted a future eating disorder. Two risk factors at age 15 were significant predictors but only one remained significant at age 16. Of the variables we examined, body dissatisfaction was clearly the most consistent and robust predictor of future eating disorder, being significant at all four early adolescent timepoints, suggesting that it is an important high-risk population to target with prevention efforts. Elevated body dissatisfaction had a mean odds ratio of 1.68 across the four assessment points, suggesting that this risk factor increased the likelihood of developing an eating disorder by 68%. The finding that body dissatisfaction was the most consistently potent risk factor among those we examined may suggest that a general deviation from a perceived physical ideal, rather than either perceived pressure to attain unreasonable thinness or an idealization of thinness, might first motivate young female adolescents to begin engaging in unhealthy weight control behaviors that typify eating

disorders, though it is not possible to draw causal inferences from such prospective effects. The overall findings also support the notion that early negative affectivity (i.e., the increased tendency to experience unpleasant emotional statues such as fear, frustration, sadness, and annoyance) functions as a significant predictor of pathological eating in young women, being significant at two of four early adolescent assessment points examined in the present study.

The present findings parallel the results from Gardner et al.,[20] who examined a younger sample, starting with girls and boys ages 6, 9, and 12, assessing them annually for three years. They report that low body esteem emerged as the first significant predictor of elevated eating disorder scores on a survey at age 9, followed by depression at age 10, body dissatisfaction and larger perceived body size judgments at age 11, and, finally, thinner ideal body sizes at age 12.

No support was found in the present study for elevated body mass to increase the risk for future eating disorders in young female adolescents. This variable was the only examined measure that did not predict eating disorder at any of the four assessment points. Although BMI has significantly predicted increases in eating pathology in some studies,[21] a meta-analytic review[29] suggested that, in general, research has found that elevated body mass functions as a stronger risk factor for specific eating disorder risk factors, such as perceived pressure to be thin, body dissatisfaction, and dieting than for future eating pathology per se. This overall pattern of findings may suggest that subjective body dissatisfaction, rather than elevated adiposity, might set the stage for the emergence of disordered eating behaviors.

A striking feature of results in the present study is that five risk factors were highly significant predictors at age 14, whereas only one factor attained significance at age 13. The average odds ratio for the five significant risk factors at age 14 was 1.98, indicating that these variables on average doubled the likelihood of eating disorder onset. This pattern suggests that age 14 may be a key developmental timepoint to intervene, perhaps due to the combination of physical and cognitive maturational processes, as female adolescents' transition from early to middle adolescence. For example, most female adolescents have completed puberty by this time, and in addition to this significant physical change experience a variety of major cognitive and interpersonal changes, including an increasing capacity for abstract thinking, a more abstract characterization of themselves, increases in the influence of peers, and the onset of dating and sexual experiences.[30]

Whereas we previously examined the onset and course of DSM-IV[2] and DSM-5[6] eating disorders during adolescence, the present study described how select risk factors change over this developmental period and examined developmental changes in the predictive power of these risk factors at various ages. An improved understanding of the developmental course and magnitude of specific eating disorder risk factors prior to the peak period of onset is vital for the optimal timing of prevention interventions, and an underlying impetus for the present study was to identify when to intervene with early adolescent girls to have the greatest likelihood of reducing risk for eating disorders prior to their peak period of onset. Risk factors presumably need to be present for the identification of high-risk populations. Therefore, the present findings support the contention that prevention programs

should target young girls at risk for eating disorder onset in early adolescence, potentially at age 14, to maximize intervention effects. The findings lend support to eating disorder prevention programs that target early adolescent girls with body image or weight concerns.[22, 29] Methods for reducing body dissatisfaction could include dissonance-based (e.g., standing in front of a mirror and generating positive statements about one's appearance that are shared with the group, role-plays in which the person works to dissuade the leader from pursuing the unrealistic thin-ideal standard of female beauty) or cognitive-behavioral (e.g., refraining from negative body self-talk, increased exposure to feared body image situations) exercises to counteract body dissatisfaction.[31] In a meta-analytic review of all identified eating disorder programs that were evaluated by controlled trial between 1980–2006,[32] there was considerable variation in the range of intervention effects for reducing body dissatisfaction, with cognitive-behavioral treatments aimed at reducing body dissatisfaction having the strongest effects ($r > .50$).[33, 34]

Regarding other eating disorder risk factors, perceived pressure to be thin showed the greatest increase in prevalence for young women from ages 13 to 21. Negative affectivity was the only examined risk factor which significantly decreased from ages 13 to 21 and it is possible that this variable is a relevant risk factor for eating disorder onset during the middle school, compared to the high school, age period. It is also possible that other forms of negative affectivity, perhaps more closely related to anxiety, trauma, and transitional stressors, might have a different developmental course and pattern of association with increased eating disorder risk. On a related note, the construct of negative urgency (i.e., the tendency to act rashly when distressed) in fifth grade children was found to predict an increased expectancy that eating would reduce negative affect and subsequent increases in binge eating behavior one year later.[35] Regarding self-reported dieting, rates were generally flat over time, which is consistent with other longitudinal research,[36] although they increased sharply at ages 19 -20 among the subset of women who developed an eating disorder during the study; potentially this variable increased as a result, rather than cause, of the eating disorder. It should be noted that controlled research with obese women has failed to find that dieting behaviors significantly increase the risk for the eating disorder symptom of binge eating.[37]

Previous meta-analytic reviews of eating disorder prevention interventions have found significantly weaker effects for programs aimed at adolescents less than 15 years of age compared to prevention interventions with adolescents 15 years of age or older.[32] Though universal psychoeducational prevention interventions with this younger age group have tended to produce nonsignificant effects on eating disorder risk factors,[32] some interactive and media literacy programs have produced significant effects in child or early adolescent samples immediately post-intervention (e.g., body dissatisfaction in girls mean age = 11.8)[38] with some effects remaining to 3-month follow-up (thin-ideal internalization in girls mean age = 10.6;[39] body dissatisfaction in girls mean age = 12.5)[40] or even 12-month follow-up (e.g., dieting and thin-ideal international in girls and boys mean age = 12.9).[41] The finding that more interventions have been found to be effective with older adolescents[32] suggests it might be valuable to adapt eating disorder prevention programs with the strongest evidence

bases in middle and late adolescence samples for delivery in early adolescence, as the present study strongly suggests that eating disorder risk factors are elevated by age 13.

Study limitations should be considered when interpreting these findings. First, with the exception of BMI, the predictor variables in this study were based on adolescent report and it is possible that some variation in the findings could be due to differential psychometric properties of the scales. For instance, body dissatisfaction may be easier to assess in a reliable and valid manner, whereas measurement of perceived pressure to be thin, thin-ideal internalization, and dieting is more complicated. However, psychometric properties, both internal consistency and test-retest reliability, for all five questionnaire-based risk factors were respectable and fairly comparable, which suggests that the pattern of results was not biased by measurement differences. Second, we investigated a small subset of putative risk factors for eating pathology and it will be important to investigate additional risk factors that may elucidate other pathways to eating pathology,[e.g.,42] that could have important implications to the design of eating disorder prevention interventions. Third, results identified common risk factors for eating disorders, rather than disorder-specific risk factors and it is likely that risk factors for specific eating disorder syndromes and specific symptomatic behaviors differ. Although considerably larger studies would be necessary to identify disorder-specific risk factors for each type of eating disorder, this type of research is needed. Fourth, because the sample contained only women, results cannot be generalized to men. Fifth, as is the case with all longitudinal research, it is possible that some unmeasured variable accounts for the observed prospective effects. Sixth, our focus in this paper was on the course and predictive effects of potential risk factors examined individually, in order to improve understanding of the developmental patterns and predictive magnitude of each specific potential risk factor; we have previously tested for interactions among risk factors to predict future eating disorder onset across the entire 8-year follow-up period.[17] Given the possibility that some omitted third variable or moderational effects explain the prospective effects observed in longitudinal studies, it will be vital to conduct randomized prevention trials that reduce suspected risk factors with active credible alternative comparison conditions in order to provide an experimental test of the relation between these risk factors and these eating pathologies.

In sum, it is hoped that prevention programs targeting high-risk populations characterized by the risk factors that predicted eating disorder onset in early adolescence, prior to peak period of risk for eating disorder onset, will permit more effective prevention efforts. The present findings illustrate that many eating disorder risk factors are present by early adolescence. The results also suggest the need for efficacious eating disorder prevention programs for early adolescent girls, perhaps targeted at age 14, when most risk factors appear to be predictive.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This study was supported by a career award (MH01708) and a research grant (MH/DK61957) from the National Institutes of Health. Thanks go to project research assistants Sarah Kate Bearman, Cara Bohan, Emily Burton, Melissa Fisher, Lisa Groesz, Katherine Presnell, Jenn Tristan, Natalie McKee, and Katy Whitenton, a multitude of undergraduate volunteers, the Austin Independent School District, and the participants who made this study possible.

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Table 1

Linear and quadratic models of change in risk factors between age 13 and 21

Outcome and model	Parameter	Estimate	Standard Error	*t*	*p*	*r*
Perceived Pressure to be thin						
Linear	Intercept	1.75	0.03	51.90	< .001	.92
	Linear	0.05	0.00	10.49	< .001	.43
Quadratic	Intercept	1.76	0.04	43.93	< .001	.90
	Linear	0.04	0.02	2.74	.006	.12
	Quadratic	1.75	0.03	51.90	< .001	.92
Thin-ideal internalization						
Linear	Intercept	3.22	0.03	112.87	< .001	.98
	Linear	0.02	0.00	6.71	< .001	.29
Quadratic	Intercept	3.21	0.03	96.95	< .001	.98
	Linear	0.03	0.01	2.63	.009	.12
	Quadratic	0.00	0.00	−0.77	.440	−.04
Body dissatisfaction						
Linear	Intercept	2.78	0.04	74.69	< .001	.96
	Linear	0.02	0.00	4.97	< .001	.22
Quadratic	Intercept	2.75	0.04	65.24	< .001	.95
	Linear	0.04	0.02	2.74	.006	.12
	Quadratic	0.00	0.00	−1.40	.161	−.06
Dieting						
Linear	Intercept	2.11	0.03	60.94	< .001	.94
	Linear	0.00	0.00	0.55	.583	.03
Quadratic	Intercept	2.17	0.04	53.67	< .001	.93
	Linear	−0.04	0.02	−2.66	.008	−.12
	Quadratic	0.01	0.00	2.94	.003	.13
Negative affectivity						
Linear	Intercept	2.82	0.03	91.39	< .001	.97
	Linear	−0.03	0.01	−5.01	< .001	−.22
Quadratic	Intercept	2.80	0.04	77.28	< .001	.96
	Linear	−0.01	0.02	−0.69	.490	−.03
	Quadratic	0.00	0.00	−0.83	.406	−.04
Body mass index						
Linear	Intercept	21.16	0.22	97.12	< .001	.98
	Linear	0.35	0.01	29.35	< .001	.80
Quadratic	Intercept	20.86	0.22	92.93	< .001	.97
	Linear	0.58	0.04	13.74	< .001	.53
	Quadratic	−0.03	0.01	−5.63	< .001	−.25

Table 2

Logistic regression analyses predicting incidence of eating disorders (within the subsequent 4 years) for risk factors assessed at ages 13, 14, 15, and 16

Risk Factor	Age	Coefficient	*SE*	*df*	*t*	*p*	Odds Ratio	95% CI
Perceived pressure to be thin	13	0.47	0.24	113	1.94	.055	1.60	0.99 - 2.56
	14	0.76	0.21	254	3.60	< .001	2.14	1.40 - 3.22
	15	0.28	0.22	120	1.27	.207	1.33	0.85 - 2.08
	16	0.17	0.16	460	1.02	.309	1.18	0.86 - 1.63
Thin-ideal internalization	13	0.35	0.31	56	1.15	.257	1.43	0.76 - 2.66
	14	0.71	0.25	297	2.88	.004	2.04	1.25 - 3.32
	15	0.01	0.19	369	0.06	.950	1.01	0.69 - 1.48
	16	0.03	0.17	449	0.16	.873	1.03	0.73 - 1.43
Body dissatisfaction	13	0.52	0.25	118	2.03	.044	1.68	1.01 - 2.77
	14	0.64	0.23	402	2.79	.005	1.89	1.21 - 2.97
	15	0.41	0.21	325	1.99	.048	1.51	1.00 - 2.25
	16	0.51	0.17	457	2.93	.004	1.67	1.19 - 2.34
Dieting	13	0.46	0.25	107	1.82	.072	1.59	0.96 - 2.61
	14	0.65	0.20	364	3.29	.001	1.92	1.30 - 2.83
	15	0.10	0.19	394	0.54	.593	1.11	0.76 - 1.62
	16	0.21	0.16	459	1.28	.200	1.23	0.90 - 1.70
Negative affectivity	13	0.38	0.28	71	1.38	.172	1.46	0.84 - 2.53
	14	0.65	0.22	356	3.01	.003	1.91	1.25 - 2.92
	15	0.67	0.19	376	3.58	< .001	1.96	1.35 - 2.83
	16	0.28	0.16	463	1.72	.087	1.32	0.96 - 1.82
Body mass index	13	0.15	0.30	49	0.48	.631	1.16	0.63 - 2.12
	14	0.08	0.20	384	0.40	.689	1.08	0.73 - 1.60
	15	0.18	0.18	346	1.01	.314	1.20	0.84 - 1.70
	16	0.13	0.15	444	0.85	.394	1.14	0.84 - 1.52